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Ph.D 7/Sept. 92

The University of Sydney

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THIS THESIS HAS BEEN ACCEPTED
FOR THE AWARD OF THE DEGREE
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**SURVIVAL TUCKER: ABORIGINAL DIETARY INTAKE AND
A SUCCESSFUL COMMUNITY-BASED NUTRITION INTERVENTION PROJECT**

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**A THESIS SUBMITTED FOR THE DEGREE OF DOCTOR OF PHILOSOPHY
IN THE FACULTY OF MEDICINE
UNIVERSITY OF SYDNEY**

March 1992

The work presented in this thesis is my own except where acknowledged in the text. It has not been previously submitted, either in whole or in part, for a degree at this or any other University.



.....
Amanda Lee
March 1992

This work is dedicated to the Aboriginal People who have suffered greatly during the last two hundred years, to my Family, my mother and particularly to the memory of my father, Alan Edward Lee.

ABSTRACT

Nutrition-related health problems in Aboriginal communities have been described frequently, but little attention has been paid to the measurement of Aboriginal dietary intake. Even less attention has been paid to formal projects aimed at improving the nutritional health of Aboriginal people, or to methods to evaluate such projects.

This thesis describes firstly the development and testing of the 'store-turnover method', a non-invasive dietary survey methodology for quantitative measurement of food and nutrient intake in remote, centralised Aboriginal communities. Secondly, it presents congruent validation of the store-turnover method against biological indicators of nutritional status and appraisal of the face validity of the method. Thirdly, it describes the use of the method (together with biochemical, anthropological and haematological indicators of health and nutritional status) as a rational basis for the planning, implementation and evaluation of a community-based nutrition intervention project in a northern coastal community. In order to maximise the potential for change, several intervention strategies were applied simultaneously in the intervention project. These addressed two major issues: increasing motivation of community members and promoting an increased variety of food choice.

Results indicated that, under the unique circumstances of food supply in remote centralised Aboriginal communities, the store-turnover method was valid for measuring the dietary intake patterns of these communities. The method was able to pinpoint potential strategies for community-based nutrition intervention projects and was also shown to be a sensitive indicator of dietary changes brought about by these nutrition intervention strategies.

During the intervention project, improvements in both dietary intake and biological indicators of nutritional health were measured serially over a twelve month period. Improvements included a decrease in sugars and saturated fat, an increase in dietary fibre and a marked improvement in nutrient density over the intervention period. Biological improvements included a 12% decrease in mean serum cholesterol concentration and a significant decrease in both diastolic and systolic blood pressure; there were also marked improvements in the concentrations of several vitamins including red blood cell folate, serum vitamin B₆ and plasma ascorbic acid.

The project consequently demonstrates that when communities are involved in all stages of the development, implementation and evaluation of nutrition intervention projects, improvements in the generally poor nutritional health of Aboriginal Australians *are* possible.

The development and validation of the store-turnover method has produced a tool to provide objective dietary data which have wide implications for future health and nutrition programs in Aboriginal communities. Further, the intervention component, unique as a formally evaluated and successful community-based Aboriginal nutrition intervention study, has produced valuable information and provides a model for other studies.

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TABLE OF CONTENTS

ABSTRACT	iv
ACKNOWLEDGMENTS	v
THESIS ORGANISATION	xiv
GLOSSARY	xvi
LIST OF ABBREVIATIONS, ACRONYMS AND SYMBOLS	xvii
CHAPTER 1: ABORIGINAL NUTRITIONAL STATUS, DIETARY SURVEY METHODOLOGY AND NUTRITION INTERVENTION	1
Introduction and aim of study	1
Traditional Aboriginal lifestyle, nutrition and health	2
A brief history of cross-cultural contact	12
Contemporary Aboriginal living places in the Northern Territory	14
Post-contact diet and factors affecting nutritional health	15
Contemporary Aboriginal health and nutritional status	24
Dietary survey methodology	35
Quantitative measurement of dietary intake in Aboriginal communities	44
Improving Aboriginal health and nutrition	61
Major points	68
CHAPTER 2: PILOT STUDIES OF DIETARY SURVEY METHODS FOR APPLICATION IN REMOTE, CENTRALISED ABORIGINAL COMMUNITIES.	71
Introduction	71
Aim	72
Study design	72
The study communities	72
Dietary methods	73
Methods of data analysis	76
Results	78
Discussion	85
Conclusion	90
CHAPTER 3: APPARENT CONSUMPTION OF FOOD AND NUTRIENTS IN REMOTE, CENTRALISED ABORIGINAL COMMUNITIES AS DETERMINED BY THE STORE-TURNOVER METHOD.	91
Introduction	91
Aim	91
Study design	92
Methods	92
Results	95
Discussion	103
Major points	111
CHAPTER 4: A COMMUNITY-BASED NUTRITION INTERVENTION PROJECT: METHODS	112
Introduction and study design	112
The communities	113
Initiation of the intervention project	121
Sampling methods	121
Determination of demographic data	121
The community survey process	122
Measurement of biological indicators of nutritional status	129
Dietary intake	133

Intervention strategies	135
Methods of statistical analysis	137
CHAPTER 5: A COMMUNITY-BASED NUTRITION INTERVENTION PROJECT: DIETARY RESULTS AND COMMENTS.	140
Population	140
Dietary intake: Foods	140
Dietary intake: Nutrients	157
Contemporary use of traditional foods at Minjilang	184
Major points	187
CHAPTER 6: A COMMUNITY-BASED NUTRITION INTERVENTION PROJECT: BIOCHEMICAL, ANTHROPOMETRIC AND HAEMATOLOGICAL RESULTS AND COMMENTS	189
Sample	189
Anthropometric and metabolic data	192
Statistical considerations	195
Substance use and abuse	199
Change in anthropometric and metabolic parameters	199
The relationship between anthropometric and metabolic variables at each survey	218
Change in folate and haematological status	223
The relationship between folic acid, haematological status and other variables	231
Change in vitamin status	236
The relationship between vitamins and other variables at each survey	246
Major points	250
CHAPTER 7: VALIDATION OF THE STORE-TURNOVER METHOD AS A MEASURE OF DIETARY INTAKE IN A REMOTE, CENTRALISED ABORIGINAL COMMUNITY.	252
Congruent validation	252
Further aspects of validity	280
Major points	286
CHAPTER 8: DISCUSSION OF RESULTS AND IMPLICATIONS FOR FUTURE ABORIGINAL NUTRITION PROJECTS	289
Dietary intake	289
Biological indicators of health and nutritional status at Minjilang	295
Intervention strategies	314
Cost-benefits of the Minjilang nutrition intervention project	325
Developments arising from the project and implications for other projects	326
Further research questions	327
Conclusion	329
ADDENDUM 1: Forms and equipment used in dietary studies and community surveys	330
ADDENDUM 2: Additional data and analysis, Minjilang survey	337
ADDENDUM 3: Examples of written feedback of individual and community results, duty guidelines and training program for the Aboriginal research assistant, ALPA nutrition policy and summary of project budget	365
ADDENDUM 4: Examples of traditional stories considering over-consumption of sugar and fat.	378
BIBLIOGRAPHY	379

LIST OF TABLES

Table 1.1.	Characteristics of hunter-gatherer and western lifestyle	16
Table 1.2.	Sources of error in dietary surveys	39
Table 1.3.	Dietary consumption in decentralised, traditionally-orientated communities	58
Table 1.4.	Factors which may affect dietary survey methodologies in remote Aboriginal communities	70
Table 2.1.	Sample	78
Table 2.2.	Potential bias in applied dietary methods	79
Table 2.3.	Comparative criteria of the applied methods	80
Table 2.4.	Mean per capita nutrient intake derived from weighed and recalled dietary intakes	81
Table 2.5.	Agreement between the two methods for classification of individual nutrient intakes into three categories	82
Table 2.6.	Presence or absence (concordance) of foods in recalled and weighed records	83
Table 2.7.	Apparent consumption of selected foodstuffs measured by the store-turnover method	84
Table 2.8.	Apparent nutrient intake measured by the store-turnover method	84
Table 3.1.	Characteristics of the six communities	92
Table 3.2.	Apparent consumption of selected foodstuffs in remote Aboriginal communities	95
Table 3.3.	Apparent consumption of selected foods in remote Aboriginal communities compared with the wider Australian community	96
Table 3.4.	Apparent intake of energy and macronutrients in Aboriginal communities and wider Australia	99
Table 4.1.	Population estimates, Minjilang	117
Table 4.2.	Demographic data Minjilang June 1986	117
Table 4.3.	Population estimates, Control community	120
Table 4.4.	Collection and handling of blood samples	126
Table 5.1.	Percentage visible fat of total meat supplied to Minjilang store	151
Table 5.2a.	Dietary intake measured by the store-turnover method: Minjilang	159
Table 5.2b.	Dietary intake as measured by the store-turnover method: Control Community	160
Table 5.3a.	Major food groups contributing to total energy intake	168
Table 5.3b.	Major food groups contributing to total intake of sugars	168
Table 5.3c.	Major food groups contributing to total intake of fat	169
Table 5.3d.	Major food groups contributing to total intake of folate	169
Table 5.4.	Frequency of hunting/gathering activities during the previous three month period	186
Table 6.1a.	Total sample screened at each three month period	191
Table 6.1b.	Characteristics of sample screened at each three month period	192
Table 6.1c.	Characteristics of sub-sample screened at June 1989 and June 1990	192
Table 6.1d.	Characteristics of sub-sample screened at every three month period	192
Table 6.2.	Repeated intra-batch measures of anthropometric and biochemical parameters	193
Table 6.3.	Repeated inter-batch measures of metabolic parameters	194
Table 6.4a.	Intra- and inter-individual variance of key variables	197
Table 6.4b.	Intra- and inter-individual variance of additional variables	198
Table 6.5.	Anthropometric and metabolic data: pre-intervention (June 1989) and post-intervention (June 1990)	200
Table 6.6.	Anthropometric and metabolic data: pre-intervention (June 1989) and post-intervention (June 1990) (excluding diabetics)	201
Table 6.7.	Anthropometric and metabolic data for those participating in any survey	202
Table 6.8.	Anthropometric and metabolic data for all those participating in any survey (excluding diabetics)	203
Table 6.9.	Anthropometric and metabolic data for those participating in every survey	204
Table 6.10.	Anthropometric and metabolic data for those participating in all surveys (excluding diabetics)	205
Table 6.11.	Prevalence of impaired glucose tolerance by age and sex group June 1989	209
Table 6.12.	Body mass index by glucose tolerance group	210
Table 6.13.	Ratio of waist to hip circumference by glucose tolerance group	211
Table 6.14.	Fasting serum triglyceride concentration by glucose tolerance group	211
Table 6.15.	Fasting serum fructosamine concentration by glucose tolerance group	211
Table 6.16.	Fasting serum insulin concentration by glucose tolerance group	212
Table 6.17.	Two hour serum insulin concentration by glucose tolerance group	212
Table 6.18.	Folic acid status and haematological data for those participating in any survey	224
Table 6.19.	Folic acid status and haematological data for those participating in all surveys	225

Table 6.20.	Folate status and haematological data: pre-intervention (June 1989) and post-intervention (June 1990)	226
Table 6.21.	The relationship between serum and red blood cell folate concentration	228
Table 6.22.	The relationship between number of cigarettes smoked per day and serum folate concentration	233
Table 6.23.	Vitamin data for those participating in any survey	237
Table 6.24.	Vitamin data for those participating in every survey	238
Table 6.25.	Vitamin data: pre-intervention (June 1989) and post-intervention (June 1990)	239
Table 6.26.	The relationship between serum and red blood cell thiamine concentration	243
Table 6.27.	Correlation coefficients between selected biological indicators of vitamin status	246
Table 7.1.	Analysis of intra-individual variance of BMI (excluding diabetics)	254
Table 7.2.	Analysis of intra-individual variance of BMI (including diabetics)	255
Table 7.3.	Analysis of intra-individual variance of diastolic blood pressure	256
Table 7.4.	Analysis of intra-individual variance of systolic blood pressure	258
Table 7.5.	Analysis of intra-individual variance of serum cholesterol concentration (including dietary intake of fatty acids)	260
Table 7.6.	Analysis of intra-individual variance of serum cholesterol concentration over the intervention period (including intake of dietary fibre	262
Table 7.7.	Analysis of intra-individual variance of red blood cell folate concentration	266
Table 7.8.	Analysis of intra-individual variance of serum folate concentration	268
Table 7.9.	Analysis of intra-individual variance of serum vitamin B ₆ concentration	269
Table 7.10.	Analysis of intra-individual variance of serum vitamin B ₁₂ concentration	271
Table 7.11.	Analysis of intra-individual variance of red blood cell thiamine concentration	273
Table 7.12.	Analysis of intra-individual variance of serum thiamine concentration	274
Table 7.13.	Analysis of intra-individual variance of plasma ascorbic acid concentration	275
Table 7.14.	Analysis of intra-individual variance of plasma β -carotene concentration	277
Table 7.15.	Analysis of intra-individual variance of plasma α -tocopherol concentration	279
Table 7.16.	Mean turnover of some specific foods affected by season	281
Table 7.17.	Mean turnover of some specific foods not affected by season period	282
Table 7.18.	Summary of the relationship between change in biological variables and dietary intake as measured by the store-turnover method	288
Table A2.1a	Minjilang population, determined by three monthly census	337
Table A2.1a	Minjilang population, determined fortnightly	337
Table A2.1a	Control community population	337
Table A2.2a.	Recommended nutrient density for age/sex groups	338
Table A2.2b.	Recommended nutrient density, Minjilang	338
Table A2.3a.	Traditional vegetable foods at Croker Island	339
Table A2.3b.	Traditional animal foods at Croker Island	340
Table A2.4.	Fatty acid analysis of some traditional foods used at Minjilang	341
Table A2.5.	Anthropometric, metabolic and haematological data Minjilang, June 1989	342
Table A2.6.	Anthropometric and metabolic data Minjilang, June 1989 (excluding diabetics)	342
Table A2.7.	Anthropometric, metabolic and haematological data Minjilang, June 1990	343
Table A2.8.	Anthropometric and metabolic data Minjilang, June 1990 (excluding diabetics)	343
Table A2.9.	Anthropometric, biochemical and haematological data by age and sex groups pre-intervention (June 1989) and post-intervention (June 1990)	344
Table A2.10.	Analysis of inter-individual variance of BMI (excluding diabetics)	347
Table A2.11.	Analysis of inter-individual variance of diastolic blood pressure	347
Table A2.12.	Analysis of inter-individual variance of systolic blood pressure	348
Table A2.13.	Analysis of inter-individual variance of total serum cholesterol concentration	348
Table A2.14.	Analysis of inter-individual variance of total serum cholesterol concentration (including fasting serum triglyceride concentration)	349
Table A2.15.	Analysis of inter-individual variance of HDL-cholesterol concentration	349
Table A2.16.	Analysis of inter-individual variance of HDL-cholesterol concentration (including fasting serum triglyceride concentration)	350
Table A2.17.	Analysis of inter-individual variance of serum triglyceride concentration (excluding diabetics)	351
Table A2.18.	Analysis of inter-individual variance of serum triglyceride concentration (including serum cholesterol concentrations; excluding diabetics)	351
Table A2.19.	Analysis of inter-individual variance of two-hour plasma glucose concentration (excluding diabetics)	352
Table A2.20.	Analysis of inter-individual variance of serum γ gt concentration	353
Table A2.21.	Analysis of inter-individual variance of red blood cell folate concentration	353
Table A2.22.	Analysis of inter-individual variance of serum folate concentration	354
Table A2.23.	Analysis of inter-individual variance of haemoglobin concentration	354

Table A2.24.	Analysis of inter-individual variance of mean cell volume	355
Table A2.25.	Analysis of inter-individual variance of haematocrit	356
Table A2.26.	Analysis of inter-individual variance of red cell distribution width	357
Table A2.27.	Analysis of inter-individual variance of serum ferritin concentration	358
Table A2.28.	Analysis of inter-individual variance of serum vitamin B ₆ concentration	358
Table A2.29.	Analysis of inter-individual variance of serum vitamin B ₁₂ concentration	359
Table A2.30.	Analysis of inter-individual variance of serum thiamine concentration	359
Table A2.31.	Analysis of inter-individual variance of red blood cell thiamine concentration	360
Table A2.32.	Analysis of inter-individual variance of plasma ascorbic acid concentration	360
Table A2.33.	Analysis of inter-individual variance of plasma retinol concentration	361
Table A2.34.	Analysis of inter-individual variance of plasma α -tocopherol concentration	361
Table A2.35.	Analysis of inter-individual variance of plasma β -carotene concentration	362
Table A2.36.	Analysis of intra-individual variance of haemoglobin concentration	362
Table A2.37.	Analysis of intra-individual variance of mean cell volume	363
Table A2.38.	Analysis of intra-individual variance of haematocrit	363
Table A2.39.	Analysis of intra-individual variance of red cell distribution width	364

LIST OF FIGURES

Figure 3.1.	Contribution of macronutrients to total energy intake, individual Aboriginal communities	97
Figure 3.2.	Contribution of macronutrients to total energy intake, Aboriginal communities combined and apparent consumption data for the wider Australian community	97
Figure 3.3.	The apparent per capita daily intake of nutrients in Aboriginal communities	100
Figure 3.4.	Foods contributing to total energy intake, Aboriginal communities	101
Figure 3.5.	Foods contributing to total fat intake, Aboriginal communities	101
Figure 3.6.	Foods contributing to total intake of sugars, Aboriginal communities	102
Figure 3.7.	Foods contributing to intake of dietary fibre, Aboriginal communities	103
Figure 4.1.	Location and Map of Croker Island	113
Figure 4.2.	Rainfall, humidity and temperature graphs, Croker Island	115
Figure 4.3.	Traditional Iwadja Calendar	116
Figure 5.1.	The total weight of fruit and vegetables supplied during the previous three months from June 1986 to June 1990	142
Figure 5.2.	The total weight of sugar supplied during the previous three months from June 1986 to June 1990	144
Figure 5.3.	The total weight of bread supplied during the previous three months from June 1986 to June 1990	146
Figure 5.4.	The total weight of flour supplied during the previous three months from June 1986 to June 1990	148
Figure 5.5.	The total weight of 'take-away' foods supplied during the previous three months from June 1986 to June 1990	150
Figure 5.6.	The total weight of meats supplied during the previous three months from June 1986 to June 1990	152
Figure 5.7.	The total volume of beverages supplied during the previous three months from June 1986 to June 1990	154
Figure 5.8.	The total weight of fats and oils supplied during the previous three months from June 1986 to June 1990	156
Figure 5.9.	Change in nutrient density over the intervention period, Minjilang	157
Figure 5.10.	Percentage of total energy derived from sugars during the previous three month period from June 1986 to June 1990	163
Figure 5.11.	Percentage of total energy derived from fat during the previous three month period from June 1986 to June 1990	163
Figure 5.12.	Percentage of total energy derived from protein during the previous three month period from June 1986 to June 1990	164
Figure 5.13.	Percentage of total energy intake derived from complex carbohydrate during the previous three month period from June 1986 to June 1990	164
Figure 5.14.	Percentage of total energy derived from saturated fat during the previous three month period from June 1986 to June 1990	165
Figure 5.15.	The ratio of polyunsaturated to saturated fatty acids during the previous three month period from June 1986 to June 1990	165
Figure 5.16.	Density of folate intake during the previous three month period from June 1986 to June 1990	170

Figure 5.17.	Density of thiamine intake during the previous three month period from June 1986 to June 1990	171
Figure 5.18.	Density of niacin intake during the previous three month period from June 1986 to June 1990	172
Figure 5.19.	Density of riboflavin intake during the previous three month period from June 1986 to June 1990	172
Figure 5.20.	Density of vitamin B ₆ intake during the previous three month period from June 1986 to June 1990	173
Figure 5.21.	Density of vitamin B ₁₂ intake during the previous three month period from June 1986 to June 1990	175
Figure 5.22.	Density of ascorbic acid intake during the previous three month period from June 1986 to June 1990	176
Figure 5.23.	Density of retinol intake during the previous three month period from June 1986 to June 1990	177
Figure 5.24.	Density of β -carotene intake during the previous three month period from June 1986 to June 1990	178
Figure 5.25.	Density of intake of vitamin A equivalents during the previous three month period from June 1986 to June 1990	179
Figure 5.26.	Density of intake of vitamin E during the previous three month period from June 1986 to June 1990	180
Figure 5.27.	Density of iron intake during the previous three month period from June 1986 to June 1990	181
Figure 5.28.	Density of zinc intake during the previous three month period from June 1986 to June 1990	181
Figure 5.29.	Density of calcium intake during the previous three month period from June 1986 to June 1990	182
Figure 5.30.	Density of dietary fibre during the previous three month period from June 1986 to June 1990	183
Figure 5.31.	Density of dietary cholesterol during the previous three month period from June 1986 to June 1990	184
Figure 6.1.	Distribution of body mass index, pre-intervention (June 1989) and post-intervention (June 1990)	207
Figure 6.2.	Distribution of serum cholesterol concentration	215
Figure 6.3.	Distribution of serum γ gt concentration	217
Figure 6.4.	Distribution of red blood cell folate concentration	227
Figure 6.5.	Distribution of serum folate concentration	227
Figure 6.6.	Distribution of serum thiamine concentration	240
Figure 6.7.	Distribution of red blood cell thiamine concentration	240
Figure 6.8.	Distribution of serum vitamin B ₁₂ concentration	241
Figure 6.9.	Distribution of serum pyridoxal concentration	242
Figure 6.10.	Distribution of plasma ascorbic acid concentration	244
Figure 7.1.	Percentage of energy intake derived from saturated fat and the mean serum cholesterol concentration over the intervention period	260
Figure 7.2.	The inverse ratio of polyunsaturated to saturated fatty acids and the mean serum cholesterol concentration over the intervention period	261
Figure 7.3.	The inverse density of dietary fibre intake and the mean serum cholesterol concentration over the intervention period	262
Figure 7.4.	Density of folate intake and mean red blood cell folate concentration over the intervention period	267
Figure 7.5.	Vitamin B ₆ intake and mean serum vitamin B ₆ concentration over the intervention period	270
Figure 7.6.	Dietary ascorbic acid intake and mean plasma ascorbic acid concentration over the intervention period	276
Figure 7.7.	Dietary β -carotene intake and mean plasma β -carotene concentration over the intervention period	277
Figure 7.8.	Dietary α -tocopherol intake and mean plasma α -tocopherol concentration over the intervention period	280
Figure 7.9.	Density of folate and ascorbic acid intake at Minjilang and the control community when managed by store manager 'A' and store manager 'B'	285
Figure 7.10.	Density of β -carotene and thiamine intake at Minjilang and the control community when managed by store manager 'A' and store manager 'B'	285
Figure 7.11.	Density of dietary fibre intake at Minjilang and the control community when managed by store manager 'A' and store manager 'B'	286

THESIS ORGANISATION

The thesis is organised within eight chapters. Each is preceded by a brief summary. Where relevant the major points arising from each chapter are included as a conclusion.

Chapter one presents the aims of the study and reviews the relevant literature. The review initially considers the social, cultural, historical, economic and biological factors which contribute to the generally poor nutritional status of contemporary Aboriginal groups and which must be addressed in order to develop effective nutrition intervention programs. Dietary survey methodology is reviewed in order to investigate potential techniques for application in remote Aboriginal communities. Finally, past attempts to improve the nutritional health of remote Aboriginal communities are appraised in order to highlight useful approaches and potential intervention strategies.

Chapter two comprises a brief report of pilot studies which trialled five dietary methods in two remote, centralised Aboriginal communities. The methods are appraised according to their practicality, acceptability and face validity. Where quantitative data were produced, congruent validity of the methods is also presented.

The third chapter further considers one of these methods (the store-turnover method) as applied in six remote, centralised communities; potential implications for community-based nutrition intervention programs are also highlighted.

Chapters four to seven deal specifically with a community-based nutrition intervention project, in which the store-turnover method was exercised during all phases. Chapter four outlines the methods applied in the initiation, planning, implementation and evaluation of the project at Minjilang, a remote, centralised Aboriginal community in northern Australia.

Chapter five presents results of the apparent food and nutrient intake at Minjilang and the control community as measured by the store-turnover method. The qualitative intake of traditional foods at Minjilang from June 1989 to June 1990 is also considered. Due to the complex nature of the relationship between variables, it was necessary to comment periodically on results before presenting subsequent sections in both chapters five and six.

Chapter six presents the results of the biological measurements assessed at three month intervals throughout the intervention period at Minjilang. Findings are presented in three specific clusters; anthropometric and metabolic data, haematological data (incorporating folate data) and other vitamin data.

Chapter seven presents the results of validation of the store-turnover method for application in remote, centralised Aboriginal communities. The method is congruently validated against biological measurement of nutritional status over the intervention period

(that is, by analysis of intra-individual variance investigating the relationship between change in nutrient intake and change in biological parameters). Aspects of the face validity of the store-turnover method are also considered in that chapter.

Chapter eight provides a broader interpretation of all phases of the successful community-based nutrition intervention project at Minjilang including the role of the store-turnover method and details of the intervention strategies applied. This final chapter also considers developments arising from the project, implications for other community-based Aboriginal nutrition projects and further research questions.

Additional detailed data, analysis and information not central to the major thesis are presented in four Addendum. The bibliography is presented in the final section.

GLOSSARY

Aboriginal	Person of Aboriginal decent who identifies as an Aboriginal and is accepted as such by the community in which he (she) lives
Bush food	Traditional food procured by Aboriginal people from the land and sea
Centralised community	A relatively large community which tends to have community buildings/services located in a hub surrounded by a residential area
Damper	Unleavened bread usually cooked in the ashes of an open fire
'Dry' community	An Aboriginal community which has elected to restrict the availability of alcohol within a proscribed surrounding area under the provisions of the <i>NT Liquor Act, 1979</i>
Dry season	Months of minimal rainfall in tropical Australia, usually extending from May to October
Outstation	Traditional homeland community
per capita	per head; per man, woman, child
Production team	Group of people working together in order to procure bush foods
Remote	Aboriginal and non-Aboriginal people may consider remoteness in different ways. Communities within the tribal country, even when affected by physical distance from the main non-Aboriginal population and industry centres, are not generally considered 'remote' by Aboriginal inhabitants
Snack food	Purchased food including extruded snacks, potato crisps, corn chips
Store food	Non-traditional Aboriginal food usually purchased from the local community store
Take-away food	Purchased food requiring minimal preparation before consumption, including meat pies, pasty, sausage rolls, chicko rolls (usually high in saturated fat)
Top End	Northern (tropical) region of the Northern Territory, Australia
Wet season	Months of monsoon rainfall in tropical Australia, generally extending from October to May

LIST OF ABBREVIATIONS, ACRONYMS AND SYMBOLS

α	Alpha
ABS	Australian Bureau of Statistics
ACN	Advisory Council on Nutrition (Australia)
AGPS	Australian Government Printing Service
AHMAC	Australian Health Ministers Advisory Council
AIAS	Australian Institute of Aboriginal Studies
ALPA	Arnhem Land Progress Association
anon	anonymous
ANU	Australian National University
ANZAAS	Australian and New Zealand Association for the Advancement of Science
ASAP	As soon as possible
ATSIC	Aboriginal and Torres Strait Islander Commission
β	Beta
BMJ	British Medical Journal
BMR	Basal metabolic rate
Btwn.Grp.	Between groups
CAAC	Central Australian Aboriginal Congress
Cal	kilocalorie
CDEP	Community Development Employment Project
CDH	Commonwealth Department of Health (Australia)
CDCSH	Commonwealth Department of Community Services and Health (Australia)
CHO	Carbohydrate
CPI	Consumer Price Index
CSIRO	Commonwealth Scientific and Industrial Research Organisation
DAA	Department of Aboriginal Affairs
DCD	Department of Community Development (NT)
FAO	Food and Agricultural Organisation (United Nations)
FFQ	Food frequency questionnaire
γ GT	gamma-glutamyl transferase
g	gram

GLIM	Generalised linear interactive modelling
HDL	High density lipoprotein
Hg	mercury
HPLC	High pressure liquid chromatography
hr	hour
IAD	Institute of Aboriginal Development, Alice Springs
ICD	International Classification of Disease
IGT	Impaired glucose tolerance
IHD	Ischaemic heart disease
IU	International units
kJ	kilojoule
l	litre
LDL	Low density lipoprotein
LFT	Liver function tests
LRCF	Lipid Research Clinic Program
MCV	Mean cell volume
mg	milligram
ml	millilitre
MLIC	Metropolitan Life Insurance Company
mmol	millimole
MRC	Medical Research Council (United Kingdom)
MRFIT	Multi-risk factor intervention trial
ms	mean square
MSHR	Menzies School of Health Research (Darwin)
MUP	Melbourne University Press
NAHS	National Aboriginal Health Strategy
NARU	Northern Australia Research Unit of the Australian National University
NCHS	National Centre for Health Statistics
NHMRC	National Health and Medical Research Council (Australia)
NHF	National Heart Foundation
NIDDM	Non-insulin dependant diabetes mellitus
NLC	Northern Land Council

NRC	National Research Council (United States)
n/a	Not applicable
ns	Not statistically significant
NT	Northern Territory (Australia)
NTHD	Northern Territory Health Department
NTDHCS	Northern Territory Department of Health and Community Services
PNHHS	Pitjantjatjara/Ngaanytjara Homeland Health Service
Pl.	Plasma
RACO	Royal Australian College of Ophthalmologists
RBC	Red blood cell
RDI	Recommended Dietary Intake
RDH	Royal Darwin Hospital
RDW	Red cell distribution width
S.	serum
sd	standard deviation
se	standard error
ss	sum of squares
sq.	square
SWFA	Standard weight for age
TCA	Trichloroacetic acid
μg	Microgram
UPK	Uwankara Palyanyku Kanyintjaku (An environmental and public health review within the Anangu Pitjantjatjara lands)
USDHEW	United States Department of Health, Education and Welfare
USDHHS	United States Department of Health and Human Services
UQP	University of Queensland Press
Vit.	Vitamin
Vitamin A eq.	Vitamin A equivalents
VLDL	Very low density lipoprotein
WHO	World Health Organisation
°	Degrees
\geq	Greater than or equal to
\leq	Less than or equal to

CHAPTER 1: ABORIGINAL NUTRITIONAL STATUS, DIETARY SURVEY METHODOLOGY AND NUTRITION INTERVENTION

This chapter presents the aims of the study and reviews the relevant literature. The review initially considers the social, cultural, historical, economic and biological factors which contribute to the generally poor nutritional status of contemporary Aboriginal groups and which must be addressed in order to develop effective nutrition intervention programs. Dietary survey methodology is reviewed in order to investigate potential techniques for application in remote Aboriginal communities. Finally, past attempts to improve the nutritional health of remote Aboriginal communities are appraised in order to highlight useful approaches and potential intervention strategies.

1.1. Introduction and aim of study

1.1.1. Background

European invasion of the Australian continent 200 years ago heralded major dietary and lifestyle changes for Aboriginal people. The transition from a nomadic lifestyle incorporating a varied, nutrient-dense diet, high in fibre and low in fat and refined carbohydrate, to a settled existence with poor hygiene and an energy-dense, high fat, high sugar diet, has contributed to the deterioration of Aboriginal health. It has been argued that increased attention to nutritional-related diseases and their origins should play a greater part in strategies aimed at improving the health of Aboriginal people.

Although the high prevalence of nutrition-related problems has been frequently described, little attention has been paid to the quantitative measurement of dietary intake in Aboriginal communities. Even less attention has been paid to formal projects aimed at improving the nutritional health of Aboriginal people or to appropriate methods to evaluate such projects.

1.1.2. Aim

The long term aim of this study was to help reduce the high prevalence of nutrition-related health disorders experienced by Aboriginal Australians living in remote communities.

Specific objectives were:

- 1: To develop a method (the 'store-turnover' method) for the quantitative measurement of dietary intake in remote centralised Aboriginal communities
- 2: To validate the store-turnover method by:
 - a) congruent (relative) validation against biological indicators of nutritional status
 - b) consideration of face validity
- 3: To apply the store-turnover method in the measurement of dietary intake in several communities in order to:
 - a) compare dietary intake in different regions
 - b) identify practical nutrition intervention strategies
- 4: To use the store-turnover method as a rational basis for the development of a community-based nutrition intervention project
- 5: To use the store-turnover method to evaluate the effectiveness of the community-based nutrition intervention project at an outcome level

1.2. Traditional Aboriginal lifestyle, nutrition and health

It is believed that Aboriginal ancestors migrated to Australia from South-East Asia up to 50,000 years ago (White and O'Connell, 1979). Aboriginal groups from the more fertile and hospitable regions were driven from their land in the early years of European settlement and Aboriginal society was generally obliterated in agricultural areas (Basedow, 1932; Barwick, 1971; Jones, 1971; Reynolds, 1982). Little reliable information is available about the traditional lifestyle, health or nutrition of Aboriginal populations from these areas (Smyth, 1878; Curr, 1886, 1887; Flood, 1974; Jones, 1977; Woodward *et al*, 1986). Large-scale European economic activities were only introduced into the Northern Territory early this century; consequently traditional values have been relatively maintained amongst Aboriginal groups in these regions. However, information about traditional subsistence adaptation and lifestyle remains fragmentary (Peterson, 1978). Ethnographic sources include written records of those Europeans who first contacted

Aboriginal society, such as early explorers (Eyre, 1845; Grey, 1841; Leichhardt, 1847; Lindsay, 1884, 1890; Giles, 1889; Carnegie, 1898; Sturt, 1969), early settlers, including mission and government representatives (Chaseling, 1957; Kyle-Little, 1957; Long, 1971), anthropological observers (Smyth, 1878; Curr, 1886, 1887; Spencer and Gillen, 1899, 1904; Spencer, 1914, 1928; Tindale, 1925; Thompson, 1939, 1962) and a small number of medical observers (Breindl, 1912; Holmes, 1913; Basedow, 1932; Elphinstone, 1971). The need for cautious interpretation of early records has been pointed out as the degree of "*judgement, generalisation or abstraction*" of facts is unknown (Malinowski, 1913:19; Rose, 1987:4).

1.2.1. Traditional lifestyle

People lived a nomadic lifestyle imposed by the constant necessity to seek food and, in the drier regions of the continent, water. Strong religious beliefs were expressed through ritual practices, myths and deep spiritual and emotional attachment to the land. Within each region there was a marked seasonal diversity in lifestyle, dependent on availability of water, foods, restriction of movement and ceremonial life (Birdsell, 1953; Abbie, 1960; Strehlow, 1965; Lawrence, 1971; Long, 1971; Maddock, 1972:22-3; Tindale, 1974; Jones, 1980; Rose, 1987:49, 191). Nomadic lifestyle ensured that no campsite was occupied long enough to become fouled (Abbie, 1960). Little value was placed on the accumulation of material possessions (Spencer and Gillen, 1969:30; Lawrence, 1971; Long, 1971; Mulvaney and Golson, 1971:375). Several extensive, detailed reviews of Aboriginal social structure are to be found (Hiatt, 1968; Maddock, 1972; Elkin, 1974; Yengoyan, 1976; Berndt and Berndt, 1977). All emphasise the importance of kinship relationships to survival.

There is evidence of dynamic innovation and adaptation in traditional Aboriginal lifestyle (Lawrence, 1971; Blainey, 1982:118) which is illustrated by the diverse methods of food production (Roth, 1897; Basedow, 1925:129; McCarthy, 1957:47; Campbell, 1965; Jones, 1969; Latz and Griffin, 1978; Latz, 1982). The most important resource was a knowledge of land, plants and animals. Effective education systems consisting of intensive and rigorous training transmitted knowledge from one generation to the next (Penny and Moriarty, 1978). Teaching methods were contextual, informal, holistic and practical (Harris, 1977a, 1977b).

1.2.2. Traditional Aboriginal diet

"In taking a general survey of the blackfellow and of the life he leads, one cannot do other than devote much attention to matters of food and food-getting, since these play so important a part in his affairs.." (Finlayson, 1935:87).

1.2.2.1. Sources of information

Several early studies concentrated on Aboriginal food and food habits (Roth, 1897; Noetling, 1910; White, 1915; Basedow, 1925:120-154; Tindale, 1925; Daley, 1931; Cleland and Johnson, 1933, 1937; Campbell, 1939; Hyam, 1939, 1940; Cleland, 1957). Where direct observation has been impossible, records and diaries of early explorers, settlers and government officials, supplemented by archaeological evidence, have been used to reconstruct a picture of pre-contact Aboriginal diet (Hiatt, 1967; Lawrence, 1971). Unfortunately there is no accurate quantitative account of the diet of Aborigines living wholly on a traditional diet and following a traditional lifestyle (McArthur, 1960c; Barnes, 1963:200-201; Rose, 1987:16,172; White, 1985).

1.2.2.2. Qualitative diet

Extensive lists of botanical and/or zoological foods utilised in various environments have been produced (Cleland and Johnston, 1937, 1938; Irvine, 1957; Meggitt, 1957; Specht, 1958a; Worsley, 1961; Gould, 1969; Lawrence, 1969; Golson, 1971; Silberbauer, 1971; Peterson, 1973; Cribb and Cribb, 1976; Levitt, 1981). However the relative contribution of individual food sources is difficult to determine from these records (Peterson, 1978). Although seasonal fluctuations in food supply were common in all regions and greatly influenced foraging patterns, most observers describe a varied and ample range of available foods (Cleland and Johnston, 1933; Campbell, 1939a, 1939c; Strehlow, 1965; Marbutt, 1971; Gould, 1980).

A statement that the central Australian desert diet was comprised of "70 - 80% *plant foods*" (Meggitt, 1957), has been widely accepted (Gould, 1969; Peterson, 1973, 1978; Thompson, 1975; Meehan, 1982:150; Bell, 1983; Rose, 1984:56). However, this report was not substantiated by quantitative data and was probably over-estimated as foods were compared on the basis of their contribution to the total weight of the diet. Recent evidence

suggests that stereotyping of the sexual division of labour may have contributed to a de-emphasis of the hunting of animal foods, such as small marsupials, shellfish, reptiles and insects by women (Meehan, 1980:17; Altman, 1982, 1984; Devitt, 1988).

There is now increasing evidence that both tropical savanna/coastal (McArthur, 1960c; Jones and Bowler, 1980:19-22; Meehan, 1982) and desert diets (Lockwood, 1964; Hamilton, 1980; Devitt, 1988) were meat-orientated. Vegetable foods provided an important supplement, rather than an alternative, to animal foods with proportions changing throughout the year (Finlayson, 1935:77-83; Campbell, 1939a:13,15; Cleland, 1966:118; Lawrence, 1971:255; Tindale, 1974; Jones, 1980:135-136).

The seasonal diversity of diet was particularly marked in the relatively richer (Calaby, 1971) areas of tropical Australia (Spencer, 1914; Tindale, 1925; Thompson, 1939, 1948, 1949a, 1949b, 1949c; Warner, 1958; McArthur, 1960a, 1960c; Berndt and Berndt, 1970; Peterson, 1973; Carrol, 1979; Levitt, 1981:23-25; Meehan, 1982; Rae *et al*, 1982; White, 1985). The early dry season (April to July) was the time of greatest abundance and the peak of the wet season (January to March) the most lean (Basedow, 1925:133; Thompson, 1949a; Specht, 1958b; Warner, 1958:138; Scarlett, 1976; White, 1985; Brock, 1988).

1.2.2.3. Nutritional analysis of traditional foods

The first call to evaluate the nutritional adequacy of Aboriginal traditional diet was documented in 1895 (Maiden, 1895). Limited analysis of indigenous foods followed (Dadswell, 1934; Morrison and Penfold, 1952; Fysh *et al*, 1960; Elphinstone, 1971; Peterson, 1977a; Jones and Hegarty, 1981; Williams *et al*, 1981; Rivett *et al*, 1983). More extensive analysis of traditional foods has more recently been conducted (Maggiore, 1976; Brand *et al*, 1982a, 1982b, 1983, 1985; James *et al*, 1982, 1984, 1986; James, 1983; Brand and Chirikoff, 1985; Chirikoff *et al*, 1985; Naughton *et al*, 1986, Brown *et al*, 1987).

The vegetable foods in the diet were typical of uncultivated plants world-wide, being high in fibre and relatively high in protein with a generally low energy density. The carbohydrate in most bush foods is slowly digested and absorbed, producing lower glucose and insulin levels following oral ingestion than similar western foods and may have been protective against diabetes (Thorburn *et al*, 1987a, 1987b). Most land animals are very lean, with traditional meat foods having a much lower carcass fat content and

intramuscular lipid content than meat from domesticated animals, such as cattle and sheep (Naughton *et al*, 1986). Although lipid levels in the muscle of wild animals tend to be low irrespective of season, some animals have varying carcass fat depending on season. Most of this carcass fat is stored in discrete depots within the abdomen. These fat depots tend to be small and were traditionally shared by many people (O'Dea, 1987). Tropical seafood has been shown to contain both ω -6 and ω -3 polyunsaturated fatty acids (O'Dea and Sinclair, 1982). Coastal animals with a high carcass fat content, such as dugong and turtle, have not yet been analysed for fatty acid composition. Muscle lipid of land animals is primarily structural (phospholipid and cholesterol) and is relatively rich in highly polyunsaturated long-chain fatty acids of both the ω -3 and ω -6 families, and provides a low energy density of about four kJ/g (Naughton *et al*, 1986). Available data suggest that even when the traditional Aboriginal diet contained a high proportion of animal foods, it would have been low in total fat, extremely low in saturated fat and relatively high in polyunsaturated fatty acids including the long-chain highly polyunsaturated fatty acids of both the ω -3 and ω -6 families, and hence protective against cardiovascular disease and related conditions (Naughton *et al*, 1986).

On the basis of available evidence the traditional diet generally had a low energy density but high nutrient density. A balance was established between minimising effort required to procure food and maximising the certainty of a regular, high-quality food supply.

1.2.2.4. Traditional food practices

It is likely that the beliefs and attitudes associated with some food practices, such as preparation and cooking, meal timing, food distribution and taboos may continue to influence dietary practices in traditionally-orientated communities. These factors should be considered in community-based nutrition programs. As such a discussion of past food practices may provide insight into some contemporary observations.

Meat was only lightly cooked and usually eaten at one sitting; if food was plentiful, as when large game was caught, people tended to rapidly consume extremely large quantities (Finlayson, 1935:70; Campbell, 1939b:52; Meehan, 1982:151; Wadsworth, 1984). Methods of cooking animals were usually prescribed by regional traditional Law (Basedow, 1925:108; Finlayson, 1935:83; Campbell, 1939b:48-50; Gould, 1967). Most plant foods were eaten raw or lightly roasted (Tindale, 1925; Campbell, 1939b:47-49; McArthur, 1960c:103,110-112; Levitt, 1981:48-51). There was no overcooking, no

leaching of nutrients, little storage and very little wastage (Campbell, 1939a:13; Wilson, 1954; McArthur, 1960b, 1960c:115-118; Naughton *et al*, 1986). Therefore food preparation practices were presumed to have retained maximum nutritional value (Campbell, 1939a, 1939b; Sweeney, 1947; McArthur, 1960b).

Foods were proportioned and distributed according to traditional Law; strict cultural practices determined by kin obligations (Tindale, 1925:83; Bourne, 1953; Chaseling, 1957:38; Gould, 1967; Berndt and Berndt, 1970:44; Elphinstone, 1971; Penny and Moriarty, 1978; Meehan, 1982:137-138; White, 1985; Reid, 1986). In comparison to the European perspective of 'physiological metabolism', the phrase 'social metabolism' has been used to describe the food perspective of Aboriginal people (Stacy, 1975). "*The giving and receiving of food is perhaps the most constant and explicit means of expressing social relationships in Aboriginal society*" (Reid, 1986).

There is evidence that the older men had greater access to protein and delicacies such as liver and fat and the old women received the smallest portions (Chaseling, 1957:38; Meehan, 1982:151; White, 1985). Children were breast-fed until approximately 3 years of age; the age of weaning depending on the arrival of another sibling (McArthur, 1960c:123; Hamilton, 1971, 1981:68-69, 1982; Cowlshaw, 1981). Solids were introduced to toddlers on eruption of teeth (McArthur, 1960c:123; Hamilton, 1981:52, 1982). Responsibility for feeding tended to rest with the child, who was expected to indicate desire for food and was fed on demand (Middleton and Francis, 1976:86; Hamilton, 1982:66). Older children had priority over the feeding of small infants (Hamilton, 1981:81, 1982).

Various food-related taboos have been reported which may have had the potential to influence nutritional status of some sections of traditional society (Spencer and Gillen, 1899; Spencer, 1914; Chaseling, 1957:38; McArthur, 1960c:124-125, Spencer, 1969:472; Berndt and Berndt, 1970:49; Akerman, 1975; Middleton and Francis, 1976:82).

1.2.3. Traditional Aboriginal attitudes towards food

1.2.3.1. Traditional dietary preferences

"Men and women shared a preference for meat, fat and honey" (Devitt, 1988:125).

Ethnographic evidence suggested that conservative dietary preferences affected traditional food-related attitudes, behaviour and diet (Golson, 1971; Mulvaney, 1975:58; Blainey, 1982:15-24). It has been suggested that "*peoples in all cultures are conservative in their food habits but hunger can cause a radical reversion of these ideas*" (Calaby, 1971:89). Southern migration of Aboriginal ancestors through the Australian continent would have necessitated dietary adaptations to unfamiliar species; the process of "*eat, die, and learn*" (Webb, 1973). Such beliefs were supported by the initial reluctance of desert Aborigines to eat rabbits for some years after these appeared in central Australia (Calaby, 1971).

Meehan described the Anbarra in central Arnhem Land as "*affluent hunters with high gastronomic standards*" (Meehan, 1982:160). "*Freshness of food is important to the Anbarra; in many ways they are fastidious about it. They always smell food that is a few hours old before cooking or eating it*" (Meehan, 1982:160). Meehan notes that food which was acceptable to her was often discarded by the Anbarra as "*too old*" or "*no good*".

It is thought that the strong dietary preference for some animal food (section 1.2.2.2) was largely due to its fat content (Spencer and Gillen, 1899; Meehan, 1982:147). "*In a land where mammals are nearly always lean, the big lizards and the emu are practically the only sources of fat-supply and are cherished accordingly*" (Finlayson, 1935:55). Other favoured foods such as witchetty grubs (*Cossidae sp*) and marine mammals, also have a high fat content (Basedow, 1925:125; Blainey, 1982:135; Cherikoff *et al*, 1985; Rose, 1987:51). More generally "*...people constantly discuss the best time to collect various foods so that the animals will be in the most desirable condition for eating, that is, when they are 'fat'*" (Meehan, 1982:26). There have also been many reports of game being left uneaten when it was considered to contain insufficient fat (Tindale, 1972:248; Devitt, 1988:120).

A "*sweet tooth*" has been described as a "*leading characteristic of both sexes at all ages*" (Finlayson, 1935:85) and many early observers have commented on the dietary preference for sweet foods (Campbell and Lewis, 1926:375; Campbell, 1939a:14; Gould, 1969). Traditional diet was generally low in sugars (Rose, 1987:51). However sweetness was provided by honey ants (*Melophorus inflatus*), the honey of the native bee, blossoms (*Grevillea sp*), lerp (secretion from the insect *Psylla* living on the leaves of *Eucalyptus sp*) and gums. Gillen noted that a favoured drink was produced from the blossoms of *Hakea sp*. soaked in water (McFarlane, 1978). The enthusiastic pursuit of honey has been noted

to be out of all proportion to the small quantities obtained (Finlayson, 1935:86; McArthur, 1960c:110; Devitt, 1988:121).

The need for variety of vegetable and animal foods in the diet was seen to be very important (Berndt and Berndt, 1970:43; Peterson, 1973; Lamilami, 1974:116-117; Middleton and Francis, 1976:81-82; White, 1977:284; Jones and Bowler, 1980:20; Meehan, 1982:141-161; O'Dea, 1984; Waddy, 1986).

1.2.3.2. Traditional classification of food

Rather than a term for 'food' or edible things in general, most traditional Aboriginal groups had a variety of classification terms, with a major division between animal and vegetable foods and, frequently, 'sweet' foods and others (Berndt and Berndt, 1970:34; Hamilton, 1981:53; Bryce, 1986). European foodstuffs have sometimes been incorporated into indigenous classifications (Taylor, 1977).

1.2.3.3. Traditional Aboriginal attitudes to food and health

Aboriginal belief systems generally equate 'health' with social and spiritual well-being, rather than with a physiological perspective (Hamilton, 1971, 1974:19; White, 1977; Soong, 1981:112; Reid, 1982:91). Traditionally, deaths of old people and infants were accepted as inevitable (Stacy, 1977), but others who fell ill were expected to recover (Cleland, 1928; Taylor, 1977). 'Health' is believed to be maintained through observation of kinship responsibilities and religious ceremonies, rather than through adequate nutrition, exercise and the maintenance of a hygienic environment.

Although there appears to be some evidence that traditional lifestyles and consumption of traditional foods are associated with contemporary 'health' (Reid, 1986), the notion of 'nutrition' and the link between food and health, particularly the concepts of 'good' and 'bad' foods, was generally inappropriate in Aboriginal society (Sinclair, 1968; Hamilton, 1981:54). However some reference to concepts of diet and 'health' have been made. For example, emphasis has been placed on the value of meat (Peterson, 1973; Wiminydji and Peile, 1978). Also, traditional myths from tropical coastal regions revealed that warnings about over-consumption of various foods did exist. Over-consumption stories tended to focus on preferred foods, such as fat and sources of sweetness. Several examples are included (Addendum 4) due to their potential use in culturally-appropriate nutrition

intervention programs.

1.2.4. Traditional health and nutritional status

Information about the health and nutritional status of Aboriginal groups with little or no previous contact with European society is sparse and historically disperse. Most past opinions about the adequacy of traditional nutritional status have been subjective; many have been contradictory (Noetling, 1910; Campbell, 1939c:74; Davidson, 1957; McArthur, 1960a; Stanner, 1960:70; Bleakley, 1961:6,17; White, 1977; Peterson, 1978; Cowlshaw, 1981:39; Thomson, 1982). Past misconceptions that Aborigines "*were living always on the borders of starvation*" have been criticised (Rose, 1987:49) as there is considerable evidence to suggest that people were generally in good nutritional health prior to European settlement (Spencer and Gillen, 1899:44; MacPherson, 1903; Basedow, 1904:24, 1925; White, 1915:726; Tindale, 1925:76; Cleland, 1928; Stanner, 1933; Australia, 1979:7). Photographic evidence of well-nourished people living in central and northern Australia between 1894 and 1927 was recorded by Spencer (Vanderwal, 1982). More recent opinions have interpreted the long term maintenance of the gene pool without supplement by migration (Peterson, 1978), the wide variety and abundance of foods consumed and nutrient-conserving "*primitive methods of preparation*" (Campbell, 1939c:87), as support for traditional nutritional adequacy (Wadsworth, 1984:80).

There was no evidence of rickets or other nutritionally-related disease in traditional Aboriginal groups as revealed by review of traditional medicines (Webb, 1969, 1973; Levitt, 1981:32-67; Devanesen and Henshall, 1982; Scarlett *et al*, 1982; NT Aboriginal Communities, 1988), clinical examinations (Breinl, 1912; Basedow, 1913; Holmes, 1913; Breinl and Holmes, 1915; Cleland, 1928, 1929; Hackett, 1936; Black and Cleland, 1938; Davidson, 1957; Mann, 1957; Billington, 1960; Packer, 1961; Abbie, 1966; Hargreaves and Jones, 1980) or palaeopathological studies (Mattingly, 1915; Campbell, 1939e; Sandison, 1973, 1980; Prokopec, 1979). Reports of scurvy have been described amongst nomadic Aborigines forced to seek food at Hermannsburg Mission due to drought in surrounding cattle-degraded areas (Cleland and Fry, 1930). However the condition also existed amongst mission Aborigines at that time and the surrounding environment had changed since European contact. Two cases of probable scurvy were also described in a traditionally-orientated group following a long period of drought (Elphinstone, 1971). No other clinical evidence of nutritional deficiency was observed in either area.

Biochemical nutrition studies were conducted in 1948 in four missions in Arnhem Land (Billington, 1960:27). Results indicated that there was no sign of rickets, scurvy, vitamin A deficiency or thiamine deficiency in recently nomadic people. Angular stomatitis was described particularly in young people at Groote Eylandt and was considered to indicate possible riboflavin deficiency. Similarly, glossitis in four young children at Oenpelli was considered to suggest nicotinic acid deficiency (Billington, 1960:38).

Low birth weights were documented among recently nomadic groups. Growth in the first six months of life paralleled European infants, although there was a reduced velocity of weight gain after four to six months compared with European children (Parsons, 1965; Abbie, 1974). It has been suggested that infants of low weight would advantage nomads, providing lighter burdens for women during the food quest (Hamilton, 1971).

Some observers have described severe loss of subcutaneous fat and muscle tone in some women beyond early adult life, compared with relative maintenance of weight in older men (Chaseling, 1957; Billington, 1960:31,34,36; Abbie, 1968, 1975; White, 1985; Reid and Swain, n.d).

Many early observers noted the lack of obesity amongst traditional groups (Basedow, 1925, 1932; Billington, 1960; Elphinstone, 1971) and were supported by the low BMI calculated using early anthropometric measurements (Abbie, 1971, 1975; Elphinstone, 1971; White, 1985; O'Dea, 1987). The apparent lack of obesity was considered so unusual that early writers ascribed differences between both races to hereditary factors, for example Aborigines must "*not physiologically store much fat*" (Campbell, 1939c:76). It is now believed that physical size of adults is determined more by a combination of adequate nutrition and freedom from infection in early life than by inheritance, while genetic factors affect relative dimensions and body build (Moodie, 1973:179; Abbie, 1975:91; Coles-Rutishauser, 1987).

Although early anthropometric measurements suggest that some Aboriginal groups, in comparison with other populations, may have experienced lower energy intake relative to energy expenditure, it is not possible to comment on the nutritional consequences of this without reference to biochemical evidence. Unfortunately few biochemical studies of Aboriginal groups with little contact with European society are available. Of these, acceptable serum protein levels were described (Curnow, 1957; Elphinstone, 1971), both blood pressure (Nye, 1937; Casley-Smith, 1959b; Abbie, 1960; Abbie and Schroder,

1960; Van Dongen *et al*, 1962; Woods, 1966; Macfarlane, 1978) and serum cholesterol levels (Schwartz *et al*, 1957; Schwartz and Casley-Smith, 1958a, 1958b, 1959; Charnock *et al*, 1959) were low compared with 'normal' Caucasian reference ranges, haemoglobin (Davis and Pitney, 1957; Davidson, 1957; Casley-Smith, 1958, 1959a, 1960; Abbie, 1960; Elphinstone, 1971) and ascorbic acid levels (Billington, 1960; Hodges, 1960; McArthur, 1960b) were acceptable, cyanocobalamin levels were relatively high (Davis and Pitney, 1957; Pitney, 1962; Elphinstone, 1971; Davis *et al*, 1975) and serum folate levels tended to be higher than in Aborigines living at early settlements and missions (Davis *et al*, 1965, 1975). Due to the limited data, and in order to avoid repetition, details of early biochemical investigations of these parameters are discussed with more contemporary data and relevant findings of the present study (section 8.2).

1.3. A brief history of cross-cultural contact, with particular emphasis on policies affecting Aboriginal health and nutrition in the Northern Territory

In the Northern Territory there is no evidence of any significant disturbance of the basic pattern of Aboriginal life until European settlement (Thompson, 1949c; Warner, 1958; McArthur, 1960a; MacKnight, 1976). The first permanent town, Palmerston, was established on Port Darwin in 1869 and was renamed Darwin in 1911. Early cattle stations were established in the north at Oenpelli (1876) and Coburg Peninsula (1884-1889). In the centre, cattle reached Alice Springs in 1872, but sheep were also widely introduced to Central Australia from these years (Frith, 1978). The first mission was established west of Alice Springs at Hermannsburg in 1877. Others followed in the north from 1882 and at other central Australian locations from 1930. The first state welfare settlement was established west of Alice Springs at Jay Creek in 1937.

The process of general alienation of land and its devastation upon Aboriginal societies has been documented in detail (Rowley, 1970; Reynolds, 1982). Ecological pressure forced Aboriginal people to accept rations from railway sidings and telegraph stations, from pastoralists and miners and from missions; one aspect of a reaction process which has been termed "*intelligent parasitism*" (Elkin, 1951). The Aboriginal population was susceptible to introduced infectious diseases (Basedow, 1932) and, as early as 1914, it was observed of Aboriginal groups that "*..in all parts where they are in contact with outsiders.. they are dying out with great rapidity*" (Spencer, 1914:41). In the first official phase of government intervention in Aboriginal affairs, a policy of protection and preservation was adopted from 1913 to 1960. This policy aimed to segregate Aborigines

by countering the drift to towns by the provision of trading and health facilities in remote regions and the development of Aboriginal reserves. However, introduced resources proved an irresistible attraction and eventually groups were centralised at cattle stations, Government settlements or missions (Long, 1970; Altman, 1980). Factors considered to have encouraged centralisation include the desire for rations, the desire to join groups already centralised and curiosity (Berndt and Berndt, 1942; McArthur, 1960a:5; Meggitt, 1962:27; Long, 1970:319; Rowley, 1970; Hamilton, 1972; Turner, 1974:179; Brokensha and McGuigan, 1977:11; Brady, 1986). Cattle stations became the principal source of employment; employees and their dependents were rationed for payment (Rowse, 1988). In the centre dingo scalps were traded in exchange for money, tea, flour and sugar (Hilliard, 1968:81).

From the 1930s it was clear that the Aboriginal population of the north was no longer dying out and policy became intermingled with assimilationist endeavours. There was a drift of Aborigines to missions and to Darwin from the early 1940s (Rowley, 1970:332). After the war trading posts were established in remote areas to encourage people to remain in their own lands (Kyle-Little, 1957). Some failed due to shortage of staff and funds, but others developed into service centres and eventually settlements. With assimilation adopted as official government policy in 1961 (Rowley, 1971a:399), attempts were made to integrate people into European society through education and example. People were encouraged to centralise and became dependent on welfare handouts or allowances for employment which were set well below award wages. An 'Aboriginal welfare problem' was recognised during this phase prompting an escalation in government intervention and expenditure on Aboriginal education, employment training and enterprise. However there was a lack of appreciation of Aboriginal cultural values and, with the increasing involvement of white administrators, Aboriginal people were deprived of all responsibility for their own lives.

Communal feeding was established in an attempt to improve Aboriginal nutrition (Sinclair, 1977) and "*properly sustain a supervised population*" (Rowse, 1988). However many settlements and missions lacked adequate facilities to feed their total population (Wilson, 1953; Sinclair, 1977). In 1967, the national referendum granted Aborigines the right to be counted in census, the right to vote, the right to drink alcohol and the right to equal pay. The referendum effectively allowed the Federal government to legislate for all Aborigines, facilitating '*monetisation of rationing*' (Rowse, 1988). For the first time Aboriginal people received some social security entitlements. Station and settlement employees received cash

wages, although award wages were not generally introduced until 1969. Standard charges were introduced for meals, settlement stores became more popular and communal dining rooms eventually folded (Middleton and Francis, 1976:87; Young, 1984:5; Rowse, 1988). In the north the introduction of a cash economy theoretically enabled some seasonal freedom of movement and an opportunity for the performance of ceremonies and subsistence activities on traditional lands (Altman, 1987:5). In more arid areas, by the 1970s, many people were subsisting almost entirely on food purchased from settlement stores (Middleton and Francis, 1976:81; Peterson, 1978).

In 1972 a policy of self-determination was introduced, facilitating the gradual return of local political power to Aboriginal people. There were also increased funds for welfare programs. Unemployment benefits replaced training allowances but low incomes generally persisted (Anderson, 1976:1-11; Stanley, 1976; Peterson, 1977b). Aboriginal councils were established to administer settlements, while Aboriginal representative bodies, such as Land Councils and Progress Associations, were also formed, helping establish a new social awareness of Aboriginal identity (Kirk, 1983:198). Opportunity was also provided for community ownership/management of retail stores (Young, 1984:5,9). 'Self-management' became official policy in 1975 and two years later Northern Territory land rights legislation was passed by the Federal coalition government and served to facilitate decentralisation. By 1980 most Aboriginal people in the Northern Territory were economically dependent on the public sector.

1.4. Contemporary Aboriginal living places in the Northern Territory

The food supply of traditionally-orientated and transitional Aboriginal people living in the Northern Territory is directly affected by several aspects of contemporary living areas. Most transitional people continue to reside in relatively large centralised communities far from European centres, although an increasing proportion may dwell at outstations either permanently or temporarily throughout the year, and several groups occupy excisions on cattle-stations. A high degree of population circulation between urban and rural communities has been recorded (ABS, 1990:10,16).

It has been stated that conditions in larger settlements, "*reflect not how things used to be normally but how they were on the infrequent but regular occasions when large numbers of people gathered together*" (Hamilton, 1982:4). This concept led to the definition of centralised communities as "*super-waterholes*" (Hiatt, 1968). Other commentators have

been more critical describing settlements as "*ecologically hazardous concentrations*" (Stanner, 1974:8) and "*total institutions*" (Stanner, 1974:7) characterised "*by poverty, by lack of a stable economic base and by the absence of that sense of community which would engender pride in Aboriginal culture*" (Kirk, 1983:174). In larger settlements friction may be created by many clans living together on one group's country, and it has been argued that clan structures and traditional patterns of authority may be undermined. However it has been demonstrated (Turner, 1974; Altman, 1980) that even at centralised communities where Aboriginal groups are economically dependent on the Government sector, traditional social organisation and religious life continues.

Outstations are small homeland centres, usually in remote areas, which are dependent on basic facilities and linked administratively to a group such as a resource centre or larger community council (Coombs, 1974; Gray, 1977; Coombs *et al*, 1982; Loveday, 1982). Aboriginal rationale for the establishment of outstations included: the desire to maintain a presence on traditional land and its associated sites of spiritual significance and the opportunity for traditional elders to reassert social control over smaller family groups (Coombs, 1978). Potential improvements in health status associated with outstation lifestyle have been frequently raised (Hamilton, 1971; Cawte, 1973; Morice, 1976; Moodie, 1981; Kirk, 1983). However, attention has also been drawn to difficulties if insufficient support is provided (Cutter, 1978).

1.5. Post-contact diet and factors affecting nutritional health

"We had flour, tea, sugar and golden syrup with us. We had enough for two or three days - enough to get us through to OenPELLI" (LamiLami, 1974:121).

1.5.1. Introduction

The rapid shift to a sedentary life on cattle stations, missions and settlements, limited availability of and accessibility to bushfoods, and people were increasingly forced to depend on available European foods (Gould, 1972). This precipitated widespread social, demographic and economic change (Peterson, 1978; Brady, 1986) and had a dramatic effect on the nutritional and health status of Aboriginal people (Kyle-Little, 1957; Macfarlane, 1978; Coyne and Darnton-Hill, 1979; Moodie, 1981; White, 1985; O'Dea, 1988). These changes have been described qualitatively (O'Dea, 1988) and are

summarised in Table 1.1.

Table 1.1. Characteristics of hunter-gatherer and western lifestyle

	hunter-gatherer lifestyle	western lifestyle
Physical activity level	high	low
Diet		
Energy intake	low	high
Protein	high	low-moderate
animal	high	low-moderate
vegetable	very low	low-moderate
Carbohydrate	low (slowly absorbed)	moderate (rapidly absorbed)
sugars	low	high
dietary fibre	high	low
Fat	low	high
vegetable	very low	moderate
animal	low (polyunsaturated)	high (saturated)

1.5.2. The first introduced European foods

The first European foods introduced to Aborigines at ration depots and cattle stations were flour, sugar, tea (Wilson, 1953; Middleton and Francis, 1976:89) and to a lesser extent meat, (fresh, tinned or salted). These foods were also those of 'pioneer Australia' (Walker and Roberts, 1988). The early popularity of these foods may be due to such factors as their relative durability, low bulk, transportability, cheapness and the limited cooking and storage facilities required for their preparation; factors which may remain relevant today.

1.5.3. Transitional diet

Early studies provided a qualitative assessment based on descriptive observation of food availability and intake, supplemented by ration listings available from early mission and settlements (Billington, 1960; Mountford, 1960; McArthur, 1960b). Ration scales varied according to location and time of observation but were generally high in flour and low in

fresh produce. Nutritional analysis indicated that sufficient iron and thiamine were usually provided, but energy and protein content were sometimes low and many diets were inadequate in calcium, vitamin A and vitamin C (Cleland, 1929; Campbell and Barrett, 1953; Wilson, 1953; McArthur, 1960b; Cordon, 1962; Middleton and Francis, 1976:88-91).

1.5.3.1. Contemporary food preferences

Flour, sugar, tea and meat became the confirmed dietary staples (Brokensha, 1975:25; Peterson, 1978; Taylor, 1979). With the advent of the cash economy, a variety of other foods have been incorporated into the diet (Cutter, 1978; Peterson, 1978) but traditional dietary preferences for meat, fat and sugar (section 1.2.3.1) have continued to shape contemporary dietary intake (Sinclair, 1968). Flour does not provide a substitute for a traditional preferred food, but represented a convenient replacement of a reliable staple with several economic consequences (Hamilton, 1975:178; Taylor, 1979; Meehan, 1982:149; Bell, 1983:46; Brady, 1986; Altman, 1987:42; Sladden, 1987:137).

It has also been suggested that contemporary food preferences may originate from the early contact period. For example, wholemeal bread may be viewed with suspicion as 'dirty' in areas where arsenic was added to flour to 'control' the Aboriginal population during the years of early contact with Europeans (Young, 1984:105). Unlike some traditional food which may be taboo at certain times, European food is always considered acceptable and people "*uncritically accepted European foods in all their forms as good for eating*" (Taylor, 1979). To Aboriginal people, the social function of bush food is seen to be more important than the fact that it tastes good and stops hunger; European food is now seen to fulfil the latter role (Stacy, 1975). It has also been postulated (Sansom, 1980:58-60) that some packaged and sealed cooked foods (tinned 'bully' beef, sliced and wrapped bread, soft drinks) may have been preferred as they were "*safe*" from tampering by other people or mystical beings. Similarly 'raw', uncooked 'rations' (flour, sugar) were considered "*safe*" to exchange, but cooked foods should be accepted "*only when one has utter confidence in both the moral integrity of the supplier and in the everyday competence of the supplier as a guardian of the hearth*" (Sansom, 1980:60). Such beliefs highlight that food must be considered from the Aboriginal perspective in order to fully comprehend contemporary food preference.

1.5.3.2. Contemporary role of traditional foods

It is relevant to address contemporary foraging patterns against a background of widespread availability of market foods. Persistence of foraging indicates that Aboriginal people feel a bond with traditional foods and practices, and that knowledge of traditional resources remains within some communities (Latz and Griffin, 1978). However the types of foods utilised may differ greatly from traditional times with items selected on the basis of taste preference, abundance, size, ease of preparation and availability of appropriate market substitutes (Meehan, 1982:148-150; Devitt, 1988:122-124,157,275-276).

In the short term, after establishment of new settlements and communities, there may be an increase in yields of traditional foods. Motor vehicles, boats, hunting and fishing equipment may enable increased yields of some preferred bushfoods (Sinclair, 1977; Cutter, 1978; Altman, 1987:42). The efficiency of hunting may also have increased with the introduction of suitable feral animals (Calaby, 1971; Altman, 1987:44).

However, long term evidence supports the belief that the range of bush foods currently utilised is much *reduced* from the past due to several factors including the effect of environmental degradation caused by stock and feral animals (Chippendale, 1963; Ealey, 1967; Newsome, 1971; Frith, 1978), the increasing incidence of hot, destructive bush fires due to poor land management practices; the restriction of access to some areas of land, introduction of new technology and tools which may eventually deplete resources, and population pressure affecting remaining resources around permanent settlements (Silberbauer, 1971; Peterson, 1973, 1977b, 1978; Frith, 1978; Latz and Griffin, 1978; Taylor, 1979; Cane and Stanley, 1985). Cultural loss may also mean that the knowledge to identify, obtain and prepare bush foods has diminished to such an extent that the full potential of available resources is not realised (Hiddens, 1985; Gibson, 1987). People living in areas other than their own lands may not have rights to forage in the traditional lands of their hosts (Latz and Griffin, 1978). Changing demographic patterns may also have an effect on bush food collection; a relatively greater number of children may inhibit women's opportunity to forage (Cowlshaw, 1980, 1981; Meehan, 1982:149).

Since the early 1970s people have tended to rely on community stores for the major proportion of their diet, with bush food mostly procured recreationally after hours or on the weekend (Wilson, 1953; McArthur, 1960a, 1960b, 1960c; Silberbauer, 1971; Tilley, 1974; Brokensha, 1975:25; Middleton and Francis, 1976:81; Stacy, 1977; Cutter, 1978;

Peterson, 1978; Taylor, 1979; Young, 1984:101). In comparison with the past in which the most readily available foods offered a quality diet, Aboriginal people now "*..still take what food is most readily available, and cannot easily understand that the cheapest foods offered by the white man in his store provide such a poor diet*" (White, 1977:280).

1.5.3.3. Community stores

Depending on perspective, Aboriginal communities stores may be considered to operate either as an essential service with a strong social component or as a primarily commercial enterprise. In addition to the retailing of foods and variety goods, store functions include the provision of banking or financial operations, employment, opportunities for social interaction and the development of a local power base, a potential source of income independent of the government sector, redistribution of resources, development of staff skills, education of the community in the monetary system and a role in ensuring physical well-being of the community (Young, 1984:3-5). However, a negative attitude towards store services has been portrayed frequently (Young, 1984:1). Peterson had described the issue of community stores as a "*scandal*", claiming that "*the majority are poorly and often dishonestly run, even though incorporated as associations for the benefit of Aborigines.*" He suggested that the frequently large financial turnovers could be an important social resource, used to subsidise nutritionally worthwhile foodstuffs, improve facilities and finance educational programs concerned with living on a cash income (Peterson, 1978).

Early stores were established in small, unsuitable premises with inadequate facilities for the storage of bulk food supplies, particularly perishable items. The extremely limited range of foods stocked in the first stores (Rose, 1965:34) and the fact that the established style of counter service limited direct access to products, may have contributed to the conservative choices made by Aboriginal customers and hence affected dietary intakes (Middleton and Francis, 1976:89,92; Young, 1984:8,49). With cash flow proportional to the increasing populations of communities and increasing Aboriginal incomes, store facilities have generally improved. The continued conservative food choice of Aboriginal people has even extended to a preference for particular brands (Sullivan *et al*, 1987). However it is believed that both the layout and staffing of a store can greatly influence diet (Cutter, 1978). Perishable items, such as dairy foods, fruit and vegetables, are frequently found to be in short supply; these items carry the risk of high overheads which some store managers may not be willing to bear.

Prices of foods in Aboriginal community stores have consistently been described at 30-45% above the closest urban centre and up to 70% above southern capital cities (Young, 1984:61-64,139-143; Sullivan *et al*, 1987; NTDHCS Departmental (nutrition) files, 1980-1991). Many factors have been shown to contribute to these high prices (Young, 1984:35,43-48,61). The proportion of individual incomes spent in stores ranged from 50% to 80% (Young, 1981, 1984:91-93) and food accounted for from 50% to 75% of turnover (Young, 1984:53; Sullivan *et al*, 1987). The mean per capita income in Aboriginal communities remains low relative to that of wider Australia (Young, 1984:93) and, combined with the high relative costs of food, clothing and other goods, suggests that financial considerations may be a major aspect affecting nutritional health in remote Aboriginal communities.

Storage of goods in Aboriginal community stores is generally minimal as most have a limited cash flow and may not be able to pay for large orders, particularly in advance, and most have limited appropriate storage space. In one study it was noted that stores in the Pitjantjatjara area were unable to carry more than four weeks stock (Young, 1984:47). Many goods deteriorate if kept for long periods in substandard condition; for example, sugar may become infested with ants. Lack of sufficient space in cool storage may limit both the quantities and varieties of perishable foods, particularly fruit and vegetables which may be restricted to more durable lines such as potatoes and oranges. Stores follow a variety of ordering procedures, but most attempt to follow standardised orders, particularly for dry goods and allow for seasonal fluctuations in demand (Young, 1984:56,59-60).

Ownership, employment and management systems vary markedly for different stores. Although Aboriginal involvement has increased in the first two aspects, management generally still remains under the control of Europeans, and due to common financial problems, control from owners has frequently been relinquished to external supervisory/consultant groups (Young, 1984:64-79).

1.5.3.4. Income and budgeting: the effect of the cash economy on diet

Many studies documented the low levels of per capita income during the sixties and seventies (Rose, 1965:65-74; Silberbauer, 1971; Stevens, 1974:82-92; Anderson, 1976; Middleton and Francis, 1976; Stanley, 1976; Peterson, 1977b; Thomson, 1982). The poor diet during early settlement years, particularly the reliance on flour, sugar and tea has

been related to the low income of these years (Cawte, 1973:225; Sinclair, 1977); in particular an inverse association between income and apparent flour intake was noted (Peterson, 1977b).

However the relationship between diet and income may not be straightforward and there is no evidence that the nutritional value of the diet will necessarily improve with increased income (Anderson, 1976; Taylor, 1979). Regardless of the availability of money, people tended to purchase 'familiar' items rather than cheaper, less familiar alternatives (Tilley, 1974; Peterson, 1977b). There is little evidence that nutritional concerns dictate purchasing patterns. Claims that the high proportion of total money spent on flour (10.9%) and chicken (10.5%) represent 'sensible buying' due to their relatively high value for money on the basis of both protein and energy content (Middleton and Francis, 1976:89), do not necessarily hold for sugar (10.7%), tinned corned beef (12.4%) or soft drink (6.6%). Rather explanations for such purchases may lie in taste preference and convenience as previously discussed (section 1.5.3.1).

With increased income has come increased demand for material goods and services (vehicles, appliances, 'travel', clothing) which are now affordable (Tilley, 1974; Taylor, 1979); available cash may be diverted towards the purchase of deferred consumer demands by various strategies including the pooling of extended family financial resources and gambling (Goodall, 1987).

1.5.3.5. Contemporary methods of storing, preparing, cooking and distributing food

In centralised communities most foods are consumed within 24 hours of purchase (Middleton and Francis, 1976:90-1; Cutter, 1978; Knight and Rowse, 1990). People living a distance from stores, particularly in outstations, tend to purchase easily stored foods, such as flour and sugar. The availability of metal containers enabled the rapid boiling of food which depleted the nutritional value of the diet due to leaching of nutrients into the cooking water (Middleton and Francis, 1976:85). Convenience foods requiring little preparation are also popular (Wise *et al*, 1970). Distribution of European food is rarely bound by Traditional Law (Stacy, 1975).

1.5.3.6. Agriculture

Although most settlements had vegetable gardens in early years of establishment, there are few remaining examples (Miles, 1978). Much discussion has focussed on the possibilities of agriculture as an economic or subsistence basis in Aboriginal communities and outstations (Hetzl and Frith, 1978). However most commentators do not address the many difficulties confronting, and frequent failure of, European agricultural incursions into the tropical northern and central arid regions of Australia (McArthur, 1960b; Bauer, 1964:209-10; MacKenzie, 1980:55-65) or the cross-cultural stress caused by imposing an agricultural pattern on a hunting and gathering society (Kimber, 1976; Cutter, 1978; Latz and Griffin, 1978; Maggs, 1978; Miles, 1978; Till, 1978; Coombs *et al*, 1982; Cane and Stanley, 1985). In some areas horticultural management of traditional plant foods may also be prohibited by tribal Law.

1.5.4. Use of drugs

Alcoholism (Albrecht, 1974; Bain, 1974; Lickiss, 1974), petrol sniffing (Nurcombe, 1974; Brady and Morice, 1982) and the use of kava (Mathews *et al*, 1988) have been reported in many areas.

The House of Representatives Standing Committee on Aboriginal Affairs stated that alcohol was "*the greatest present threat to the Aboriginals of the Northern Territory*" (Australia, 1979:24). Many articles have commented on the direct and indirect implications of alcohol abuse, for the health and nutrition of Aboriginal individuals and communities (Kamien, 1975, 1978:145-160; Cawte and Mununggurr, 1977; Reid and Mununggurr, 1977; Collmann, 1979; Hunt, 1981; Brady and Palmer, 1984; Thomson, 1984b; Valliappan, 1986), including the diversion of substantial amounts of money to the purchase of alcohol rather than food (Gracey, 1986a). However there is little factual information about the extent and severity of the problem. It has been estimated that in major communities in the 'Top End' 10.7% of women and 63.4% of men drank alcohol, but that this was reduced to a total of 28.2% of all people in communities where alcohol use is restricted and in these communities only 12.1% of all drinkers consumed alcohol sometime each week (Watson *et al*, 1988). Figures were relatively higher in Central Australia and Katherine regions. Both rates and frequency of consumption were higher in communities where alcohol was freely available. Overall, fewer Aboriginals than

Caucasians were found to drink alcohol in the Northern Territory (Watson *et al*, 1988).

Smoking can contribute to nutritional problems and is one of the major contributing factors to cardiovascular disease (MRFIT, 1986). A recent survey of smoking habits (Watson *et al*, 1988), found that 56% of all Aboriginal people in the Northern Territory smoke cigarettes, including 43% of the women and 71% of the men. However relatively few women in Central Australia smoked cigarettes. The purchase of cigarettes may also potentially divert finances from food expenditure.

Kava is an psychoactive drink, prepared from the root of *Piper methysticum* widely used throughout the South Pacific, but introduced into some Northern Australian Aboriginal communities during the late 1970s, where it became a popular alternative to alcohol (Alexander, 1985; Watson *et al*, 1988). Knowledge of the physiological effects of the various chemical constituents is incomplete. A pilot survey in one eastern Arnhem community investigated the health effects of kava usage (Mathews *et al*, 1988). Amongst measured parameters, weight loss, scaly skin rash, grossly elevated levels of gamma-glutamyl transferase, low albumin, low plasma protein, low urea and low bilirubin levels, haematuria, increased red cell volume and possible evidence of pulmonary hypertension, were associated with heavy kava usage as determined by consensus ranking. The extent to which kava consumption affects dietary intake and hence nutritional status, and the extent to which the negative health effects associated with kava consumption are mediated by nutritional status, are unknown.

1.5.5. Reduced energy expenditure

"The apathy and lack of desire for activity were noticeable features of the majority of adults at all settlements.. The lack of interest in gathering traditional foods may seem to indicate no great desire for food, but it must be remembered that in general, considerable distances had to be walked, under conditions of heat which were not conducive to great physical effort, and for a prize of uncertain magnitude" (Billington, 1960:36).

Early studies make subjective, apparently racist comments about genetic differences in energy expenditure *"..under suitable conditions of climate, nutrition and avoidance of physical exertion (Aborigines) are capable of leading a more lethargic existence than seems possible to the average white man"* (Wardlow and Lawrence, 1932). This

observation has been challenged by the argument of selected, physiological adaptation to the hunter-gatherer lifestyle (Campbell, 1939c:76). Compared with the effort required to procure traditional foods, energy expenditure for the production of contemporary 'store foods' is much reduced (Moodie, 1981).

1.6. Contemporary Aboriginal health and nutritional status, with particular emphasis on the Northern Territory

1.6.1. Demography

There are many methodological problems with determination of base-line population for community-based studies. Census figures from the Australian Bureau of Statistics do not compare well with known population figures and tend to underestimate the number of individuals residing in Aboriginal communities (Gray and Smith, 1983). For logistic reasons electoral rolls for remote communities are also considered incomplete (Kamien, 1978). Early studies (Abbie and Schroder, 1960; van Dongen *et al*, 1962), used population records compiled in recently-established missions and settlements as demographic bases. The accuracy of such records is unknown and early settlements may not have been representative of the whole tribal population. Defining the age of subjects was a particular problem in early studies and age stratification and standardisation were rarely performed. Mobility of Aboriginal people may present particular problems in remote communities, particularly during longitudinal studies.

1.6.2. Mortality data

The lack of comprehensive data describing Aboriginal health on a national basis severely limits meaningful analysis (Thomson, 1984a, 1985; Gray, 1990). However, an analysis of available data confirmed that the general level of Aboriginal mortality is significantly higher than that of the Australian population as a whole (Gray, 1990). A markedly reduced life expectancy at birth for both men and women, Aboriginal (53 and 63 years respectively) and non-Aboriginal Australia (71.4 and 78.4 years respectively) has been noted (Thomson, 1991). For the Northern Territory in 1980-1982, the standardised mortality rate of Aborigines was 3.4 times that of the non-Aboriginal population (Thomson, 1985).

Most public attention has focussed on the high infant mortality rate, which had been

approximately 100 per thousand in the early 1970s. Despite a marked decline from this level, in 1981 the national Aboriginal rate of 26.4 deaths per 1000 live births was still 2.8 times the non-Aboriginal rate (Dugdale, 1980; McNeilly *et al*, 1983; Thomson, 1983, 1985). In the Northern Territory the infant mortality rate had decreased from 143 to 50 deaths per 1000 live births between 1965 and 1975, and was 23 deaths per 1000 live births in 1984 (Walker *et al*, 1982; Devanesen *et al*, 1986). All other indicators suggest that Aboriginal foetal and infant mortality is still unacceptably high when compared to the data for non-Aboriginal Australians (Thomson, 1983, 1985).

Diseases of the circulatory system (ICD 390-459), are now the leading cause of death among Aborigines (Thomson, 1984a; Devanesen *et al*, 1986:24-27; 36-37; Plant, 1990). However, the limitation of such data obtained from death certificates has been used to argue that community based studies may better identify the prevalence of different types of heart disease and better identify related causal and associated risk factors (Sladden, 1987:9,24).

1.6.3. Infant malnutrition

High infant mortality and morbidity rates have been reported for communities in many parts of Australia, with some of the earliest reports coming from the Northern Territory (Crotty, 1958; Kettle, 1966; Kirke, 1969). Numerous studies have indicated that infection, particularly of the gastrointestinal and respiratory tract, associated with malnutrition and growth retardation was, and continues to be, the most common cause of death in young children, particularly before the age of two (Gandevia, 1967; Jose *et al*, 1968; Rountree, 1969; Moodie, 1969, 1973:51-54; Lickiss, 1970; Jose and Welch, 1970; Edwards, 1970; Walker and Harry, 1972; Doherty, 1973, 1974; Ford *et al*, 1976; Middleton and Francis, 1976; Gracey, 1977; Gracey *et al*, 1980; Harris *et al*, 1984). Growth retardation amongst Aboriginal infants after the age of four to six months has consistently been noted (Jacobs and Clements, 1968; Propert *et al*, 1968; Maxwell and Elliot, 1969; Jose and Welch, 1970; Cox, 1979; Gracey *et al*, 1983; Rae, 1989). Relatively poor growth of young children has also been shown to persist in older children (Hitchcock *et al*, 1987). Low Aboriginal birth weights have been frequently documented and are considered to be the result of a number of factors, of which poor maternal nutritional status is thought to be dominant (Seward and Stanley, 1981; Gracey *et al*, 1984; Thomson, 1984a; Rae, 1989). Some specific nutrient deficiencies, such as low folate status (section 1.6.10) have been implicated in the aetiology of low birth weight in disadvantaged groups (Inyegar and

Rajalakshmi, 1975; Gandy and Jacobson, 1977). In addition to intestinal and respiratory tract infections, particularly high incidences of contagious skin disorders, anaemia, eye and ear infections have been described in growth retarded children (Jose *et al*, 1968; Kirke, 1971; Moran *et al*, 1979; RACO, 1980; McNeilly *et al*, 1983; Sunderman and Dyer, 1984).

Most studies suggested that a combination of poor diet, the occurrence of infection, secondary lactose intolerance, poor maternal nutritional status, 'lack of parental nutrition knowledge' and poor environmental conditions contributed to infant's 'failure to thrive'. Theories that genetic differences account for the differing growth pattern between Aboriginal and European children are not supported by studies which indicate similar growth in similar environments (Maxwell and Elliot, 1969; Cockington, 1980) or the greater stature reported for nomadic children compared to those Aboriginal children living on reserves and settlements (Abbie, 1975). The synergism of malnutrition and infection is well recognised (Scrimshaw *et al*, 1968; Jose *et al*, 1975; Mata *et al*, 1977; Chandra, 1980, 1983; Neumann, 1980; Suskind, 1980; Chandra, 1983; Dinarello, 1985; Gershwin *et al*, 1985:4). In particular, folic acid, pyridoxine, vitamin C, vitamin A, zinc, copper, selenium and vitamin E have been implicated in impaired cell mediated immunity and T-cell dependent antibody responses.

Clinical evidence of multiple vitamin deficiencies in 600 growth-retarded children were identified in one Queensland community in the late 1960s (Jose and Welch, 1970). Various studies have suggested specific nutrients such as zinc, vitamin C, vitamin D, iron and vitamin A, may be major contributing factors to poor health in Aboriginal children (Kalokerinos, 1969, 1971, 1974; Jose and Welch, 1970; Cheek *et al*, 1981). However it seems most unlikely, in the face of the complex factors contributing to poor dietary intake, infection and malnutrition, that administration of any single nutritional element (or 'magic bullet') will have a positive and lasting effect on Aboriginal child health (Nobile, 1974; Gracey, 1986b; Gracey and Spargo, 1987). Similarly the call for increased energy density of the diet by incorporation of fats and oils to infants' food (Dearden *et al*, 1980), may have negative consequences in later life, or for adults if incorporated into the family diet (Dugdale and Lovell, 1981; Dugdale, 1986).

1.6.4. The study of non-communicable disease in Aboriginal communities

1.6.4.1. Introduction

The recent reduction in morbidity from communicable disease, particularly in children, has been countered by an increase in non-communicable (or 'lifestyle') disease such as obesity, NIDDM, hypertension and cardiovascular disease in adults. The emergence of non-communicable disease in transition from traditional to western lifestyle is believed to be associated with reduced physical activity and the concomitant high energy, high fat, high refined carbohydrate, low dietary fibre and nutrient depleted diet (Trowell and Burkitt, 1981). Despite inconsistencies in methodologies, definitions and parameters studied, a clear pattern of the increasing prevalence of these conditions is evident in Aboriginal communities.

1.6.4.2. Methodology

In addition to the problems associated with accurate determination of the population basis for measurement of community morbidity (section 1.6.1), various difficulties make comparison of past community-based studies of non-communicable disease difficult. Problems exist with the application of dissimilar sampling frames; inter-observer variation; differing and frequently poor response rates; confirmation of fasting status; standardisation of measurements; diagnostic criteria; the application of different protocols; varied specimen handling, storage and transport, and differing stratification of results. The difficulties with comparison of recent survey data have been practically illustrated (Gault, 1990:21,23,27,29).

1.6.5. Cardiovascular disease

1.6.5.1. Diet and cardiovascular disease

Although coronary heart disease remains the most frequent cause of death in western nations (Castelli, 1984), mortality rates from the disease have significantly decreased over the last 25 years in several countries (Rose, 1984; Thom *et al*, 1985) including Australia (Heller, 1986). The extent of the reduction in incidence of ischaemic heart disease is related to the extent of the change in risk factors which include hypercholesterolaemia, hypertension and cigarette smoking (Butler *et al*, 1985; Castelli *et al*, 1986; Martin *et al*,

1986; MRFIT, 1986). In developing countries, however, there is evidence that more affluent urban groups comparatively recently exposed to urbanisation, are increasingly affected by coronary heart disease (Marmot, 1980).

Dietary modification has been advocated as the preferred therapy to reduce unacceptably high serum cholesterol levels (WHO, 1986a; USDHHS, 1988:94). Dietary lipids have been shown to affect plasma lipoprotein levels and quantitative relationships between dietary lipids, dietary cholesterol and plasma serum cholesterol levels have been demonstrated (Keys *et al*, 1957, 1965; Hegsted *et al*, 1965). It is only recently that the link between different specific dietary lipids and serum lipid fractions has been investigated; both polyunsaturated and monounsaturated fatty acids tend to reduce low density lipoprotein (LDL) cholesterol (Mensink and Katan, 1989). Dietary fibre, particularly soluble fibre, is also thought to be protective against ischaemic heart disease secondary to its effect on plasma lipids (Jenkins *et al*, 1983).

Other conditions associated with dietary factors, including hypertension, NIDDM and impaired glucose tolerance (IGT), obesity (specifically abdominal obesity) and hyperlipidaemia have been shown to be major predictors for ischaemic heart disease (Fuller *et al*, 1980; Larsson *et al*, 1984; Zimmet *et al*, 1986; Fontbonne *et al*, 1989).

1.6.5.2. Aboriginal diet and risk of cardiovascular disease

Despite containing a high proportion of animal foods, the traditional Aboriginal diet would have protected against coronary heart disease in several ways (section 1.2.2.3). Twenty years ago Moodie wrote briefly of degenerative heart and vascular disease, predicting that ideal conditions for its treatment were unlikely to exist for a large section of the Aboriginal population, "*so that severe disability and death for those affected is likely to occur at younger ages than in the white population*" (Moodie, 1973:212).

Research into Aboriginal cardiovascular disease has concentrated on ischaemic heart disease, in particular describing prevalence of accepted risk factors and associated conditions (Schwartz *et al*, 1957; Schwartz and Casley-Smith, 1958a, 1958b, 1959; Wise *et al*, 1970, 1976; Edwards *et al*, 1976; Bastian, 1979; Simons *et al*, 1981; Stanton *et al*, 1985; O'Dea *et al*, 1982, 1988a, 1988b, 1990; Sladden, 1987; Gault, 1990). Methodological approaches to enable reliable comparison of results have not generally been considered (section 1.6.4.2).

Compared with non-Aboriginal groups, these studies have most commonly shown less hypercholesterolaemia but more hypertriglyceridaemia in all age groups in both sexes, more obesity and hypertension in younger men and women, and more cigarette smoking in all age groups. Fasting serum triglyceride levels have been shown to be strongly associated with impaired glucose tolerance (IGT) and hyperinsulinaemia. Aboriginal groups have much higher prevalence rates of diabetes and impaired glucose tolerance than non-Aboriginal groups (section 1.6.6). Studies in Aboriginal groups alone have most commonly shown lower serum cholesterol and triglyceride levels in women than men, lower serum triglycerides in younger men and women, lower blood pressure in young women than young men, more smoking among younger people, and more obesity in older women than men (Schwartz *et al*, 1957; Schwartz and Casley-Smith, 1958; Charnock *et al*, 1959; Wise *et al*, 1976; Bastian, 1979; Boyden and Agar, 1981; O'Dea *et al*, 1982, 1988a, 1988b, 1990; Sladden, 1987; Gault, 1990).

1.6.6. Diabetes, hyperinsulinaemia and insulin resistance

1.6.6.1. Diagnosis of non-insulin dependent diabetes

Various definitions of diabetes have been applied in the past; however the current definition of the World Health Organisation (WHO, 1985:10-12) has been generally accepted in recent years. This definition is based on a standard 'glucose tolerance test': the administration of 75 g of glucose following an overnight fast and an intake of at least 200 g of carbohydrate during the previous three days. Diabetes is diagnosed on the following criteria: fasting venous plasma glucose ≥ 7.8 mmol/l and venous plasma glucose ≥ 11.1 mmol/l two hours following administration of the 75 g glucose load. Impaired glucose tolerance (IGT) is defined as venous plasma glucose 7.8 mmol/l to 11.0 mmol/l inclusive, two hours following administration of the 75 g glucose load. Both diabetic and impaired glucose tolerant states are associated with the development of complications of diabetes, and have been considered together as 'abnormal glucose tolerance' to indicate the risk of such conditions in communities.

1.6.6.2. Abnormal glucose tolerance in Aboriginal groups

Since the early 1960s, higher prevalence rates of diabetes (8-24%) (Winterbottom, 1961; Wise *et al*, 1970; Finlay-Jones and McCormish, 1972; Kamien, 1976; Wise *et al*, 1976; Bastian, 1979; Duffy *et al*, 1981; Cameron *et al*, 1986; Williams *et al*, 1987) have been

consistently described in Aboriginal groups relative to wider Australia (3.4%) (Glattaar *et al*, 1985). Rates have been shown to rise with increasing 'westernisation' (Wise *et al*, 1976).

Diabetic complications (retinopathy and blindness, renal disease, poor wound healing and gangrene, peripheral neuropathy and increased risk of occlusive vascular disease such as coronary heart disease, stroke and thrombosis) have been described in Aboriginal diabetics (Patel *et al*, 1991), but little data is available on the pattern of diabetic vascular complications in Aborigines. Diabetic retinopathy and proteinuria have both been found to be more common in Aborigines than Caucasians (Stanton *et al*, 1985); proteinuria has been reported in up to 80% of diabetics in one Aboriginal community (van Buynder, 1991). However it is difficult to control for duration of the disease and compliance with treatment in such studies.

A group of metabolic characteristics consistent with underlying resistance to the glucose lowering effects of insulin (mild impairment of glucose tolerance, hyperinsulinaemia and elevated total and very-low-density lipoprotein concentrations) have been observed in several Aboriginal communities (Bastian, 1979; O'Dea *et al*, 1982, 1988a, 1988b, 1990). The contemporary lifestyle experienced by many 'westernised' Aboriginal people, which incorporates low physical activity and an energy-dense diet rich in rapidly absorbed carbohydrate and fat, is thought to produce hyperglycaemia and stimulate a high post-prandial insulin response (Collier *et al*, 1984). This may produce increased glucose turnover in insulin-sensitive tissues and exacerbate selective insulin resistance, favouring those non-gluco-regulatory metabolic pathways where insulin is functioning normally or near normally; that is, hepatic triglyceride and VLDL synthesis and accumulation of fat in adipose tissue (O'Dea *et al*, 1988a). While physical inactivity and high intakes of refined carbohydrate and saturated fat are maintained, insulin resistance, hyperinsulinaemia and glucose intolerance become more pronounced leading to obesity, type 2 diabetes and hypertriglyceridaemia (Neel, 1962; O'Dea *et al*, 1982, 1988a). However this sequence of events has been shown to be potentially reversible (Stanik and Marcus, 1980; Hughes *et al*, 1984; O'Dea, 1984). A recent study identifying hyperinsulinaemia and impaired glucose tolerance in Aboriginal children between the age of 7 and 18 years (White *et al*, 1990), indicated that the precursors of the disease may be evident at an earlier age than previously recognised. This emphasises the need for early intervention in order to prevent diabetes in Aboriginal communities.

1.6.7. Obesity and body fat distribution

Most Aboriginal groups tend to experience an increase in weight with increasing acculturation (Barrett and Brown, 1971; Brown and Barrett, 1973; Wise *et al*, 1970; Bastian, 1979; O'Dea, 1987; O'Dea *et al*, 1990). However, more recent studies of maternal nutritional status (Rae, 1989) and other women of child-bearing age (Gracey *et al*, 1984) have described low BMI in young women. In older women a rapid onset of obesity, with a central distribution of subcutaneous fat, has been described (Rutishauser and McKay, 1986). It has been postulated that individuals who experience under-nutrition as infants tend to suffer from obesity in adult life (Dugdale and Lovell, 1981; Dugdale, 1986).

With increasing BMI, both male and female Aborigines tend to exhibit an android body profile (central distribution of fat) (Rutishauser and McKay, 1986; O'Dea, 1987; Sladden, 1987:116), compared with the gynoid (peripheral) distribution of body fat most commonly seen in overweight and obese Caucasian women. Android distribution of body fat (indicated by a waist circumference:hip circumference ratio of ≥ 1.0 for men, or ≥ 0.8 for women) is associated with hypertriglyceridaemia, higher fasting glucose levels, hyperinsulinaemia, impaired glucose tolerance and hypertension in Caucasians (Kissebah *et al*, 1982; Krotkiewski *et al*, 1983; Larsson *et al*, 1984; Ohlson *et al*, 1985; per Bjorntorp, 1985; Haffner *et al*, 1986) and increased susceptibility to the metabolic complications of obesity (O'Dea, 1987; Zimmet *et al*, 1986).

1.6.8. Blood pressure

Blood pressure is known to be affected by many variables, including age, obesity, level of physical activity, alcohol, psychological stress and diet. Of dietary factors, high sodium and saturated fat intakes have both been implicated in the aetiology of hypertension (McGregor, 1983; Rowse and Beilin, 1983; Pietenen and Huttunen, 1987). Insulin resistance has been shown to be associated with impaired renal sodium excretion (DeFronzo, 1981) and has been postulated as an independent risk factor for hypertension (Modan *et al*, 1985), supporting the association of hypertension with obesity, glucose intolerance and hypertriglyceridemia described in many studies. Most Aboriginal studies have not controlled for these factors (Kirk, 1983). Problems with comparability of data also arise due to the different methods of measurement applied in different studies.

Although few measures of blood pressure for Aboriginal groups who had experienced little or no contact with western lifestyle are available (Moodie, 1981), those measured in settlements during the 1960s were generally low compared with European levels and blood pressures are presumed to have been lower in traditional Aboriginal people (Nye, 1937; Casley-Smith, 1959b; Abbie, 1960; Abbie and Schroder, 1960; Van Dongen *et al*, 1962; Woods, 1966; Macfarlane, 1978). More recent studies have indicated a higher prevalence of hypertension in Aboriginal communities (Wise *et al*, 1970; Edwards *et al*, 1976; ; Neilson and Williams, 1978; Bastian, 1979; Duffy *et al*, 1981; Simons *et al*, 1981; Phillips and Kubisch, 1985; Gault, 1990:31). Results have been summarised (Thomson, 1984a; McGrath *et al*, 1991:43-45).

1.6.9. Anaemia and haemoglobin status

Although anaemia may be regarded as a nutritional disorder, it is not necessarily due to dietary deficiency. Malabsorption of iron, protein and/or folic acid, or abnormal iron and protein losses such as may occur in heavy hookworm infestations in tropical areas may also contribute to low levels of haemoglobin (Crotty, 1958; Billington, 1960:46; Hodges, 1960; Moodie, 1973:191). Early data from recently nomadic Aboriginals generally indicated high haemoglobin concentrations for both men and women (Davis and Pitney, 1957; Davidson, 1957; Casley-Smith, 1958, 1959a, 1960; Abbie, 1960; Elphinstone, 1971). There has been an increase in both incidence and prevalence of anaemia in more recent studies (Crotty, 1958; Pitney, 1962; Kamien *et al*, 1974; Davis *et al*, 1975; Holt *et al*, 1980). Microscopic examination of blood cells suggested iron deficiency was implicated in most cases of anaemia (Pitney, 1962; Kamien *et al*, 1974). However in the Northern Territory severe macrocytic anaemia with associated megaloblastic bone marrow was also described; it was claimed that some cases responded only to folate supplementation (Watsford, 1955).

1.6.10. Vitamin and mineral status

Vitamin status has been measured infrequently in Aboriginal subjects and there is a general paucity of longitudinal data available.¹ Studies have been conducted in a variety of Aboriginal groups and environments; from people of Aboriginal descent in western

¹ Due to the limited data, and in order to avoid repetition, details of biochemical investigations of vitamin status of Aboriginal groups are discussed with relevant findings of the present study (section 8.2).

New South Wales townships (Kamien *et al*, 1974; Nobile, 1974), sub-groups of full-descent populations, particularly children (Jose and Welch, 1970; Cheek *et al*, 1989) and groups living in remote areas (Davis *et al*, 1965, 1975; O'Dea, 1987; Mathews *et al*, 1988; Gault *et al*, 1990). Samples have generally been small and non-randomly selected; subjects have often been selected from 'stress' groups of the community (that is, infants and pregnant and lactating mothers), perhaps in an attempt to describe the worst possible scenario (Jose and Welch, 1970; Nobile, 1974; Cheek *et al*, 1989). Various methods of assays and functional tests have been applied in different studies, particularly as 'the state of the art' of analysis has changed over time. Different 'normal' ranges have also been applied. Therefore quantitative comparison of published results and prevalence rates of deficiency states may be misleading.

Multiple biochemical vitamin deficiencies have frequently been described in the same subject and suggest the generally poor nutritional status of such individuals, rather than a specific dietary problem.

Relative to 'normal' European levels, very low serum folate levels have generally been reported in acculturated groups of Aborigines and have been described in the majority of subjects (Davis *et al*, 1965, 1975; Kamien *et al*, 1974; Nobile, 1974; Watson and Tozer, 1986; Cheek *et al*, 1989; Gault, 1990:49; Patel *et al*, unpublished; Lion *et al*, unpublished). In contrast, 'normal' red blood cell folate levels have been recently described for a small traditionally-orientated group of Aboriginal people living at a remote outstation in North East Arnhem land (O'Dea *et al*, 1988b).

Very high levels of serum cobalamin relative to the European reference range have been described for both traditional and acculturated Aboriginal groups (Davis and Pitney, 1957; Pitney, 1962; Elphinstone, 1971; Davis *et al*, 1975; Holt *et al*, 1980; Gault, 1990:49).

Poor thiamine status was described for most age and sex groups in a '*part Aboriginal*' community (Kamien *et al*, 1974), but was particularly marked for women of child bearing age (Kamien *et al*, 1974; Nobile, 1974). Fortification of flour resulted in improvements in the thiamine status of this community (Kamien *et al*, 1975b). Surprisingly large ranges of both red blood cell and serum thiamine concentrations, including some high values, have been described in remote Aboriginal communities (Gault, 1990:49; Riley *et al*, 1990). Relative to the 'normal' European reference range, low levels of vitamin B₆ have been

described in high-risk groups at four Aboriginal communities (Kamien *et al*, 1974; Nobile, 1974; Davis *et al*, 1975). Relatively low riboflavin status has been described in two Aboriginal studies (Kamien *et al*, 1974; Nobile, 1974).

Despite dramatic fluctuations in the availability of the vegetable component of the traditional diet, little evidence of scurvy has been recorded for traditional Aboriginal groups (Billington, 1960; Hodges, 1960; McArthur, 1960b; O'Dea *et al*, 1987). Low plasma vitamin C levels have been reported in high-risk Aboriginal groups (Hodges, 1960; Jose and Welch, 1970; Kamien *et al*, 1974; Nobile, 1974). However claims that vitamin C deficiency was a major factor in the aetiology of morbidity in Aboriginal children (Kalokerinos, 1974) have not been substantiated.

The suggestion that Aborigines are the only Australian group who appear to have a consistently low intake of vitamin A (Rutishauser, 1990a) has not been supported by biochemical data. Vitamin A plasma levels were generally found to be acceptable in Aboriginal communities (Kamien *et al*, 1974; Nobile, 1974; O'Dea *et al*, 1987; Cheek *et al*, 1989). However, low plasma β -carotene levels have frequently been described (Kamien *et al*, 1974; Nobile, 1974; Cheek *et al*, 1989) and are presumed to reflect the low apparent intake of fruit and vegetables in Aboriginal communities.

Relative to 'normal' Caucasian concentrations, low levels of plasma vitamin E have been described in some Aboriginal groups (Kamien *et al*, 1974; Nobile, 1974; O'Dea *et al*, 1987). However these results may not have been important as the Aboriginal diets tended to be low in polyunsaturated fatty acids; a ratio of serum α -tocopherol to total lipid ratio less than 0.8 mg/g is considered acceptable for adults (Roberts, 1990).

Several studies in Aboriginal communities have measured low zinc levels in hair (Eastwell, 1981; Smith *et al*, 1982; Reilly and Harrison, 1983) and studies focussing on the Kimberley area have suggested that zinc deficiency may contribute to growth retardation and under-nutrition in Aboriginal children (Holt *et al*, 1980; Cheek *et al*, 1981, 1982; Smith *et al*, 1982). However, a placebo-controlled double-blind trial of zinc supplements (Solomons, 1980; BMJ, 1981) in the same region showed an increase in the concentration of plasma zinc in 200 children over 10 months, but no response in growth (Smith *et al*, 1985). The relationship between the concentration of zinc in hair and zinc status remains controversial (Solomons, 1980; Hambidge, 1982; Klevey *et al*, 1987).

Similarly plasma zinc concentration, which has also been found to be low in Aboriginal groups (Cheek *et al*, 1989) may not accurately reflect zinc status (Solomons, 1980; BMJ, 1981).

1.7. Dietary survey methodology

"The measurement of the habitual food intake of an individual must be among the most difficult task a physiologist can undertake" (Garrow, 1974).

Dietary survey methodologies were reviewed in order to identify possible methods to quantitatively measure food and nutrient intake in Aboriginal communities. Most dietary methods were first described during the 1940s and 1950s, however the literature on dietary survey methodology remains scattered and contradictory. Choice of a particular dietary method will depend on several factors including the type of study under consideration, the objectives of the study, the target population and sample size. The validity and precision of dietary methods must always be assessed in terms of the study and the group being surveyed. However, the methodological difficulties in measurement of dietary intake have been considered to be so marked that accurate quantitative assessment of dietary intake may not possible (Wynder, 1976).

1.7.1. Dietary survey methods

1.7.1.1. Accounting techniques

At the national level, food balance sheets are accounts of the annual production and utilisation of food monitored as a measure of apparent dietary intake of the population on a per capita basis. In Australia apparent consumption data are collected annually, and defined as the sum of commercial production, estimated home production, imports and opening stocks, less the sum of exports, stores of ships and aircraft, usage for processed foods, wastage, non-food usage and closing stocks (Cashell, 1981). Apparent consumption data do not address individual variation in dietary intake, but with consistent, standardised methods of collecting, collating and processing data, may indicate large dietary differences between countries and within countries over time (Baghurst and Baghurst, 1981; Cashell, 1981). Although data collected at the national level are not able to be directly validated (Margetts, 1982) it has been reckoned that apparent consumption figures overestimate

actual energy intake by up to 30% in developed countries (Baghurst and Baghurst, 1981).

At the level of households, the food account method is a record of all foods purchased and brought into the household for a period of at least one week, assuming there is no significant change in household food inventories (Pekkarinen, 1970). The inventory method is the food account method conducted with an inventory of food in the house at the beginning and end of the study period (Klaver *et al*, 1982).

Food sales data provided by retailers have more recently been investigated in an attempt to measure apparent dietary intake on a regional basis (Nelson, 1983). However low co-operation rates and incomplete data collection have limited comparisons of results to proportional dietary consumption (Nelson, 1983).

1.7.1.2. Recorded intake methods

Several approaches to dietary records have been described, involving variations in the person recording (subjects or trained observer), the method of recording, the time of study and methods of nutritional analysis of data (Marr, 1971), including the weighed-record method (Klaver *et al*, 1982), the 'precise weighing' method (Marr, 1971), the 'duplicate portion' method, the 'weighed inventory' method (Widdowson, 1936, 1947; Widdowson and McCance, 1936), the 'cumulatively weighed' method (Marr, 1965; Bingham *et al*, 1982) and the 'estimated method' (Youmans *et al*, 1942; Marr, 1971).

1.7.1.3. Interview methods

The 24-hour recall method has been the most widely used method of dietary assessment, being relatively non-invasive, rapid and inexpensive (Willett, 1990:52). The amount, in household measures, of all food consumed over the previous 24 hours is recalled, usually in response to prompts from an interviewer. Food models and varying sizes of utensils and serving bowls may be used to aid estimation of serving sizes.

The original diet-history method incorporated three phases: the establishment of the 'usual' daily menu, a 'cross-check' of frequency of consumption and preference for particular foods and a 3-day food record (Burke, 1947). Several modifications of this method have been used (Pekkarinen, 1970; Marr, 1971; Bazzare and Myers, 1980; Baghurst and Baghurst, 1981).

Food frequency methods were initially developed in an attempt to devise a 'short' method of determining relative qualitative dietary intake of individuals in epidemiological studies and usually have been used to assess the intake of specific foods or nutrients rather than the total diet (Wiehl and Reed, 1960; Stefanic and Trulson, 1962; Acheson and Doll, 1964). The rationale for food frequency methods is based on the observation that frequencies of intake of foods are highly correlated with total weights of the same foods consumed over an extended period (Heady, 1961). Food frequency questionnaires (FFQ) may be self-administered by co-operative, literate and numerate subjects or administered by an untrained observer. FFQ can be faster, less invasive and less expensive than the 24-hour recall method. Portion sizes may be included to produce semi-quantitative data (Abramson *et al*, 1963; Epstein *et al*, 1970; Hankin *et al*, 1970, 1975; Marr, 1971; Baghurst and Baghurst, 1981; Jain *et al*, 1982; Willett, 1990:9-92).

1.7.2. Measurement of dietary intake of populations

In assessment of Aboriginal diet, measure of the long term 'usual' intake of groups would be of more use/interest than intake of individuals over a short time span. In general large numbers of observations (that is, observations on a large number of individuals or a large number of observations on a small group of individuals) have been shown to be required to detect meaningful differences in the mean intakes of groups; for example, up to 106 days have been shown to be required for vitamin A to lie within 20% of the true value (Liu *et al*, 1978; Baghurst and Baghurst, 1981; Bingham, 1982; El Lozy, 1983; Willett, 1990:45). The dietary intake of groups of individuals varies from day to day and may be systematically influenced by factors which are subject to cultural and ecological factors, such as day of the week and/or seasonality (Willett, 1990:34). The extent of inter-individual variation will affect the power to determine dietary differences between groups for a given number of observations (Bingham, 1987). In studies within industrialised countries daily intra-individual variation in nutrient intake has consistently been shown to be large; as high as 20%-30% for energy and 44%, 60% and 105% respectively for the more variable nutrients riboflavin, vitamin C (Liu *et al*, 1978; Beaton *et al*, 1979, 1983; Sempos *et al*, 1985; Bingham, 1987) and vitamin A (Willett, 1990:44). The effects of seasonality are believed to substantially increase intra-individual dietary variation in the non-industrialised setting (Brown *et al*, 1982).

1.7.3. Validation of dietary survey methods

"From the limited information available it has to be concluded that the errors involved in using some methods of dietary assessment are far greater than generally acknowledged" (Bingham, 1987).

In every study attempting to measure dietary intake the validity of the dietary survey method applied must be assured. Many different terms dealing with the validity and reproducibility of dietary methods have been used in the literature, resulting in some confusion (van Staveren and Burema, 1982). Errors incurred in the measurement of diet may be either random or systematic. The effect of random errors on the precision of a method may be minimised by increasing the numbers of observations, whereas bias is independent of the number of observations. An accurate method should be both free of bias (valid) and precise (reproducible). In validation of methods used in determination of the dietary intake of groups, determination of reproducibility is less important than of bias (Hautvast and Klaver, 1982:60). Reproducibility reflects precision and is a measure of variability affected by random error, arising from both random response or technical errors, and biological variability. In dietary surveys biological variability consists of inter-individual variation (variation in 'usual' dietary intake between individuals), intra-individual variation (day-to-day variation within the same individual), day-of-the-week effect and methodological random error (van Staveren and Burema, 1982).

Validity specifically refers to lack of systematic error (bias). Various aspects of validity may be considered. The concept of *construct validity* would ideally be assessed by determination of dietary intake in comparison with an unbiased method (van Staveren and Burema, 1982). However if there is no unbiased method (or 'gold standard') of dietary assessment against which other methods may be calibrated (Beaton *et al*, 1983), construct validity can be verified only by an independent check, such as biological measurement (section 1.7.4), or *"if the food intakes have been observed by the investigators"* (Bingham, 1987). Direct, discrete observation of actual dietary intake of individuals is clearly not possible in most studies of *"free-living"* individuals (Bingham, 1987) and could be expected, due to cultural and ethical reasons, to be particularly difficult in Aboriginal communities. Therefore most studies purporting to assess the accuracy of dietary intake methodology actually compare two non-perfect methods in order to determine *congruent validity* or *relative validity* (Marr, 1971; Bingham, 1987). When comparison of the results of two dietary survey methods indicates significant differences, all that is known is that

systematic error is present in either one or both methods. *Face validity* reflects the extent that a method appears to measure what it purports to measure (van Staveren and Burema, 1982). *Internal validity* refers to inference about the subjects actually participating in the study and is affected by accuracy of the method, whereas *external validity* refers to inference beyond the study population (van Staveren and Burema, 1982). Several reviews of dietary survey methodologies have summarised the results of comparative studies, enabling an impression of the likely type and extent of systematic error to be made (Young and Trulson, 1960; Pekkarinen, 1970; Marr, 1971; Morgan *et al*, 1978; Beaton *et al*, 1979, 1983; Bazzarre and Myers, 1980; Krantzler *et al*, 1982; Simopoulos, 1982; NRC, (1985); Bingham, 1987; Willett, 1990:54-62). Likely sources of error in common dietary survey methods (based on Bingham, 1987) are presented in Table 1.2.

Comparative studies have generally reported statistical results in terms of correlation coefficients, which tend to depend on the range of observations and do not truly reflect a measure of accuracy of the methods (Woolf, 1954; MRC, 1983:14; Bland and Altman, 1986; Bingham, 1987).

Table 1.2. Sources of error in dietary surveys

Source	Records Weighed	Records Estimated	24-hour Recall	Diet history	Food Frequency
Sampling bias	+	+	+	+	+
Response bias	±	±	±	+	±
Reporting errors					
-wrong food	-	-	+	+	+
-wrong weight	-	+	+	+	+
-wrong frequency	-	-	-	+	+
Coding errors	+	+	+	+	+
Food tables	+	+	+	+	+
Dietary change	±	±	-	-	-

+ Error known to be present - Error not present ± Error may be present

It has been frequently considered that the subjects' perception of the importance and applicability of dietary survey results is the most important factor affecting response rates and compliance (Bingham, 1982). This observation is likely to be important in dietary studies in Aboriginal communities, where the significance of quantitative dietary data may not appear immediately relevant to the general community.

1.7.3.1. Weighed intake studies

Recorded methods are thought to be less prone to systematic error and easier to standardise than recall methods (Bingham, 1987) and the precise weighing method has commonly been used as a basis for comparison with other methods (Pekkarinen, 1970). However dietary record methods tend to be invasive and place a substantial burden on the subject. Such methods are believed to be limited to literate, highly co-operative and motivated subjects (Mahalko *et al*, 1985). It is also believed that the weighing of food tends to alter dietary intake (Trulson and McCann, 1959; Mahalko *et al*, 1985; Prentice *et al*, 1986). The reported intakes of certain foods have also considered to reflect perceived social desirability (Worsley *et al*, 1984). In relatively few studies has the observer weighed all the foods, which would be necessary in recorded studies of the diet of semi-literate and/or semi-numerate Aboriginal groups.

1.7.3.2. 24-hour recall

"Daily variation is the main factor determining the precision of recording methods of assessing dietary intake and the 24-hour recall" (Bingham, 1987). The success of the method depends on the subject's memory, ability to communicate and willingness to participate (Willett, 1990:52). On balance of evidence, the 24-hour recall method has consistently been shown to underestimate weighed dietary intake and is not considered an appropriate method to determine the usual diet of an individual (Acheson *et al*, 1980; Baghurst and Baghurst, 1981; Hautvast and Klaver, 1982:61; Kerr *et al*, 1982; Bingham, 1987; Willett, 1990:63). The 24-hour recall has frequently been used to assess the average intake of groups (Block, 1982; Hautvast and Klaver, 1982:61; Beaton *et al*, 1983). However the associated bias of under-recall has often been shown to be greater than the difference to be detected between groups (Baghurst and Baghurst, 1981; Bingham, 1987). It has been speculated that it is easier to alter dietary history during the course of an interview than to alter the actual diet consumed during a period of recorded intake (Keys, 1965; Karvetti and Knuts, 1985). There is evidence that subjects tend to under-recall

observed large food intakes and over-recall relatively small intakes, producing a "*flat-slope syndrome*" (Young, 1952; Madden *et al*, 1976). Social status, attitudes and personal food habits of the interviewer may influence recall of the subject (Willett, 1990:56).

1.7.3.3. Diet history

The diet history method tends to overestimate dietary intake compared with weighed record of food intake (Baghurst and Baghurst, 1981; Bingham, 1987). Systematic errors associated with bias in the diet history method, tend to be of the same order of magnitude as, or greater than, the likely differences between groups (Bingham, 1987).

1.7.3.4. Food frequency

The validity of FFQs appears to be largely dependent on the questionnaire design (Baghurst and Baghurst, 1981). Possible issues to be addressed in design include specific measurements and goals, instrument length, possible response biases, instructions, wording, subject memory processing capacities and the ease with which the target population can respond to questions relating to serve sizes and conceptualise the time reference (Baghurst and Baghurst, 1981; Worsley, 1981; Willett, 1990:69-89). Validation studies cannot be generalised to different populations and to modifications of design. Reproducibility of food frequency questionnaires has been found to be satisfactory (Baghurst and Baghurst, 1981; Willett, 1990:96-97). FFQs have not generally been shown to classify individuals accurately into extremes of distribution of nutrient intake of the population (Bingham, 1987), but may be more valuable for assessing the usual dietary intake of groups (Willet, 1990:96-115).

1.7.4. The relationship between dietary intake and biological indicators of nutritional status

Biological markers may function as independent checks of validity of dietary survey methods (Bingham, 1983, 1987). However "*for a calculated intake and biochemical indicator to be correlated requires that intake of foods be accurately reported, the nutrient content of the foods be known accurately and be constant within specific foods, the nutrients be similarly bioavailable from the different foods, and the level of the biochemical indicator be responsive to nutrient intake over the range being studied*" (Willett, 1989:27). Response to dietary intake is affected by several factors. Homeostatic

processes control the concentration of many nutrients in body compartments, operating through such mechanisms as saturation of absorption, excretion of excess or by complex hormonal pathways, and tend to "*cause the increase in biological levels to be attenuated, or plateau, with higher nutrient intake*" (Brubacher, 1982; Hunter, 1990). The sensitivity of the biological indicator to dietary intake over time should also be considered. If the intra-individual variation in a biochemical marker is large, the power of a single measurement to predict 'usual' dietary intake will be low, as the indicator is likely to fluctuate in response to short-term variations in dietary intake (Hunter, 1990). Non-dietary factors, such as genetic variation, environmental conditions, smoking status, obesity, gender, age and concentrations of associated biochemical markers may also affect the concentration of biochemical indicators (Brubacher, 1982; Hunter, 1990). As intakes of specific nutrients also tend to be highly correlated in the diet, associations of a biological indicator with one nutrient may be confounded by other aspects of the diet.

Biochemical measurements are also subject to problems of measurement error (Hunter, 1990). Factors affecting the reliability of biochemical markers include contamination, stability of samples and the varying analytical methods applied, and are affected by such processes as the timing of sample collection, the collection process, sample storage and analytic techniques. Errors may be minimised by the adoption of both specimen and analytic quality controls (Hunter, 1990).

No accepted markers for available carbohydrate or alcohol intake are available (Bingham, 1987). Without acceptable methods to independently determine the dietary intake of such energy-providing macronutrients, it is not possible to estimate the total energy intake. However the estimated habitual energy intake of groups of individuals may be compared with measurements of energy expenditure (Prentice *et al*, 1986; Bingham, 1987). In most circumstances, 24-hour urine nitrogen may identify systematic bias in measure of total protein intake (Bingham, 1987). The adipose tissue concentration of fatty acids has been shown to be a useful marker of the type of fatty acids consumed by groups (Beynen, 1982; Beynen *et al*, 1980). Urinary sodium and potassium excretion are also accepted measures of intake (Hunter, 1990). A positive correlation has been found between dietary iron intake measured by 7-day food records and serum ferritin (Mahalko *et al*, 1985). More generally serum ferritin has been shown to be correlated with meat intake (the major source of haem iron), but not total iron intake (Hunter, 1990). Faecal weight and dietary intake of non-starch polysaccharides tend to be positively correlated (Cummings, 1981; Bingham, 1987).

Although strong associations between diet and serum cholesterol levels have been demonstrated for countries (Keys, 1970; Keys *et al*, 1986) and groups (Gordon *et al*, 1981; Shekelle *et al*, 1981; Kushi *et al*, 1985; McGee *et al*, 1985), correlation between dietary intake and individual serum cholesterol level would be expected to be low due to: the high inter-individual variation in cholesterol level (Keys, 1970; Liu *et al*, 1978); the error of dietary measure (Bingham, 1982); the reduced variability of exposure to risk factors within homogeneous populations (Rose, 1985); and the complex interaction of dietary factors involved (Mattson and Grundy, 1985; Willett, 1990:345). Such issues may help to explain the difficulty in demonstrating relationships between individual dietary intake and serum cholesterol levels (Nichols *et al*, 1976; Gordon *et al*, 1981). Some studies have demonstrated positive associations between serum cholesterol and percentage of dietary energy derived from total fat, saturated fat and serum cholesterol, and negative associations with percentage of energy derived from total and complex carbohydrate (Garcia-Palmieri *et al*, 1980; Shekelle *et al*, 1981). However lipoprotein levels and total serum cholesterol have not generally been shown to be sensitive indicators of dietary cholesterol and fatty acid intake (Hunter, 1990).

"The present biochemical indicators of vitamin status may be a good estimate of the average vitamin intake of groups" (Brubacher, 1982). Many confounding variables are known to affect the relationship between measurement of the intake of vitamins and apparent vitamin status (Brubacher, 1982). Although correlation coefficients tend to be low, biochemical indicators of several vitamins have been shown to be significantly correlated with individual dietary intake: plasma vitamin C and serum folate concentration are correlated with short-term intake of the respective vitamins; plasma β -carotene and red blood cell folate concentration are sensitive indicators of longer term intake; plasma α -tocopherol concentration can represent long term dietary intake of vitamin E when adjusted for total plasma cholesterol (Brubacher, 1982; Hunter, 1990). The serum concentration of other B group vitamins may reflect dietary intake in populations with sub-clinical deficiency and low dietary intake (Hunter, 1990). There is evidence that the mean value of such biochemical measurements may reflect the mean vitamin intake of a specific group of the population (Brubacher, 1982).

1.7.5. Nutrient intakes and food consumption data

Nutrient intake is dependent on choice of food rather than nutrients. Therefore dietary recommendations based on intakes of specific foods are generally more meaningful than

those based on nutrients, particularly for Aboriginal groups. However, practical problems with consideration of dietary intake in terms of foods include their large number, complex inter-relationships, varied nutrient composition and complexity (Willet, 1990:24). The representation of dietary intake in terms of composite nutrients is advantageous as it enables exploration of the relationship between biological metabolism and diet.

The calculation of nutrient intake from food consumption data requires accurate data. Many assumptions are made in the compilation of food composition data (Baghurst and Baghurst, 1989). However, the effect of the random error introduced by the use of analytical averages of representative food samples has been shown to be reduced when the nutrient intake has been calculated for either a large number of individuals and/or for individuals over an extended time (Widdowson and McCance, 1943). Under these conditions, and in the absence of systematic errors, the errors incurred in the use of food composition tables may be less than expected (Bingham, 1987; Willett, 1990:28). Food composition data should be as current and accurate as possible, utilise the same method of analysis for all foods, be comprehensive in terms of variety of foods included, complete in nutrient analysis and comprehensive in terms of nutrients analysed (Willet, 1990:28-29).

1.8. Quantitative measurement of dietary intake in Aboriginal communities

"The need for accurate, quantitative dietary intake data can not be overstressed. Such data is essential as a base from which to work in dietary intervention programs. There is a need for the systematic evaluation of the currently available methodology for dietary intake assessment and, if necessary, new and more appropriate approaches to this difficult problem may have to be developed" (O'Dea, 1986b).

Qualitative descriptions of post-contact Aboriginal nutrition have illustrated such a poor diet that one may assume a detailed knowledge of dietary intake was not required to devise strategies to improve dietary habits. But a non-invasive dietary survey methodology to measure food and nutrient intake accurately in remote, centralised Aboriginal communities would have several applications and enable:

1. Evaluation of the nutritional adequacy of diets of Aboriginal groups
2. Evaluation of nutrition education and intervention programs
3. Correlation between dietary and physiological variables

4. Dietary comparison between different Aboriginal groups
5. Comparison of the validity and reliability of other dietary methodologies applied in Aboriginal communities

Many qualitative observations have been made concerning contemporary dietary intake in Aboriginal communities. However, with two exceptions where ration listings were used (Wilson, 1953; McArthur, 1960b), only recently has there been any attempt to obtain quantitative data. Quantitative methods applied for this purpose have rarely been satisfactorily described and many methodological problems exist.

Many subjective assessments of the diet have been made by observers with little formal nutritional training or background. For example, at Ernabella the results of an 'incomplete' household food consumption survey, which were also claimed to be supported by unpublished store purchase observations, were used to justify the claim that: "*The mission has been largely successful in conveying to Aborigines the connection between various imported and introduced foods, and the specific and general nutritional needs of infants, children, adults and pregnant and nursing women*" (Silberbauer, 1971). In particular, generalisations and extrapolations from a discussion of one component of the diet, particularly estimations of total energy and protein intake, have been made to comment on the adequacy of the total diet. Unsupported quantitative assessments have also frequently been made. For example it has been estimated that 61.5% of Aboriginal people, compared with only 10.9% of Europeans, ate "*below average diets*" in a general practice in a rural town in New South Wales (Coolican, 1974:128). Unfortunately dietary studies have generally not corresponded in time or locality with health studies (Moodie, 1981).

1.8.1. Factors affecting measurement of dietary intake in Aboriginal communities

In addition to the well-recognised limitations of most dietary intake measurements in populations (Black, 1981; MRC, 1983; Willett, 1990:62-64), there are many methodological problems specific to dietary studies in Aboriginal communities (Coles-Rutishauser, 1985). The general problem of very large intra- and inter-individual variance in individual daily dietary intake in similar communities has been highlighted (Willett, 1990:45,46). The marked fortnightly purchasing cycle and the associated variation in dietary intake in Aboriginal communities has been described frequently (Middleton and Francis, 1976:90; Wise *et al*, 1976; Cutter, 1978; Young, 1984:84; Knight and Rowse, 1990). Methods to measure the usual dietary intake of individuals and groups should be

applied over this cycle. This implies that very large numbers of subjects, appropriately extended time periods, or both, would be required to meaningfully describe usual dietary intake for groups of individuals using either the weighed intake method or the 24-hour recall method.

An unusually detailed description of methodological problems encountered in the quantitative measurement of Aboriginal dietary intake and general health research in Aboriginal communities has been described (Middleton and Francis, 1976:80, 191-206). Language dependent techniques were not possible due to limited literacy skills of the subjects and lack of skilled interpreters; precise weighing techniques and the household inventory method were not applicable due to lack of 'meals' consumed and limited storage of foods. Modification of these approaches was not considered due to the invasiveness of these methods. The authors chose to estimate the diet of the whole community by observing quantities of foods obtained from the bush, the settlement dining room and the store (Middleton and Francis, 1976:80). For many reasons, only qualitative results were able to be described (Middleton and Francis, 1976:81).

Coles-Rutishauser (1985) has more recently drawn attention to the difficulties in quantifying Aboriginal dietary intake. It was stressed that there was "*no typical Aboriginal diet*", implying problems inherent in extrapolation of dietary data between Aboriginal groups and the need to randomly select cases for study. Specific problems with measuring Aboriginal dietary intake at rural settlements were highlighted. These included the need to consider variations in food intake over the usual income cycle, wastage, seasonality of the food supply (Meehan, 1982:141-161) and consider fluctuations in availability and use of bushfoods (Rae *et al*, 1982), garden produce and sources of food other than the community store. The lack of appropriate nutrient composition data was also raised.

Low co-operation rates in dietary studies have also been a problem and have been thought to be due to the poor history of racial relationships and the resultant suspicions of Aboriginal people towards non-Aborigines (Frith *et al*, 1974; Sibthorpe, 1989:157). Particular facets of this problem, such as difficulty in obtaining access to homes (Frith *et al*, 1974:59) and the aspect of "*shame*" associated with response to direct questioning by non-Aboriginal researchers, have been described (Sibthorpe, 1989:158). Many other problems have been noted (Middleton and Francis, 1976:81, 199-206). The value of interpreters was questioned as they appeared to prompt and edit responses and may have increased the respondent's discomfort if of an inappropriate kin relationship or sex. The

language used for testing was thought to affect response and it was considered advantageous (though highly unlikely in practice) for the researcher to be fluent in the language and the culture of the subjects. Difficulty in translation of various concepts was also noted; concepts of causality, seriation and quantification were thought to be different in Aboriginal groups. Perception and interpretation of 'European-style' photographs and drawings used as visual cues were questioned. There appeared to be an (unmeasurable) response bias to attitudinal questioning, with answers revealing "*what the Aborigines know of European views rather than what they themselves believe*" (Middleton and Francis, 1976:205). It was thought that such responses indicated a tendency for Aboriginal people to acquiesce. A general reluctance to answer was also noted; it was thought to have been due to passive resistance to 'demanding Europeans' (Hamilton, 1971), or to lack of confidence, ignorance or boredom. A critique of methods used to interview Aboriginal people during a demographic study in south-eastern Australia has highlighted the need to establish trust with the subjects, respect individual privacy and express questions in non-complicated language (Vesper, 1987). Other considerations have not been raised in the literature. These include the mobility of traditionally-orientated Aboriginal people and perceptions of the value of measurement, particularly of dietary intake, which may be perceived as being of a very personal nature in Aboriginal groups.

1.8.2. Measurement of dietary intake in Aboriginal individuals

Studies attempting to measure the usual dietary intake of Aboriginal individuals have addressed a wide variety of Aboriginal groups living in many environments. Although the most popular method to describe usual intake of groups of Aboriginal individuals, particularly children, has been the 24-hour recall method (Hitchcock and Gracey, 1975a; Allen *et al*, 1977; King *et al*, 1985; King *et al*, 1988), a variety of other methods has been applied. For these reasons comparison of results is difficult, and, in the following discussion, each study will be considered separately.

Qualitative description of dietary intake for the "*part Aboriginal*" population of Bourke, was collected by observation and questioning at three different periods. Data was collected by researchers who were known to the subjects (Kamien *et al*, 1974). No other detail of the methodology applied in this descriptive phase of the study was provided. On the basis of these observations it was argued that the 'staple diet' consisted of "*overcooked stews, fried flour, syrup and aerated waters*", which was considered to be "*almost devoid of any vitamin value*" (Kamien *et al*, 1974:129). A quantitative assessment of the diet followed

(Kamien *et al*, 1975a) with the measurement of the dietary intake of 17 people (4 adults and 13 children) in two 'fringe dwelling' families over six days. Families were chosen for participation on the basis that they qualitatively represented the best living conditions and dietary habits of their respective sections of the community - town and reserve dwellers. Food was weighed and aliquots were analysed for some vitamins, while intake of energy and other nutrients was calculated using food composition tables (Thomas and Corden, 1970). Anthropometric, haematological and clinical data were also collected. The plasma concentration of several vitamins was measured but dietary and biochemical comparisons were not reported. It was noted that alcohol consumption ceased during the survey period, and that children appeared to reduce dietary intake during the first few days, but '*ate normally*' later. However no attempt was made to justify this perception of 'normal' eating and it is difficult to establish the effect of observer bias in this study. Reported energy intakes were very low and may have been influenced by the study process. Apart from the report that fruit was consumed within two days of purchase, there was no mention of the likely change in diet with availability of money which may have affected the significance of intake measured for this short period of consecutive days. Dietary intakes were found to be generally low with respect to energy, calcium, iron, ascorbic acid, thiamine and vitamin E. Popular foods were white bread, both plain and fried damper, golden syrup, jam and honey. The weighed intake of sugar appeared surprisingly low, as did the contribution of refined carbohydrate to the total diet (approximately 14%).

During the Davenport survey (Wise *et al*, 1970), dietary intake was estimated for a random sample of 98 out of 149 adult individuals by a dietitian using a diet history method. No details of the dietary methodology, the methods of sampling, the method of analysis or the composition of the sample were provided, although it was suggested that the results probably reflected consumption on a day of high intake. Very high daily variability was reported; for example energy results were in the range 7,560 to 13,860 kJ/adult male/day and 6,300 to 7,980 kJ/adult female/day.

The dietary intake of a small group of urban Aboriginal adults living in Kempsey, was determined by a series of four or less 24-hour dietary recalls obtained over several months (Sibthorpe, 1989:155-178). Although initial attempts were made to obtain a randomised sample of participants, householders with an "*alcohol problem*" were excluded and those who chose not to participate, or who eventually dropped out, were replaced by individuals with whom the researcher "*became better acquainted*". The ultimate sample was small (n=38) and appeared to be highly biased. A greater proportion of the sample was obese,

female and of higher socio-economic class and employment rate than the general population of Kempsey. Of the sample, 26% were diabetic compared to only 10% of the general population; all diabetics included in the study had previously received dietary advice. It was considered that "*as diabetics had to take an interest in their diets in order to control their disease.. they were more willing to participate in a dietary survey*" (Sibthorpe, 1989:173). Three of the sample (8%) were 'vegetarian', being Seventh Day Adventists, while 57.5% of all respondents were "*on diets of some sort*" (Sibthorpe, 1989:259). Such bias may help to explain some unusual aspects of the results, such as the relatively low reported intake of sugar, meat and meat products. Results suggested a very low mean intake of energy (males 8402 ± 2276 kJ, females 5589 ± 1802 kJ) and associated low intake of most nutrients. The researcher considered, particularly as there was little evidence of previous dietary compliance in obese and/or diabetic subjects (Sibthorpe, 1989:259), that a degree of under-reporting, or modification of the 'usual' diet, may have contributed to such low values. Anthropometric data, which may have revealed useful data in this regard, was not collected. The nutrient density of the diet was comparable to that described by the Australian dietary survey (CDCSH, 1987a). However there is no evidence that *selective* reporting on the day of recall did not occur during the Kempsey study. The dietary intake of such a sample could not be considered representative of the general diet of Kempsey Aboriginals or of other Aboriginal groups. However results were used to criticise assumptions made about general urban Aboriginal diets: "*the high fat, high sugar, largely vitamin-deficient diet frequently assumed to be ubiquitous in Aboriginal communities is not supported..*". The claim that "*similar studies of dietary practices in other urban communities would yield similar results*" (Sibthorpe, 1989:267), may be unfounded.

In an attempt to determine the 'usual' diet of 26 Aboriginal children living in three different environmental conditions in a rural Aboriginal community in South West Australia, a single 24-hour recall was conducted with their mothers (Hitchcock and Gracey, 1975a). The authors state that of a possible 21 responses only 14 meaningful results were obtained and satisfactory information about household purchases was obtained from only eight. Nutrient analysis claimed to reveal low energy and vitamin C intake in the children. However it has been observed that this study provided no evidence to suggest that the amount of energy and protein available to young children was less adequate than for other family members (Coles-Rutishauser, 1985). Bias may have been introduced due to the potentially selective reporting of 24-hour recall from groups of different educational and social background. Perhaps the major value of this study was the identification of the

differing qualitative dietary patterns observed in relation to the living conditions of different groups in the community (Hitchcock and Gracey, 1978), although stratification of results produced very small cell numbers. The most restricted diets were found to be consumed by those living at the reserve, emphasising the socio-economic component of poor quality Aboriginal diets. Data from this study has been adapted to illustrate the notion that the contrasting diets described in different environments may be due to methodological artefacts (Coles-Rutishauser, 1985).

The dietary pattern of 129 Aboriginal children aged between 8 and 12 years from 6 different Kimberley communities was investigated by 274 interviews using the 24-hour recall method collected on at least 3 occasions for each child covering different seasons (King *et al*, 1985). These results also appear to have been included in a wider study comparing 530 collected 24-hour recall interviews conducted over 3 years with 169 Aboriginal children aged 7.8 to 13.1 years, with a single set of interviews with 70 Caucasian children of similar age living in Adelaide (King *et al*, 1988). Quantitative estimates were obtained with the aid of food photographs for the major items, with children measuring similar amounts of milk powder and sugar used on each occasion the previous day into a cup for subsequent weighing. It is difficult to comment on these data as the only currently published report of the studies is in abstract form. It is possible that cultural differences affecting response to questioning and interpretation of photographs, the young and varied age of respondents and the different seasons of recording could introduce serious bias in both studies. In the first study (King *et al*, 1985) results were used to indicate the frequency of consumption of particular foods. Meat, flour, sugar and powdered milk were claimed to be staple items. Foods consumed more than twice a day included tea, sugar, milk. Those consumed once or twice a day included bread, butter, cordial or soft drink. Foods consumed three to six times per week were beef, fresh fruit, rice and jam. Cereal, porridge, damper, canned meat, chicken, eggs, biscuits, sweets and soup were consumed once or twice per week. Sausage, cheese, nuts, cakes/buns, fish, kangaroo, dugong and turtle were consumed less than once a week. Although specific results were not reported it was claimed that they showed "*evidence of low energy intake and marginal to low intake of folic acid and vitamins A, B₁ and B₆. Intakes of protein, calcium, vitamin C and vitamin B₁₂ were estimated to be adequate*". In the second study (King *et al*, 1988), the estimated mean energy intake of the Aboriginal children (5308 kJ) was found to be significantly less than that of the Caucasian children (6795 kJ); and there was no increase in energy amount with age for the Aboriginal children. The Aboriginal children had significantly lower intakes of fibre, protein, fat, calcium, vitamin D,

thiamine, riboflavin, vitamin E and vitamin B₆. There was no statistical difference between intakes of carbohydrate. However, considering the differences in dietary fibre intake described, it is likely that the Aboriginal children had a tendency to consume a greater intake of refined carbohydrate. The very low apparent intake of fat soluble vitamins was likely to be due to the extremely low fat intake described in Aboriginal children. This low apparent intake of fat is surprising in view of the previous observation that meat, a significant source of saturated fat, (particularly in the low quality fatty cuts frequently supplied to remote communities), was a staple food item, and raises questions concerning the appropriateness of the nutrient data base (Baghurst and Record, 1984) used for analysis. Bush food intake for Aboriginal children was found to be very minor, providing less than one percent of total energy intake.

The dietary methods used in an ascorbic acid supplementation trial of school children has been documented in unusual detail (Allen *et al*, 1977). Sample size (29 Aboriginal and 43 European children) was estimated utilising the variance of the main variable (ascorbic acid intake) measured from a study of children in Melbourne. The authors were aware that the young subjects had difficulty in accurately estimating portion size and tended to under-report all foods eaten. Results were 'cross-checked' against the amount of money each child had available for food purchase and information about the foods sold at the school tuckshop, although details and comparisons were not provided. A greater proportion of Aboriginal children were found to have intakes of several nutrients (protein, iron, thiamine, riboflavin and ascorbic acid) which were below FAO/WHO recommendations; five had relatively low intakes for three or more nutrients (Heywood and Zed, 1977). For nutrients where estimates of the mean and variance of requirement was available, the probability of specific intakes being below the requirement was also calculated.

Dietary intake was quantitatively determined for a small group of 14 Aboriginal diabetics and others from the Kimberley region, before and twice during temporary reversion to a traditional lifestyle (O'Dea *et al*, 1987). With the exception of the initial travelling period the group existed entirely on traditional bush foods. An estimation of the diet was made during the initial 10 days travelling period and during the following two week period on the coast, and compared with an estimation of the composition of the 'usual' urban diet. During a two week period in the final phase of the study at an inland location, all food was weighed before it was eaten and samples were collected and stored in liquid nitrogen in the field, pending laboratory analysis for fatty acid composition (O'Dea and Sinclair, 1982; O'Dea *et al*, 1987). Results were presented in terms of proportional contribution of

macronutrients to the total energy intake (O'Dea, 1984; O'Dea and Sinclair, 1985). The very low energy intakes described were consistent with the observed weight loss and metabolic changes.

1.8.3. Measurement of dietary intake in traditionally-orientated outstations

The majority of community dietary data has been collected in the course of anthropological rather than health or nutritional studies. Anthropologists and economists have generally concentrated on traditionally-orientated outstations. Their agenda generally has focused on traditional hunting and foraging activities, with quantitative assessment of the diet as one factor of an extensive analysis of economic and/or social aspects of contemporary lifestyle. Most work has centred on small, isolated outstations and cannot be extrapolated to the larger, centralised communities where most Aboriginal people in the Northern Territory now live (section 1.4).

Again many methodological problems with quantifying data have been noted. To document subsistence activities of Aboriginal groups, anthropologists have generally applied conservative observational methods over an extended period although quantitative data has been collected most frequently for short periods (Meehan, 1982:45-47; Altman, 1987:31-45; Devitt, 1988:74-76). It has also been pointed out that there is no 'typical' climatic year (Meehan, 1982:146).

Other studies have been conducted for a small number of days and it is difficult to assess 'usual diet' from such data. This is particularly so for early studies which attempted to document subsistence activities of Aborigines living completely on traditional foods (McArthur, 1960c). The situations described by McArthur were also artificial (McArthur, 1960c:90-92,133; Jones, 1980:135; Jones and Bowler, 1980:20).

1.8.3.1. Bush food intake in traditionally-orientated outstations

Altman (1987:31) accompanied one production team each day to weigh and measure bush foods as they were hunted and collected. Numerous key informants were used to document returns of other production teams and results were verified by comparison with reference sample weights of game. As women-only groups were not accompanied for reasons of social propriety, direct observation of their subsistence activity and returns was not possible. A similar problem confronted Devitt (1988:81,86) who did not accompany male-

only hunting parties in her study at Utopia, which meant that a substantial proportion of her quantitative data was actually estimated. Attempting to overcome similar problems, Meehan and Jones cooperated in an attempt to document the subsistence activities of a group of Anbarra living in the Blyth River region of Arnhem Land from July 1972 to July 1973. Meehan (1982:45) concentrated on the activities of women and children and Jones on the men. But even with an observer of each sex available there were obvious difficulties including the need to report simultaneously on activities of several foraging parties in different places.

Specific problems have been frequently encountered in attempts to weigh yields of gathered fruits: "*..in general it was an impossible task. These foods are eaten by anyone whenever they are available, being swiftly taken from the tree and popped into the mouth. People resent any slowing down of this process and readily show their discontent, so I desisted*" (Meehan, 1982:148; Devitt, 1988:74). Unlike Devitt and Meehan, Altman did not estimate consumption of these foods for inclusion in nutritional analysis of the diet.

1.8.3.2. Store food intake at outstations

Because of the complexity of the food supply to the decentralised outstation communities in the Utopia region, Devitt attempted to calculate the minimum estimate of consumption of store foods on the basis of actual individual intake rather than initial purchase for three distinct periods of one month each. A "*snack factor*" was used to cover the consumption of items observed but not systematically recorded (soft drink, confectionary, potato crisps and biscuits). Copies of orders and dockets for all foods provided to the Anbarra community from Maningrida were used to quantify the European food provided to the Anbarra (Meehan, 1982:149) but other 'store foods' either provided by visitors and neighbours (Meehan, 1982:36,40) or purchased during shopping expeditions, have not been addressed in analysis. The purchases of the Momega band were documented during the regular fortnightly visit of the mobile store. Retrospective, unvalidated, oral accounts of purchases during shopping expeditions to Maningrida were also included (Altman, 1987:31).

1.8.4. Measurement of dietary intake in community settlements

Early studies (Wilson, 1953; McArthur, 1960b) relied on the questioning of people responsible for the provision of rations in government settlements and missions supported

by documented records where available. The effects of seasonality were estimated by responses to informal questioning and must also be considered to be subjective. Different methods were applied at various settlements and results would not appear to be directly comparable. Nutritional analysis of food intake was limited. Such valiant attempts to quantify Aboriginal diet were remarkable for their time; however the major value of both studies would appear to lie in the useful descriptive data provided.

1.8.4.1. Store-based studies

Most Aboriginal people in northern and central Australia now live in centralised settlements in rural areas (section 1.4). These communities are serviced by a community store which is often the only source of purchased food for several hundred kilometres (White, 1977; Duffy *et al*, 1981; Young, 1984:5; Coles-Rutishauser, 1985). As traditional food has been depleted by population pressure around these settlements and people may be disinclined to hunt and gather bush foods in the face of acceptable market substitutes (section 1.5.3.2), it can be assumed that store foods account for most of the community's nutritional intake. Unfortunately there are no detailed quantitative analyses of the variable contribution of purchased food to the diet in such communities (Sladden, 1987:137-139). It has been often assumed that most people obtain most food from the store and attempt to procure bush foods only at the weekends. Cutter estimated that of total energy intake, purchased foods contributed up to 90% on stations and settlements, and approximately 70% on outstations, occasionally dropping lower for short periods only (Cutter, 1978). Young estimated that store foods provided in excess of 80% and in some places 90% of the total nutrient intake (Young, 1984:101). Under these circumstances the turnover of foodstuffs from isolated community stores has potential as a useful source of information about contemporary dietary intake. Different approaches to the collection of store food data have been utilised previously (Coles-Rutishauser, 1979; Kailis, 1979; Peterson, 1977b; White, 1977; Taylor, 1979; Young, 1984:101-106; Sladden, 1987:138-140). However these studies have generally collected data for short periods only, often not covering the financial and associated purchasing cycle involved; have not considered the effect of seasonality; have not appraised bias due to selective reporting of store managers; have not estimated foods available from other sources; have not addressed problems with determination of the consumer population; have only been partially analysed and have not attempted to validate methods.

1.8.5. Observer bias in dietary studies

With one exception (Devitt, 1988:77-78) there has been little consideration of the effect of observer bias in dietary studies in Aboriginal communities. Shoppers may alter usual purchases under observation. Anthropologists wishing to report *traditional* subsistence activity may inadvertently de-emphasise the contribution of purchased food to the diet (for example, see Meehan, 1982:36,40,154-6).

1.8.6. Nutritional analysis

Anthropological assessment of the diet has frequently been based on determinations of gross weight of the food supply, which have little nutritional relevance, and can lead to an overestimation of the relative significance of the contribution of vegetable foods to the diet. Data have been analysed for protein and total energy content primarily, which tended to over-emphasise the nutritional contribution of meats, flour and sugar, while under-emphasising the dietary significance of other foods, such as fruits and vegetables. Such incomplete analysis does not allow for a discussion of the adequacy of the total diet, although frequently subjective assessments have been made.

The lack of complete, accurate nutrient data for both traditional foods (Coles-Rutishauser, 1985) and contemporary Australian foods (Young, 1984:102) remains a matter of concern (Australia, 1989; English, 1989). Many foods available in remote Aboriginal communities are also likely to vary from those available elsewhere; perhaps due to the effect of long-term storage and transportation of foods in less than optimal conditions, and the effect of different dietary preferences such as the increased acceptability of fatty cuts of meat.

The issue of nutrient analysis has been approached in several different ways. Meehan derived a set of "*standard food values*" which were based on food analysis tables (Thomas and Cordon, 1970), past analysis of Arnhem Land foods (Fysh, 1960) and approximations for unanalysed 'bushfoods' based on "*equivalent*" European foods (Meehan, 1982:146). However the derived composite values for all purchased foods (Meehan, 1982:149) do not appear to be founded on typical nutrient composition. Altman applied similar values to Meehan, while Devitt based the analysis of purchased foods on these tables and bush foods on the basis of analysed samples (Devitt, 1988: Appendix II). Young preferred to use British food composition tables (McCance and Widdowson, 1978).

1.8.7. Wastage factors

It has been considered that food wastage was minimal "*as the aborigines eat everything that is edible from both animal and vegetable food*" (McArthur, 1960c:128). For centralised communities, estimations of wastage have not been included in calculations (Taylor, 1979; Young, 1984:101-106). Descriptive evidence suggests that most store food is consumed immediately after purchase, particularly where storage facilities are minimal (Young, 1984:107). Under these circumstances it may be argued that wastage would be negligible.

Conversely, large estimations of wastage of purchased food stuffs have been made for outstations. Meehan, supported by Altman, applied a correction factor of 20% to allow for wastage due to: rain and water damage, spillage, inadequate storage, preparation of food and the consumption by children, or discarding of undissolved sugar in the bottom of billy tea, pillaging and/or feeding of dogs, and transfer of market goods back to service centres by visitors (Meehan, 1982:149; Altman, 1987:32). However it could be argued that this correction factor is exaggerated as the consumption of sugar residue by children is not strictly 'wastage', rain and water damage would essentially be seasonal in a tropical climate, loss during preparation of high status food is likely to be insignificant and the contribution of market foods by visitors and neighbours was not included in analysis. Devitt's (1988:82) calculations of bush food consumption at Angkwele incorporated a 20% waste factor, by weight, for all game and for fruits with thick inedible skins. Conversely Altman (1987:32) considered that all bush foods were under-reported, especially when there was a shortage and people were secretive about their household consumption. He estimated that bush foods were underestimated by at least 10% and applied a correction factor of 1.1 to all quantities recorded; this adjustment is likely to have introduced bias resulting in overestimation of the contribution to diet of animal foods hunted by men and bush foods during the wet season.

1.8.8. Determination of population denominators

The use of the population denominator in the calculation of per capita dietary intake (Altman, 1982:23,103-107; Meehan, 1982:142-3) implies that foods were distributed equally throughout the community which is unlikely to be the case. However, given the problems with the determination of accurate/unbiased individual dietary intake, it is arguably valid to ignore the variation in individual intake and use the mean as the best

available indicator of 'average consumption'.² Devitt alone included herself as a member of the small outstation population for this purpose. Of studies in centralised communities Taylor conducted a census for the relevant study month, but the source of the centralised community population data in other studies is unclear. Perhaps to avoid the difficulties with determination of population estimates, data have been presented as a function of the 'usual' intake of a hypothetical family (White, 1977; Sullivan *et al*, 1987), which must be considered to be an essentially speculative approximation.

1.8.9. The use of recommended dietary intakes

The quality of the Aboriginal diet has usually been assessed for the communities studied by comparison with calculated recommended dietary allowances, extrapolated from recommendations for non-Aboriginal populations with adjustments for age, sex, weight, activity and obstetric status. However there are major problems in attempting an assessment of the nutritional adequacy of dietary intake in the absence of biochemical measurements (McArthur, 1960c:131; MRC, 1983).

Sex-age profiles of outstations and settlements have been frequently used to compare observed per capita dietary intake with the daily requirements for a 'reference person', using standard dietary allowances for (non-Aboriginal) Australians (Thomas and Cordon, 1970), (Taylor, 1979; Meehan, 1982:156; Altman, 1987:33; Devitt, 1988:27-29). With one exception (Altman, 1982), details of the extrapolations were not provided.

Avoiding the limitations of recommended dietary intake values, Aboriginal store purchase data (Coles-Rutishauser, 1979; Kailis, 1979) have been compared with values for wider Australia obtained from national apparent consumption figures (ABS, 1979). However the different methods and sources of data used makes quantitative comparison difficult to interpret. The comparison of nutritional data of the total Australian population with data from small Aboriginal groups should be interpreted with caution. There is little data available for more relevant comparative groups, such as different socio-economic segments of the Australian population (CDCSH, 1987a:48) or non-Aboriginal groups living in remote locations.

² The prevalence of malnutrition would be higher than indicated using 'average consumption' data if dietary intake was heterogeneous in distribution.

1.8.10. Results: traditionally-orientated groups and outstations

Quantitative dietary results from anthropological studies in small, remote, decentralised, traditionally-orientated outstation communities are presented in Table 1.3.

Table 1.3. Dietary consumption in small, remote, decentralised, traditionally-orientated communities (per capita per day)

Location	Angkwele Central Australia	Momega Arnhem Land Riverine	Kopanga Arnhem Land Coastal
Tribal group	Anmatyerre	Gunwinggu	Anbarra
Researcher	Devitt	Altman	Mechan
Source of data	(1988:80-90)	(1987:31-45)	(1982:152-6)*
Study Year	1981-1982	1979-1980	1972-1973
Study Period	3 x 1 month	10 x 1 month	3.6 x 1 month
Total no. days	109	n/a	n/a
Total intake per capita/day			
Weight (g)	893	N/A	1100
Energy (kJ)	9279	11918	9010
Protein (g)	136	133	165
Total bush foods per capita/day			
Weight g (%)	342 (38.3)	N/A	803 (73.0)
Energy kJ (%)	2962 (31.9)	5526 (46.2)	4405 (48.9)
Protein g (%)	100 (73.5)	107 (79.7)	135 (82.8)
Total Store foods per capita per day			
Weight g (%)	551 (61.7)	N/A	297 (27.0)
Energy kJ (%)	6317 (68.1)	6392 (53.8)	4605 (51.1)
Protein g (%)	36 (26.5)	26 (20.3)	30 (18.2)
Reference requirements			
Energy (kJ(Cal))	8965 (2135)	10486 (2505)	8581 (2050)
Protein (g)	56	65.5	N/A.
Intake/requirement			
Energy (%)	103	114	105
Protein (%)	244	203	N/A
Intake of specific foods g per capita per day % of total energy intake.			
Sugar (g (%))	n/a (16.4)	313 (44)	n/a
Flour (g (%))	155 (n/a)	n/a (25)	n/a
Bread (g (%))	65 (n/a)	n/a (7)	n/a

* data re-analysed on the basis of mean daily per capita intake over the total number of observation days.

In addition to the above studies, per capita daily intake of approximately 310 g of sugar and 285 g of flour was recorded in a small traditionally-oriented outstation (White, 1985). Details of the methodology applied have not been published. Even in these isolated outstations purchased foods accounted for most of the energy intake. Compared with the earlier study (Meehan, 1982) there was an increased intake of store foods and total energy intake at Momega (Altman, 1987). The increased contribution of market foods reported in the most recent study (Devitt, 1988) suggested these changes may have reflected the increase in cash availability over the decade. The highest energy intakes for the Anbarra were described during the early dry season when the level of activity increased markedly. Seasonal differences in the diet of the Angkwele group in central Australia were much less marked than those of the northern tropical groups.

1.8.11. Results: community settlements

Young (1984:103) calculated that in all four communities studied, foods purchased were sufficient to meet the FAO/WHO recommended dietary intakes for energy (114% RDA), protein (267% RDA), calcium (115% RDA), ascorbic acid (131% RDA) and thiamine (99% RDA), but less than 100% RDA for iron (66% RDA), retinol (51% RDA), riboflavin (70% RDA) and niacin (87% RDA). It is unclear from analysis whether allowances had been made for the contribution of β -carotene to retinol activity. Total calculated intakes of each nutrient were provided on a community basis, but as the population base used was not provided, mean per capita intake cannot be calculated from published results. The major foods contributing to each nutrient were listed with the percentage contribution to total intake.

Taylor (1979) presented quantitative results in the form of mean per capita nutrient intake expressed as a percentage of the recommended intake for the community as a whole. Nutrient results varied widely. The per capita daily sugar intake was 202 g in 1970 and 245 g in 1972. The lack of variety in the diet was indicated by the fact that 75% of the food dollar was spent on fresh meat, sugar, white flour, tinned meat and tea.

Peterson (1977b) listed foods purchased at Warburton on a 'normal' morning: flour 50 x 11.5 kg, sugar 34 kg, tea 2.3 kg, tinned meat 96 tins, spaghetti and meat balls 48 tins, jam 24 tins, fruit juice 72 cans, biscuits 96 packets, plum pudding 36 tins, tinned vegetables 24 tins, tinned milk 24 tins and cheese 48 x 0.25 kg pkts. Fresh fruit was available twice a month and fresh meat irregularly. The data indicated a daily per capita

intake of sugar and flour in the order of 75 g and 752 g respectively.

White (1977) calculated that an average weekly purchase for a family of five would include 11.34 kg white flour, 2.72 kg sugar, 230-460 g tea, 0.68 kg tinned meat, 0.68 kg jam, 0.85 kg can fruit, 2.72 kg 'lean' mutton, 0.23 kg butter, 3.63 kg white bread, 0.45 kg full cream milk powder, 0.45 kg 'semisweet' biscuits, 6 pieces of fruit and 12 eggs, with an additional one litre of soft drink and three packets of potato crisps for each child over the age of two years and two litres of beer for each adult. The per capita daily intake of sugar and flour indicated from this data was 78 g and 325 g respectively.

Kailis (1979) described a higher apparent daily per capita turnover of sugar at approximately 315 g. Other foods were purchased at the per capita daily rate of bread 107 g, apples 36 g, oranges 58 g, flour 53 g, chicken 32 g, meat 29 g and powdered milk 25 g. Nutritional analysis of food data was not attempted.

Recalculated as apparent intake per capita per day, the results of analysis of store orders in another northern coastal community included sugar 196 g, flour 129 g, sweetened carbonated beverages 223 ml, fruit 36 g and fruit juice 83 ml (Sladden, 1987). The store manager's assessment of the regular weekly order tended to relatively overestimate the turnover of sugar, flour and fruit.

The 'usual' purchases of a family of five for a fortnight (Sullivan *et al*, 1987) were estimated to include 4 kg sugar, (57 g per person per day), flour 10 kg (142 g per person per day) and soft drinks 40 cans (214 ml per person per day). It was stated that savoury and salty foods were found to be more popular than sweet foods including frozen ices, but this observation may be explained by selective reporting of store managers' assessment of the diet, and a failure to address the seasonal pattern of food demand and supply.

For the Aboriginal community studied by Coles-Rutishauser (1979) results were energy 16,800 kJ, protein 104 g, iron 13 mg, calcium 560 mg, retinol 610 μ g and ascorbic acid 33 mg and were similar to apparent consumption data for wider Australia. However there was a marked difference in the types of foods contributing to the nutrient intake in both communities. The main difference described concerned the apparent consumption of sugar, with sugars and syrups contributing 5,100 kJ per head per day in the Aboriginal community and only 2,400 kJ/person/day in wider Australia. Grain products also contributed substantially to the Aboriginal diet (5,750 kJ/person/day compared to 3,450

kJ/person/day), but fruit and vegetables contributed relatively less than in wider Australia (650 kJ/person/day compared with 1,000 kJ/person/day). These differences are better illustrated by presentation of results in terms of total energy density. For example, sugar provided 30% of total energy intake in the Aboriginal community compared with 17.5% for wider Australia.

1.9. Improving Aboriginal health and nutrition

"Unless the community defines the need, any attempt to conduct a (nutrition) program will be an imposition and chances of persistent behaviour changes will be minimal" (Stacy, 1975).

For many years it has been argued that Aboriginal health is a complex political problem with answers lying, not in the increasing medicalisation of clinical 'ill health' services, but rather in the empowerment of Aboriginal people to determine their own social and economic development (Moodie, 1972; Downing, 1974; Hamilton, 1974; Stanner, 1974; Tatz, 1974; Australia, 1979; NAHS, 1989). Successful improvement of the diet is widely acknowledged to require the initiation, development and 'ownership' of culturally-appropriate nutrition projects by Aboriginal communities themselves (Reid and Mununggurr, 1977; White, 1977:290; Australia, 1979:73-74; Scrimgeour, 1983; O'Dea *et al.*, 1990).

Although nutrition-related health problems have been frequently described in Aboriginal communities, no well-conducted, randomised controlled intervention trials have been evaluated (Peach, 1986). Despite growing awareness of the importance of nutrition by Aboriginal Health Workers has been noted (Sykes, 1977), there is no successful model for Aboriginal communities wishing to improve their nutritional health. Against this apparent need it is relevant to consider past nutrition intervention programs.

1.9.1. Past Aboriginal nutrition programs in the Northern Territory

1.9.1.1. Nutrition education

The earliest nutrition intervention to be introduced in Aboriginal communities was the establishment of communal feeding centres. Although the most efficient method of feeding large numbers, the feeding centres were generally ineffective as they tended to disregard

Aboriginal attitudes towards infant feeding and family food distribution practices and were generally unpopular (Jacob and Clements, 1968; Tilley, 1974; Middleton and Francis, 1976:87). The educative aims of such programs were generally not perceived to have been met as, although people were provided with an opportunity to taste European food, "*it would not be immediately apparent what ingredients should be brought from the store to prepare it*" (Middleton and Francis, 1976:87). Communal feeding removed the responsibility for feeding children from the mother, her sisters, the grandmother and the extended family, thus supporting the development of dependency rather than independence. The unsuccessful communal feeding schemes have been described as the "*height of ethnocentrism with regard to food*" and "*seemed to encapsulate the attitude that Aborigines were incapable of feeding themselves*" (Thomson, 1982).

Similar criticism could be addressed to nutritional supplementation programs during the sixties and seventies, which would appear, in hindsight, to have encouraged a 'hand out' mentality. From an Aboriginal perspective, contradictions would appear to have existed in these programs, in that people with growth retarded children were 'rewarded' by the provision of foods supplements, while those with well nourished children received nothing (Rassaby, 1978; Donald, 1980).

Most early attempts at Aboriginal nutrition education were targeted towards the problem of malnourished infants; this 'need' being wholly defined and perceived by Europeans. For many years attempts have been made to teach Aboriginal mothers about European concepts of nutritional health (Stacy, 1977). In particular nutrition education has long been a component of 'under five' or 'well baby' clinics, where children are weighed, immunised and receive clinical treatment (Morley, 1973).

In 1971 the Institute of Aboriginal Development (IAD) in Alice Springs initiated a health education program with Pitjantjatjara people (Downing, 1974). Despite high initial expectations and techniques which were 'radical' for their time (incorporating the adoption of 'two-way' teaching methods), the objectives of the nutrition component of the course were generally not achieved (Stacy, 1977). Although people could accurately repeat facts taught during the courses, there was no evidence that the knowledge had been internalised or initiated behavioural change. Stacy argued that cultural differences in perceived value of the course accounted for its 'failure' (Stacy, 1975). "*It would appear that I was busy building relationships so I could teach, and the Aborigines were participating in the program so they could build relationships*" (Stacy, 1977).

An early pilot project of nutrition education directed towards the prevention of malnutrition in children has been described (Tilley, 1974). Group activities used resources and facilities familiar and available to mothers. The sessions were reported to be popular, and it was claimed that "*a definite improvement in the nutritional status of the children*" occurred. However no formal evaluation was conducted and the project continued for only four months.

In the early years health education was initially the domain of nursing sisters resident in settlements and missions, and was conducted in a varied, usually '*ad hoc*' fashion depending on the individual sister's perceptions, priorities and skills (Soong, 1975). Nutrition has also been a component of the health syllabus taught in primary schools at settlements. Prior to 1978 a sole dietitian was employed in the Northern Territory, with major responsibilities being for nutrition surveillance and planning, including the monitoring of ration scales and communal feeding programs, rather than nutrition education (Wilson, 1953, 1954; Sinclair, 1968; NTHD, 1977; Rae, 1988). Since then, dietitians have been employed in the main population centres by the now Northern Territory Department of Health and Community Services (NTDHCS). Although departmental dietitians work with all sections of the community, the special nutritional needs of Aboriginal people have been seen as a priority (Shelley, 1982). However, poor roads and air services, seasonal weather conditions, economic considerations related to the expense of travel, the number of communities to be covered and the vast distances involved, were seen to have precluded dietitians as effective first contact community nutrition educators (Shelley, 1982). Hence dietitians aimed to train community-based Aboriginal Health Workers as 'food educators'; Health Workers were encouraged to acquire food knowledge, share that knowledge with others and promote action to improve availability of appropriate foods. A 'generic' approach to nutrition education was adopted, which aimed to incorporate bi-cultural education techniques, translate scientific nutrition information, establish the pattern of food availability and distribution, identify problems, formulate objectives with the community, plan strategies, devise tools to be utilised, implement strategies, evaluate programs, assist Aboriginal people to acquire food knowledge and the skills to use it and, ultimately, the power to be self-determining (Shelley, 1982; Lee, 1985; Rae, 1988).

Individual dietitians have worked for many years with community groups in these endeavours and have developed excellent communication and relationships with Aboriginal Health Workers and other individual community members. Various Aboriginal nutrition

education aids and resources have also been produced in the Northern Territory (Shelley *et al*, 1981; Shelley, 1982). However the continuing high rates of morbidity associated with poor Aboriginal nutritional status indicated that dietitians had not been able to transmit the necessary nutrition messages to the Aboriginal population (Rae, 1988). Limitations of the health promotion role of Aboriginal Health Workers have also been raised in the face of clinical demands, particularly those facets brought about by the "*impossibly huge and vague mandate*" (Rebgetz, 1985) of their role as "*cultural brokers*" (Soong, 1981:120-129). Despite many requests and submissions for specialised Aboriginal Health Workers to be employed to work with departmental dietitians as community-based Aboriginal Nutrition Workers (WHO, 1986b:1-2), funding had not been forthcoming (Rae, 1988).

1.9.1.2. The food supply and nutrition

It has frequently been noted that manipulation of the food supply would appear to be a simple solution to nutritional problems in Aboriginal communities, and that many dietary deficiencies could be overcome by the increased consumption of only a few nutrient rich foods (Young, 1984:104). One early example was the fortification of the bread available in a rural community with iron, thiamine and niacin (Kamien *et al*, 1974, 1975a, 1975b; Kamien, 1987). After six and a half months, remeasurement of vitamin status indicated a significant improvement in serum levels of thiamine and pyridoxal phosphate in 44% and 52% of the subjects respectively. As other vitamin levels remained similar to the initial measurements, these increases were attributed to the consumption of the fortified bread. The initiative was eventually ceased due to public outcry about 'experimentation on human beings' (Kamien, 1987). The authors also claimed that attempts at more conventional nutrition education met with little success (Kamien *et al*, 1974), but that advice on a more individual basis following biochemical and dietary intake measurements at family level appeared to be better accepted (Kamien *et al*, 1975a).

More recently, as community stores are generally the only food outlet available, they have frequently been the target for 'supply manipulation' programs. However, judging the paternalistic nature of such attempts, and considering that supply and demand are tightly interwoven in these stores, it is not surprising most attempts have failed (Young, 1984:5,106). Several factors have been suggested to hinder the development of a store nutrition policy (Young, 1984:106-107). However some specific store-based dietary changes have been described anecdotally (Young, 1984:104-106). "*Where members of the Aboriginal community.. decide, after wide consultation, to ask the store to introduce*

practices aimed at improving nutrition then some degree of success is possible" (Young, 1984:108).

1.9.2. Lessons to be learnt from past programs

Sykes has recognised that the provision of nutrition education programs may be blocked by many factors including emotional dependence of Aboriginal people to a 'familiar' diet, peer group pressure, budgeting considerations and poor self-esteem supported by "*the psychology of the history of community failure*" (Sykes, 1977). Aboriginal attitudes to health and food continue to affect the development and delivery of appropriate nutrition programs.

Past nutrition programs have tended to focus on children. However, from a cultural perspective it would appear to be more appropriate to work with Aboriginal adults, particularly elders and key-community members. "*The reason why we established some settlements was that it was thought it gave us the best chance of working with the children.. When we had succeeded with the children the settlements would wither away within a generation or two. That idea.. has wrought havoc in Aboriginal life. It has never worked and never will as long as parents care for children and children look to parents*" (Stanner, 1974:7-8). It has been argued that "*The traditional self-management of health problems within the extended family groups needs to be revived*" (White, 1977:290), particularly as the family represents the only security Aboriginal people are likely to know in present day white Australia (Wadsworth, 1984).

In particular, the role of Aboriginal women in the health system has been overlooked and trivialised and should be recognised in health programs (Bell, 1983; Gale, 1983). It was frequently assumed that Aboriginal "*mothers especially are not aware that a diet consisting primarily of sugary and fatty ready prepared foods is nutritionally damaging*" (Australia, 1979:20). However early reports indicated that mothers did have a 'working' knowledge of European foods (Tilley, 1974) and could repeat European nutrition concepts (Stacy, 1975). There appeared to be no lack of European nutrition knowledge, rather a lack of internalisation and lack of opportunity to act on that knowledge.

Often the potential impact of the community store has been overlooked in medically-orientated nutrition programs. "*The store manager is a very important person. If he is unwilling to help by stocking and encouraging people to buy better foods, little will*

change" (Rowley and Rowley, 1978).

Aboriginal commentators have suggested that cross-cultural communication is essential to the development of effective health programs (Lester, 1974). Certain qualities and communication skills would appear to be necessary in European health professionals working in a cross-cultural situation. *"Only after a considerable period of time and the repeated demonstration of trustworthiness can the medical assistant be perceived as a helpful human being and not as an alien intruder"* (Hamilton, 1974:22). However the transient nature of European health professionals working in Aboriginal health in relatively remote areas has generally precluded the development of long term individual relationships (Rebgetz, 1985).

European health professionals should be aware of several specific traditional values and beliefs which may affect health and nutrition programs in remote communities. For example, when working with Aboriginal Health Workers it is important to realise that they may be classified in traditional avoidance relationships and it may be inappropriate for them to treat specific community members (Australia, 1979:71). Traditional values may decree that it is unacceptable for both sexes to work closely together. It is also relevant to consider Aboriginal attitudes towards biological sampling in the context of medical research: *"..Blood is regarded as powerful and, according to context, a source of either strength or danger"* (Australia, 1979:73). The condition of a person who had shed blood has been described as *"socially marginal and physically dangerous"* (Reid, 1986). The belief that faeces could be used in sorcery has also been known to affect the process of medical research: *"Considerable difficulty was encountered.. in persuading the aborigines to produce stool specimens, no doubt because of their belief in the ill effects of magic performed over an individual's excreta by an enemy. For this reason some substitutes were made, even after a lengthy explanation had been made giving the reason for the collection"* (Billington, 1960:46). Such beliefs may preclude some forms of biological sampling in traditionally-orientated communities.

A holistic approach to health, incorporating Aboriginal beliefs concerning the inter-relationship of individual health and concepts of the role of land, religion and social relationships should be fostered (CDCSH, 1987b). Such an approach was adopted by the National Aboriginal Health Strategy (NAHS). However relatively little consideration was given to nutrition issues in the final report (NAHS, 1989:120,124,134-135,138).

1.9.3. Evaluation of Aboriginal nutrition programs

Strategies aimed at improving the general nutritional status of Aboriginal communities have neither been systematically applied nor evaluated (Sykes, 1981; Patterson, 1987).

Claims that Aboriginal mortality and morbidity have been improved since the establishment of a preventative health program by the Queensland government (Musgrave, 1984) have been publicly criticised (Grunseit, 1984; Thomson, 1984). Similarly attribution of improvement in growth rates of Sydney Aboriginal children to "*many factors initiated by the Aboriginal Medical Service*" particularly "*nutrition and the emphasis on preventative medicine*" (Brand *et al*, 1978), could also be criticised. Simple association with changing health indicators is not sufficient proof of the efficacy of specific programs; controls and specific evaluation techniques also need to be applied.

Conversely it could also be argued that the continuing high rates of morbidity associated with poor Aboriginal nutritional status in the Northern Territory did not necessarily indicate that nutrition programs were not effective as has been previously suggested (Rae, 1988). Although health indicators such as growth data for under 5 year old children have been regularly monitored and some aspects of process evaluation have been applied, specific nutrition programs have not been formally evaluated in the Northern Territory.

It is likely that improvements in morbidity data will occur only in the long term. Change in dietary intake and health indicators of nutritional status would appear to be the most appropriate measure in evaluating the success of nutrition programs. Evaluation of intervention projects at an outcome level typically requires a comparative measurement of pre- and post-intervention change. As discernible changes in health indicators would not be expected to be apparent over the relatively short time frame considered in most intervention projects in Aboriginal communities, it would be expected that the outcome of principal interest would be quantitative change in dietary intake.

1.9.4. Ethical considerations of research in Aboriginal communities

There is increasing (and understandable) criticism from both Aboriginal and non-Aboriginal commentators that there has been too much research and not enough action (Waterford, 1982). "*..it is now not acceptable to regard Aborigines as experimental subjects for studies which do not promise reasonably immediate benefits to the Aborigines*

themselves" (Moodie, 1981:154).

However, little past research has directly addressed potential strategies to improve Aboriginal health. It has been argued that practical research *"is still needed to find means of preventing and controlling the high morbidity and mortality among Aborigines, especially in relatively small isolated groups. Controlled trials are needed of various intervention strategies derived not only from the West but also from the Third World. The problems must be practical and the methods scientific"* (MacLennan and Pope, 1982). More attention must be directed towards the development of innovative health education strategies (Thomson, 1984).

Moreover few past studies have directly involved Aboriginal people in the research process: *"There has been something in the order of 3,500 papers written on Aboriginal people. You show me any NT (Aboriginal) Health worker who can tell you what is in five of them"* (Rebgetz, 1985).

In general *"research should be conducted in an honourable manner with mutual trust and respect between the community and their researchers. Research should be applied, relevant to the needs of the people and obligations to ensure each individual is provided with their personal results including any implications and relevant recommendations in a meaningful form should be met, so that the information is available for assimilation and direct action"* (anon). Specific guidelines for research in Aboriginal communities have been developed (NHMRC, 1991).

1.10. Major points

All available evidence indicated that as nomadic hunter-gatherers Aboriginal people were extremely lean and physically fit, consuming a varied diet derived from lean wild animals, fish and vegetable foods, which was low in energy density, being low in fat (particularly in saturated fatty acids) and high in nutrient density, protein, vitamins, minerals, trace-minerals and dietary fibre. The availability of preferred foods, that is those high in fat or sugar, was limited and their collection and distribution was controlled by traditional Law.

In contrast, the poor nutritional health of contemporary Aboriginal Australians is a well documented, serious and costly public health problem.

Contemporary Aboriginal dietary choice is considered to be affected by social, cultural and economic factors, such as lack of association between health and nutrition, limited knowledge of the nutritional value of European foods, lack of storage and cooking facilities and the liking for sweet and fatty foods. Community stores have had a major impact on dietary intake.

Rates of mortality and morbidity for contemporary Aboriginal people remain higher than those for non-Aboriginal Australians. In the past, most public attention had focused on the extremely high Aboriginal infant mortality rate which has improved over the last thirty years but is still unacceptably high. Aboriginal children are generally underweight and growth-retarded when compared with European children of the same age. Poor growth rates have been shown to be associated with poor maternal nutritional status, malnutrition and repeated acute and chronic infections.

The recent reduction in infant mortality rate and communicable diseases in general has been countered by an increase in non-communicable, or 'lifestyle' diseases, consequent on rapid acculturation and associated over-consumption of energy-dense, nutrient-depleted foods. These conditions include non-insulin dependent diabetes (NIDDM) and its vascular complications, coronary heart disease, hypertension and stroke, which are frequently associated with obesity and with android (central) distribution of body fat. Non-communicable diseases are now believed to be major causes of premature death and morbidity in many Aboriginal communities.

Since most nutrition-related health problems impact on the majority of people in remote Aboriginal communities, it is more logical, practical and potentially more effective to address and try to prevent these problems from a community, public health perspective, rather than from an individual perspective. There is a need for standardised protocols to investigate health and nutritional status of contemporary Aboriginal groups on a community basis.

Although the relatively high prevalence of nutrition-related health problems had been frequently described, little attention had been paid to the quantitative measurement of dietary intake in Aboriginal communities. In addition to the well-recognised limitations of most dietary intake measurements in populations, specific methodological difficulties in measuring Aboriginal dietary intake are summarised in Table 1.4.

Table 1.4. Factors which may affect dietary survey methodologies in remote Aboriginal communities

Cultural factors:

- Language
- Non-acceptability of direct questioning
- Limited literacy/numeracy concepts
- Sharing of foods throughout the extended family
- Less structured pattern of dietary intake
- Non-acceptability of noting individual behaviour, (particularly food consumption)
- Non-acceptability of biological sampling
- Differing cultural perception of value of dietary intake data

Social/ Environmental factors:

- Economic cycle affects dietary intake
- Availability of storage and cooking facilities
- Availability and accessibility of traditional bush foods
- Availability and variety of store foods
- Isolation

Ethical factors:

- The need to consider spiritual beliefs affecting concepts of health and food
 - Lack of desire for interaction with non-Aboriginal researchers
(may be exacerbated by ethical problems with past research)
-

Even less attention has been paid to formal projects aimed at improving the nutritional health of Aboriginal people, or to appropriate methods to evaluate such projects. Evaluation of intervention projects at an outcome level typically requires a comparative measurement of pre- and post-intervention change. As discernible changes in health indicators would not be expected to be apparent over the relatively short time frame considered in most intervention projects in Aboriginal communities, it would be expected that the outcome of principal interest would be quantitative change in dietary intake.

Therefore the development of an acceptable, reliable and accurate dietary survey methodology is essential as a base from which to work in dietary intervention programs, having application in both the identification of the contributing components of different foods to the diet, (thus highlighting, and potentially pinpointing, practical intervention strategies) and the evaluation of the dietary impact of those intervention strategies.

CHAPTER 2: PILOT STUDIES OF DIETARY SURVEY METHODS FOR APPLICATION IN REMOTE, CENTRALISED ABORIGINAL COMMUNITIES.

This chapter presents a brief report of pilot studies which trialled five dietary methods in two remote, centralised Aboriginal communities. The methods are appraised according to their practicality, acceptability and face validity. Where quantitative data were produced, congruent validity of the methods is also presented.

2.1. Introduction

In order to test potential dietary survey methods for use in Aboriginal communities, five methods were trialled initially at two remote, centralised communities in 1986. The dietary methods were developed after consideration of the many cultural, social and economic factors that affect the feasibility, acceptability and validity of conventional dietary survey methodologies in remote, centralised Aboriginal communities (as outlined in Chapter 1 and summarised in Table 1.4). While four of the methods were adapted directly from conventional dietary survey methods previously outlined in section 1.7 (weighed dietary intake, 24-hour recall and the food frequency method), the fifth (the 'store-turnover' method), was only loosely based on the food sales method (section 1.7.1). The first four methods attempted to directly measure the dietary intake of groups of individual adult subjects. The store-turnover method was devised to measure the nutrients available for consumption for the community as a whole which, under the unique circumstances of food supply in centralised Aboriginal communities in remote areas, was assumed to closely approximate the dietary intake of the community as a whole.

Within the public health agenda it was necessary to consider the degree of accuracy required for individual dietary studies in Aboriginal communities; it appeared to be more germane to produce and evaluate a method which could place individuals into broad categories of intake rather than to describe small differences in the amount of specific nutrients consumed by different individuals.

2.2. Aim

The broad aim of the pilot studies was to develop an acceptable quantitative dietary survey methodology to be validated subsequently for use in remote Aboriginal communities.

2.3. Study design

In these preliminary studies, methods were assessed primarily according to their face validity. Practical aspects of each method were also considered; that is, acceptability to the community, relative cost and the potential involvement of Aboriginal researchers. In the case of weighed intake and 24-hour recall methods, there was also an attempt at determination of congruent (relative) validity. Originally it was proposed to compare weighed and recalled dietary intakes for specific individuals over the financial cycle in both communities. However, the final data-set was too incomplete to enable meaningful statistical analysis. The study design was therefore modified in order to enable comparison of the initial set of weighed and recalled dietary data pertaining to the same day for different subjects. Due to the lack of a 'gold standard', it was not possible to determine the construct validity of any method.

2.4. The study communities

The data were collected in a northern coastal community over a three week period in May 1986 and in a central desert community over a two week period in October 1986.

Both communities were located on Aboriginal owned land; both preferred to remain anonymous for the purpose of this study. The northern coastal community was located on a small island off the north coast of Arnhem Land. Most foods, including most perishable items, were delivered fortnightly by barge and some perishable food items were occasionally flown into the community on weekdays. Unemployment benefits were paid fortnightly and pension cheques were also received fortnightly but on alternate weeks. The central desert community was located in the north-west region of South Australia. At the time of the study, dry goods and most perishable items were delivered approximately every six weeks by road transport from Adelaide. Some perishable foods, particularly meat, were flown weekly from Alice Springs. This community participated in the Community Development Employment Program (CDEP) and each adult resident received a weekly 'salary'.

In the northern coastal community the council selected both the initial extended family of eleven adult members willing to participate in the weighed and recalled dietary study, and a literate/numerate community member to be trained and employed as a research assistant. The research assistant subsequently enlisted ten volunteers for participation in the testing of the dietary history and modified food frequency methods. In the central desert community, the study was conducted as one part of the nutrition component of an extensive public health review of the region (UPK, 1987). One extended family was selected by the community, and a total of ten adults agreed to participate in the weighed and recalled dietary studies. Assistance was provided by a non-Aboriginal nutrition worker who had previously lived in the area for many years. Ten volunteers also agreed to participate in the testing of the dietary history and modified food frequency methods in the central desert community.

2.5. Dietary methods

2.5.1. Weighed dietary intake

In order to encompass the likely variation in the diet over the income cycle, an attempt was made to weigh and measure dietary intake of foods and beverages consumed by all members of the extended families on alternate days of the observation period which was based on the respective income cycle in each community. In the northern coastal community, an attempt was made to weigh all foods and beverages consumed by all eleven family members on day one, three, five, seven, nine, eleven and thirteen of this fortnightly cycle. It was not possible for ethical reasons (section 2.8.1) to collect data on the ninth day; nor was it possible to collect data beyond day eleven. Day one was a Thursday on which salaries and unemployment benefits were received. In the central desert community, an attempt was made to weigh individual dietary intake on days one, three, five and seven of the weekly income cycle. Day one was a Wednesday. CDEP payments were received on day three (Friday). In both communities not all subjects were willing to participate in the weighed dietary studies on all days and not all were present in the community for the whole period. Due to particular difficulties in collecting weighed dietary intakes from the initially selected eleven subjects in the northern coastal community (section 2.8.1), the Senior Aboriginal Health Worker arranged for the dietary intake of eighteen members of her own extended family to be weighed once on either day seven, nine or eleven of the income cycle.

In both communities food weights were recorded using Soehnle digital scales by either myself or the research assistant after appropriate training. In the northern coastal community the research assistant weighed food and beverage intake for individuals 3,4,5,6,10,12,15,16,17 and 22 and I weighed intake for individuals 1,2,7,8,9,11,13,14, 18,19,20,21,23,24,25 and 26. The division into two groups was necessary for practical reasons and the composition of each group was dictated by traditional Aboriginal Law prescribing the relationship of the Aboriginal research assistant and the subjects she observed. No such restrictions applied in the central desert study where both non-Aboriginal observers weighed dietary intake of specific individuals alternatively. During the period when dietary intake was being measured, the researchers resided with each respective family, leaving the main group when it was necessary to accompany a particular subject.

The weight of each individual's food was recorded immediately before consumption. Wastage was weighed after eating was complete and the appropriate quantity consumed recorded. To reduce the level of intrusion, foods consumed in discrete units, such as pre-sliced bread, were not individually weighed on all occasions, the mean weight of each unit being assigned to those units consumed. All foods recorded were actually observed being eaten. Initially, to determine the weight consumed of items such as beverages which were shared from person to person from one container, weights of the remaining food/beverage were measured after each individual's 'turn'. Later an approximation was made by counting the number of 'swallows' for each individual and the mean volume of ten measured swallows for each person apportioned accordingly.

2.5.2. 24-hour dietary recall

In both communities an attempt was made to collect twenty-four hour dietary recall data from all subjects corresponding to the days for which dietary intake had been weighed. In the northern coastal community an attempt was made to collect twenty-four hour dietary recalls from the initial eleven subjects on days two, four, six, eight and day eleven of the income cycle (corresponding to days one, three, five, seven and ten respectively). In the central desert community an attempt was made to collect twenty-four hour recall data on day two, four, six and eight (corresponding to day one, three, five and seven respectively). In both communities not all subjects were willing to participate in the recalled dietary studies on all days and not all were present in the community for the whole period. To increase the sample size for the comparison with weighed data, recalled

dietary intake data was also collected for the additional fifteen volunteers who participated in the weighed recorded intake study in the Northern coastal community.

Each subject was interviewed at their home during the morning and asked to recall all foods and beverages consumed on the previous day. To assist in determination of quantities of foods recalled, actual food samples were produced to help people communicate their perception of amounts consumed; for example a large prepared damper was displayed and people indicated the approximate proportion which they had consumed. For foods consumed in discrete units the number of those units recalled was recorded and appropriate weights apportioned; for example, one tin of corned beef weighed 340 g nett. All twenty-four hour dietary recalls were collected by myself. Both English and Pitjantjatjara were used to obtain twenty-four hour diet recalls in central Australia but English only was used in the northern coastal community.

2.5.3. Food frequency method

A 'questionnaire' incorporating a list of commonly available foods stocked in the respective community stores was devised; foods available in regional urban centres were also included (Addendum 1, A1.1). Volunteers from each community were questioned jointly by the research assistant and myself in both English and in the local language, and asked how frequently each food was consumed within the pay period (that is, fortnightly in the northern coastal community and weekly in the central desert community). Optional answers were prompted in an attempt to assist response; "*everyday*", "*nearly every day*", "*only on 'pay day'*". Interviews were conducted in the afternoons on the days when twenty-four hour dietary recalls had been solicited from the subjects participating in the weighed dietary studies.

There was no attempt to assess the quantity of foods consumed at each occasion; if the method produced meaningful results it was hoped that an 'average' serve may have been able to be proportioned for each type of food using the results of further weighed studies.

2.5.4. The dietary history method

An attempt was made to directly apply the classical dietary history method, with the exception of the component of three day weighed intake (section 1.7.1.3), in assessing the 'usual' dietary intake of ten volunteers in each community. Subjects were questioned at

their homes jointly by the research assistant and myself in both English and in local language. Interviews were conducted in the afternoons on the days when twenty-four hour dietary recalls had been solicited from the subjects participating in the weighed dietary studies.

2.5.5. The store-turnover method

Store data was collected meticulously on location in a standardised form over a period of twelve weeks. All food items delivered to each store during the preceding three month period were listed from invoices, total quantities supplied tabulated and average daily supply calculated. No allowances for wastage was included. For the extended period considered, the mean daily supply of food was assumed to approximate the mean daily turnover (purchases) which was assumed to approximate mean daily consumption of the community. Apparent per capita consumption of food and nutrients per day was determined at each community by dividing the mean daily store-turnover by the population of each community. The population at both communities was determined by population census at the time of data collection. It was not possible to complete a store stock-take either before or after this period. The store-turnover method was subsequently applied in an additional four communities; further details of the method were included in the relevant section (section 3.4).

2.6. Methods of data analysis

2.6.1. Qualitative validation

The results obtained by each method were appraised subjectively in terms of their ability to produce quantitative data and whether they appeared to measure what they purported to measure, that is, face validity. Possible sources of error for each method were noted and graded qualitatively in terms of the probability of potential bias. Comparative criteria of the applied methods were graded arbitrarily according to observed positive and negative characteristics. The acceptability of each method to the community was assessed subjectively.

2.6.2. Quantitative validation and statistical methods

Quantitative data obtained were analysed for nutrients using "Microdiet" software (which was based on Paul and Southgate (1978)) amended with Australian food composition data (Australia, 1989) and results of traditional bush food analysis (section 1.2.2.3). Individual nutrient data were expressed as a percentage of the appropriate Recommended Dietary Intake (Truswell *et al*, 1990) to enable comparison between different ages and sex.

The twenty-four hour dietary recall method was validated congruently against the weighed dietary intake method, determined on the same day for thirty-six different individuals, in terms of the agreement between the two methods, as measured by the kappa statistic, for classification of individual nutrient intakes into three broad arbitrary categories based on Recommended Dietary Intake (RDI); high (> 133% RDI), medium (66% to 133% RDI) or low (less than 66% RDI).¹

An attempt to investigate statistically the pattern of concordance between the methods with respect to food type, individual and day of observation, and the effect of repeat measures over the income cycle in both communities, was discontinued due to the extreme bias of the incomplete data set obtained.

Food data was analysed by application of Genstat software. For the purposes of initial analysis, two binary variables were constructed to identify the presence (1) or absence (0) of a particular food in a weighed record and in a recalled record. A new binary variable was calculated identifying concordance (1) and non-concordance (0) of weighed and recalled records with respect to the presence or absence of particular foods. Subsequently, to analyse the pattern of non-concordance (a composite representing both additions to and deletions from the recalled dietary intake) another binary variable was created to identify additions to (1) and deletions from (0) dietary recall. Logistic regression (SPSSX-3) was used to determine if there was a net tendency to over or under estimate food intake with respect to the type of food.

¹ Determination of correlation coefficients, although frequently applied in congruent validation of dietary survey methods, has been shown to be an inadequate statistical approach to the comparison of these methods (section 1.7.3).

2.7. Results

2.7.1. Qualitative results

As it was necessary to rely on volunteers for ethical reasons, the initial sample of subjects agreeing to participate in individual dietary studies was potentially biased relative to the wider communities. Furthermore, there was a very high drop-out rate for all methods which aimed to measure individual dietary intake (Table 2.1). Once they had experienced the process of one day of weighed dietary intake, most initial volunteers withdrew from the longitudinal study, which, therefore, could not be completed. All the subjects who refused to participate in even one day of weighed and recalled dietary studies were men over the age of 35 years. All the additional subjects who agreed to participate in the longitudinal weighed/recalled dietary studies were women over the age of 30 years; five of the 15 (33%) had previously been diagnosed as obese and/or diabetic and had received prior dietary counselling. The final sample in which dietary intake was both weighed and recalled for the same 24-hour period was comprised of 25 women (74% of the total sample) and was highly biased relative to the wider community. The number of initial volunteers who actually agreed to participate in dietary history and food frequency studies was also low.

Table 2.1. Sample

Method	Initial n	Refused n (%)	Addition n	Refused n (%)	Final n (%)
Weighed/recalled pairs (extended)	183	135 (74%)	-	-	48 (26%)
Weighed/recalled pairs (one day only)	21	2 (9.5%)	18	3 (16.7%)	36 (92%)
Diet history	20	9 (45%)	-	-	11 (55%)
Food frequency	20	11 (55%)	-	-	9 (45%)

The potential sources of bias experienced in the application of each method are summarised in Table 2.2. Specific characteristics of each method applied are summarised in Table 2.3.

Table 2.2. Potential bias in applied dietary methods

Source	Weighed Records	24-hour Recall	Diet history	Food Frequency	Store-Turnover
Sampling bias	++++	+++	++++	++++	+
Response bias	n/a	+++	+++	+++	n/a
Reporting errors					
-wrong food	+	++	++++	n/a	+
-wrong weight	+	+++	++++	n/a	+
-wrong frequency	n/a	n/a	++++	++++	n/a
Coding errors	+	+	+	+	+
Food tables	+	+	+	+	+
Recorded intake not <i>usual</i> intake	+++	++++	+++	+++	+

+ possible ++ probable +++ very probable ++++ Highly probable n/a = not applicable

Table 2.3. Comparative criteria of the applied methods

a: Negative characteristics

	Weighed Records	24-hour Recall	Diet history	Food Frequency	Store-Turnover
Invasiveness	++++	++	+++	++	+
Time required	++++	++	+++	+++	+
Number of personnel required	++++	+++	+++	+++	+
Resources required	++++	+++	++	++	+
Travel costs	++++	+++	+++	+++	+
Lack of individual data	+	+	+	+	++++

b: Positive characteristics

	Weighed Records	24-hour Recall	Diet history	Food Frequency	Store-Turnover
Acceptability to community	+	++	+	+	+++
Discerns individual's usual diet	+	+	++	++	n/a
Can provide retrospective data	n/a	+	++	+	++++
Potential for Aboriginal researchers	+	++	+	+	+++
Potential reproducibility	+	+	+	+	++++
Cost effectiveness	+	++	++	++	++++

+ very low ++ low +++ moderate ++++ high n/a = not applicable

2.7.2. Quantitative results

It was possible to derive quantitative data only from application of the weighed dietary intake, twenty-four hour recall and store-turnover dietary methods.

2.7.2.1. Weighed dietary records and twenty-four hour recall

Due to poor compliance in the study of the weighed and recalled methods, it was not possible to investigate the nature of dietary change over the financial cycle as originally intended.

The mean group nutrient intake derived from weighed and twenty-four hour recall data for the first day of data collection for each different subject is presented in Table 2.4. Relatively low intakes (less than 66% RDI) are highlighted in bold.

Table 2.4. Mean per capita nutrient intake derived from weighed and recalled dietary intakes (% RDI)

Nutrient	Central desert (n=10)		Northern coast (n=26)	
	Weighed mean \pm sd	Recalled mean \pm sd	Weighed mean \pm sd	Recalled mean \pm sd
Energy	80.7 \pm 30.7	85.4 \pm 24.2	89.3 \pm 26.5	123.3 \pm 50.6
Protein	159.8 \pm 115.0	190.3 \pm 70.3	97.5 \pm 44.2	206.2 \pm 103.0
Calcium	33.1 \pm 25.5	29.7 \pm 18.3	40.3 \pm 25.8	68.0 \pm 47.2
Iron	154.7 \pm 189.2	142.9 \pm 94.1	96.7 \pm 70.6	181.7 \pm 110.6
Zinc	79.9 \pm 72.8	111.8 \pm 64.0	58.3 \pm 30.0	147.8 \pm 72.7
Retinol equivalents	54.6 \pm 50.6	37.3 \pm 18.7	40.0 \pm 29.6	105.8 \pm 163.5
Thiamine	68.6 \pm 43.1	88.9 \pm 33.1	78.9 \pm 43.3	148.3 \pm 92.3
Riboflavin	63.7 \pm 46.4	67.3 \pm 34.1	95.4 \pm 51.6	179.0 \pm 123.2
Niacin	63.9 \pm 50.4	87.0 \pm 44.9	78.5 \pm 36.0	174.0 \pm 59.1
Vitamin C	242.0 \pm 250.0	553.0 \pm 256.0	78.2 \pm 137.4	289.3 \pm 341.5
Vitamin B ₁₂	110.1 \pm 134.0	142.7 \pm 66.8	185.8 \pm 115.2	286.3 \pm 202.2
Folate	65.4 \pm 38.4	75.9 \pm 37.9	51.6 \pm 25.9	105.8 \pm 78.7
% Energy derived from:				
Protein	19.6 \pm 9.4	23.4 \pm 3.2	12.6 \pm 3.0	18.6 \pm 4.4
Total carbohydrate	50.4 \pm 14.7	33.4 \pm 6.2	58.2 \pm 10.8	39.1 \pm 12.1
Fat	30.0 \pm 11.4	43.4 \pm 5.7	29.4 \pm 10.5	42.3 \pm 14.0
Sugars	23.1 \pm 7.6	15.7 \pm 4.3	27.1 \pm 9.6	14.9 \pm 8.8
Complex carbohydrate	27.8 \pm 16.7	17.7 \pm 3.3	31.1 \pm 12.7	24.2 \pm 12.2

The 24-hour recall method was not able to accurately place individuals into broad categories along the distribution of intake as weighed during the previous day (Table 2.5). When used to classify an individual's diet into three categories, high (> 133% RDI), medium (66% to 133% RDI) and low (< 66% RDI), the classifications disagreed in a mean of 33% of the instances, with approximately 7% grossly misclassified in opposite thirds. The poor agreement between the two methods was also illustrated by the fact that only 54% of all recalled nutrient values were within 40% of the weighed value for the

same individual on the same day. In general, compared to the weighed record, protein, fat and most vitamins and minerals tended to be over-recalled and sugars tended to be under-recalled.

Table 2.5. Agreement between the two methods for classification of individual nutrient intakes into three categories (high: > 133% RDI, medium: 66% to 133% RDI and low: < 66% RDI) (n=36)

Nutrient	Total misclassified		Grossly misclassified (opposite thirds)		Kappa statistic (total agreement = 1)
	n	%	n	%	
Energy	12	33	-	-	0.56
Protein	15	42	2	6	0.30
Calcium	7	19	1	3	0.46
Iron	17	47	1	3	0.28
Zinc	16	44	2	6	0.29
Retinol equivalents	8	22	3	8	0.37
Thiamine	18	50	3	8	0.24
Riboflavin	16	44	2	6	0.34
Niacin	14	38	3	8	0.40
Vitamin C	13	36	8	22	0.39
Vitamin B ₁₂	9	25	3	8	0.54
Folate	12	33	1	6	0.49

The pattern of recall of foods was very revealing. Analysis of foods indicated that, where weighed, foods frequently omitted from recall included: sugar; sweetened carbonated beverages; potato crisps; cake; ice cream; jam; rice. Foods not included in the weighed record yet frequently added to recall included: fruit juice; fruit; dried milk; vegetables; wholemeal bread; beef; kangaroo; muesli bars (Table 2.6).

In both regions, where foods were both weighed and recalled, those consistently underestimated in quantity compared to the weighed record were sugar and sweetened carbonated beverages and those consistently overestimated were fruit, vegetables, fruit juice and beef.

Table 2.6. Presence or absence (concordance) of foods in recalled and weighed records (n= number of cases in which a food was either weighed or recalled)

	Total Concordance		Non-Concordance	
	(n)	(%)	Deleted (%)	Added (%)
All foods	696	78.3	9.8	11.8
Bread	68	82.1	5.1	12.8
Margarine	38	100	0	0
Jam	29	89.9	11.1	0
Fruit Juice	35	66.6	0	33.3
Sugar	70	85.7	14.3	0
Beef	56	87.5	0	12.5
Kangaroo	2	0	0	100
Goanna	6	100	0	0
Potato Crisps	10	20.0	100	0
Chicken	31	100	0	0
Vegetables	19	55.6	0	47.4
Fruit	45	46.6	0	53.3
Tinned Meat	28	89.9	5.5	5.5
Dried Milk	39	66.6	0	33.3
White Flour	51	92.0	0	8.0
Cake	4	50.0	50.0	0
Ice cream	13	0	100	0
Muesli Bars	14	75.0	0	25.0
Weetbix	18	100	0	0
Rice	13	84.6	15.4	0
Sweet beverages	54	76.9	23.1	0
Tea	53	100	0	0

2.7.2.2. Store-turnover

The store-turnover method was able to produce quantitative results for both communities participating in the pilot studies. The apparent consumption of selected foods (Table 2.7) and the apparent per capita consumption of nutrients (Table 2.8) determined by this method are presented in tabular form.

Although results indicated that the apparent per capita intake of fruits and vegetables, meat products and the combined flour and bread intake were similar at both communities there were some differences. The apparent consumption of fresh store meat was higher at the central desert community, while the apparent intake of carbonated beverages and sugar *per se* was higher at the northern coastal community.

Table 2.7. Apparent consumption of selected foodstuffs measured by the store-turnover method (per capita per day)

Food	Central Desert Community	Northern Coastal Community
Flour, white (g)	115	158
Bread, wholemeal (g)	12	15
Bread, white (g)	73	55
Beef (g)	198	65
Poultry (g)	56	25
Fruits (g)	49	55
Vegetables (g)	40	48
Sugar (g)	112	169
Carbonated beverages (ml)	148	400
Fruit juice (ml)	136	37
Tinned meat (g)	40	34
Pie/Pastie (g)	22	26
Snack foods** (g)	7	2

** includes potato crisps, extruded snacks

Table 2.8. Apparent nutrient intake measured by the store-turnover method (per capita per day)

Nutrient	Central Desert Community	Northern Coastal Community
Energy (kJ)	12310	13105
Protein (g)	96.7	73.3
Fat (g)	131.6	113.5
Total Carbohydrate (g)	366.0	486.7
Complex carbohydrate (g)	179.7	210.2
Sugars (g)	186.3	276.5
% Energy derived from fat	40.0	32.3
% Energy derived from carbohydrate	46.8	58.2
% Energy derived from complex carbohydrate	23.0	25.2
% Energy derived from sugars	23.8	33.1
% Energy derived from protein	13.2	9.4
Calcium (mg)	719.7	672.7
Iron (mg)	16.9	10.1
Zinc (mg)	13.6	9.3
Vitamin A equivalents (μg)	296.3	516.8
Retinol (μg)	237.5	354.4
Carotene (μg)	353.0	974.4
Folate (μg)	129.9	123.7
Thiamine (mg)	1.15	1.08
Riboflavin (mg)	1.57	1.14
Niacin (mg)	18.9	13.9
Vitamin C (mg)	95.0	41.1
Vitamin E (mg)	3.7	4.1
Vitamin B ₆ (mg)	1.26	0.97
Vitamin B ₁₂ (μg)	4.70	2.95
Sodium (mg)	3723	2788
Cholesterol (mg)	301.3	266.5
Dietary fibre (g)	12.6	12.9

2.8. Discussion

In the pilot studies in these two remote Aboriginal communities, dietary methods applied in the direct measurement of individual dietary intake did not appear to be satisfactory. Major problems included poor acceptance by individuals and the community as a whole.

2.8.1. Weighed record

The recording of the weight of all foodstuffs consumed by individuals over an extended period proved to be particularly invasive and was not accepted by the majority of subjects in either community. Problems with cooperation were experienced, particularly in the northern coastal community, where poor compliance may have arisen due to unusual financial problems within the initial family studied, (the main provider's pay cheque failed to arrive during the study period), and to a lesser extent, as a result of concurrent traditional ceremonies. It proved impossible to continue to weigh food and beverage intake for all family members every second day as planned. To persevere would have been extremely insensitive and damaging to the good relationship established between the community and myself. Individuals in the central desert community, where I had previously resided for an extended period and consequently enjoyed closer personal relationships, were generally more tolerant.

In the community setting, it was also extremely difficult for only two observers to ensure that all foods consumed by the individuals in each family group studied were actually observed and weighed; it would be necessary to assign one observer for each subject to ensure that no foods consumed were omitted (and hence require a substantial increase in the study budget). Therefore the weighed recorded method, as reported here, would tend to provide an under-estimated representation of actual individual dietary intake of some foods to an unknown degree and could not be deemed to provide an accurate or complete record of all foods consumed for each subject. The poor response and compliance rates also contributed to bias in the weighed intake studies.

However, the results obtained from the weighed studies did generally support past qualitative studies which suggested that diet in Aboriginal communities was high in sugars, moderately high in fat, low in complex carbohydrate, fibre and nutrient density (section 1.8.2). Low dietary intakes of calcium and vitamin A were also measured in the weighed six day dietary study of two fringe dwelling families in Bourke (Kamien *et al*, 1975).

However intake of nutrients weighed in the present study was generally higher, particularly with respect to intake of energy, protein and ascorbic acid (section 1.8.2).

2.8.2. 24-hour dietary recall and comparison with weighed recorded intake

It could be assumed that the unusual focus on the previous day's dietary intake during weighing, would promote a greater accuracy of recall of those foods consumed. The use of food samples produced once a food had been mentioned, to assist in the estimation of portion size, would also be expected to improve accuracy of recall. The average time taken to obtain a twenty-four hour recall was 46 minutes which would exceed the usual time available for this purpose in a large scale dietary survey. Hence it should be emphasised that twenty-four hour recall data used in this study were obtained under the very best possible conditions.

From the cultural perspective of the subjects it was hard to rationalise the study of dietary intake in general; it was particularly difficult to justify collection of twenty-four hour recalls after a day of measuring intake. This aspect of the study required careful explanation and may have contributed to bias in recall.

The limited variety of foods either weighed or recalled was noteworthy. Only twelve and nineteen different types of food were either weighed or recalled in the northern coastal and central desert communities respectively. Over 65% of the daily total energy intake was derived from fresh meats, sugar and bread and flour. The variety of food intake in the north may have been affected by the financial problems experienced by the family. Only three traditional foods ("*Tinka*"- sand goanna, "*Kampurarpa*"- desert raisin and "*Malu*"- kangaroo) were either weighed or recalled by the central desert family. No bush foods were either weighed or recalled in the northern coastal community during the study period.

The technique of direct questioning was not commonly practised in traditional Aboriginal culture and responses may be structured to please the interviewer (section 1.8.1). Relative to weighed dietary intake, the 24-hour recall method tended to overestimate the intake of protein and most vitamins and minerals, and underestimate intake of refined carbohydrate.

Conservative nutrition education programs had been operating in both communities participating in this study (section 1.9.1). It could therefore be postulated that the

consistent under-reporting of foods high in refined carbohydrate (sugar, cool drinks, ice cream and cake), and consistent over-reporting of foods high in protein (meats and milk powder) and ascorbic acid (fruit, fruit juice and vegetables), may have reflected a conscious editing of recall designed to constitute the 'right' answer in Aboriginal terms. Further, the results of dietary recall suggested that a level of non-Aboriginal nutrition knowledge was shared by Aboriginal subjects previously exposed to those health and nutrition concepts.

The increased accuracy of recall compared with weighed intake in the central desert family may have been improved by the closer relationship experienced between myself and the family concerned and the fact that communication used local language. However, weighed intake of sugar was less, and weighed intake of protein and ascorbic acid were higher, in this family than in the northern coastal community. This may have reflected not only a conscious editing of recalled response, but also a conscious alteration of actual foods consumed by the central desert family during the study period.

Energy intake appeared to be the most accurately recalled dietary component. However, this was only because energy intake represented a composite value derived from the analysis of both foods which were consistently under-estimated (sugar and 'cool drinks') and foods which were consistently over-estimated (fresh meat).

Most investigators, when comparing the 24-hour recall and weighed record dietary methods in non-Aboriginal groups, have observed that recall under-estimated the nutrients determined from weighed intake. However, some studies have found that recall gave considerably higher mean figures of the intakes of some nutrients than the weighed food record, particularly in cases of low weighed nutrient intake in low socio-economic groups, such as recorded in Aboriginal subjects (section 1.7.3.2.).

The results of the 24-hour dietary recall method applied in these two communities were similar to those reported for a rural community in NSW (Sibthorpe, 1989), and supported the notion of potential bias in that study (section 1.8.2).

As the bias in the recall of dietary intake may be due to a tendency to 'please' the interviewer, the recall method could be particularly misleading in the evaluation of nutrition intervention programs.

2.8.3. Diet history method

In general it appeared to be difficult for subjects to recall the foods and beverages that they 'usually' consumed; the diet history method tended to produce either a qualitative list of foods which had been eaten *at all* during the life of an individual subject, or a qualitative list of foods which the subjects stated that they had eaten the previous day (that is, 24-hour recall). It was not possible to determine numeric quantities by questioning. The use of the foods and food models applied in the 24-hour recall method did assist in subjects being able to indicate their meaning of responses such as "*little bit*" and "*big mobs*". However the quantitative assessments remained essentially subjective.

Although subjects initially stated that they were willing to participate in diet history interviews, in both communities it became obvious that several subjects were actually disinclined to participate. When potential subjects were eventually approached for interview, a frequently repeated response was "*maybe tomorrow*", a polite way of declining in Aboriginal society.

2.8.4. Food frequency method

For each individual the food frequency method took over one hour to apply, as it was necessary to spend a considerable time explaining the rationale of the method. Subjects appeared to have great difficulty in assigning categories of frequency of consumption to specific foods.

The food frequency method appeared to produce a qualitative list of foods which people *liked* to eat, rather than those which they did actually eat. In fact people even stated that they frequently consumed rarely accessible foods which were only available at regional urban centres (such as fried fish and chips, pizzas and cherries). Most subjects also emphasised that they frequently consumed traditional bush foods. This claim was not supported by observation and may have also reflected a preference for these foods rather than actual consumption.

In general this method appeared to cause more confusion than the dietary history method. The discomfort of subjects was illustrated by the fact that the Aboriginal research assistant employed in the northern coastal community explained that six volunteers approached for interview had actually been advised not to participate by previous subjects.

Subsequently, a booklet illustrating commonly available community foods was also produced by an Aboriginal Health Worker from Darwin, who was employed as a research assistant in Nutrition and Public Health at the Menzies School of Health Research (Addendum 1, A1.2). The booklet was left with six volunteers after demonstration/instruction by the Aboriginal Health Worker who asked people to mark a box adjacent to the appropriate illustration whenever the food was consumed in the coming fortnight. Although the booklets were apparently esteemed and carefully stored, when they were collected after a fortnight not one mark had been made in any of them.

2.8.5. Store-turnover method

The results of the store-turnover method were similar to previous quantitative estimations (section 1.8.11). Compared with the results of weighed dietary records for specific individuals expressed in terms of the percentage of energy derived from macronutrients, results of the store-turnover data were lower in protein and fat, higher in complex carbohydrate but similar in sugars. Total individual energy intake as measured by weighed dietary intake was generally lower than the mean apparent energy intake as measured by the store-turnover method. However, the weighed recorded intake method was unlikely to have produced an unbiased estimate of the dietary intake of the community as a whole (section 2.8.1).

Further comments on the results of the store-turnover method were reported in Chapter 3, where the method was tested in an additional four communities and the quantitative results were presented collectively (section 3.5).

Limitations of the store-turnover method included the lack of consideration of wastage, bulk storage and very slow stock and dietary intake from sources other than the store. Difficulties with determination of the population denominator and the application of appropriate nutrient composition values for foods as available in community stores were also problematic. Food distribution patterns within the community were not addressed by application of the store-turnover method. However, since most nutrition-related health problems impact on the majority of people in remote Aboriginal communities (section 1.6) it was considered more logical, practical and potentially more useful to try to determine the dietary intake of the community as a whole.

Major advantages of the store-turnover method were that it related to the diet of the whole

population, was specific to relatively small geographical areas, was relatively non-invasive, rapid, easy and inexpensive. It also did not rely on subjective assessment of diet or memory and avoided language, literacy, numeracy and cultural factors which may be a problem with direct measurement or recall of diet in Aboriginal communities. Potentially, retrospective data could also be collected. There was also potential for the method to be applied by Aboriginal researchers and Health Workers.

2.9. Conclusion

As a result of the pilot studies, the store-turnover method appeared to have most potential for application in remote, centralised Aboriginal communities and was further investigated as described in Chapter 3.

CHAPTER 3: APPARENT CONSUMPTION OF FOOD AND NUTRIENTS IN REMOTE, CENTRALISED ABORIGINAL COMMUNITIES AS DETERMINED BY THE STORE-TURNOVER METHOD.

In this chapter apparent per capita food and nutrient intake in six remote, centralised Aboriginal communities is described using extended, systematic collection and analysis of store invoice data. The face validity and the advantages and limitations of this method are considered in detail. Possible approaches to address limitations are outlined. Implications for community-based nutrition intervention programs are also highlighted.

3.1. Introduction

Many nutrition-related health problems occur frequently in remote, centralised Aboriginal communities and affect most people, either directly or indirectly (section 1.6). Thus from a public health perspective, it is both more logical and more practical to measure dietary intake on a community rather than an individual basis.

Most Aboriginal people in northern and central Australia now live in centralised settlements in remote areas (section 1.4). These settlements usually have a community store which is the only source of purchased food for several hundred kilometres. As traditional foods have generally been depleted by population pressure around these settlements, and people may be disinclined to hunt and gather bush foods in the face of acceptable, readily available market substitutes (section 1.5.3.2), the store has potential as a useful source of information about contemporary food intake (section 1.8.4.1). Under these unique circumstances the turnover of foodstuffs from community stores may indicate the dietary intake of the population as a whole.

Pilot studies (Chapter 2) suggested that of five dietary survey methodologies, the store-turnover method had most potential for application in remote, centralised communities.

3.2. Aim

The broad aim was to develop and apply an acceptable quantitative dietary survey methodology based on the systematic collection and analysis of store invoice data in

remote, centralised Aboriginal communities.

Specific aims were to:

- a) design an acceptable method to measure apparent dietary intake (pending formal validation)
- b) appraise some aspects of the face validity of the method
- c) consider the advantages and limitations of the method

3.3. Study design

The store-turnover method was applied in the measurement of apparent intake of food and nutrients in six remote Aboriginal communities. Some aspects of the face validity of the store-turnover method were determined by comparison of results on a regional basis and comparison of results with dietary intakes reported for the wider Australian community.

3.4. Methods

3.4.1. The Study Communities

Three central desert (C1, C2, C3) and three northern coastal (N1, N2, N3) remote, centralised communities were involved (Table 3.1). N1, N2, and N3 were coastal communities located on small islands close to the Australian mainland. C1, C2 and C3 were located to the south-west of Alice Springs. Communities N1 and C2 also participated in pilot studies of individual dietary intake methods as reported in Chapter 2. All participating communities wished to remain anonymous.

Table 3.1. Characteristics of the six communities

Community	Population	Food Supply			
		Non-perishable foods		Perishable foods	
		Service Centre	Transport	Service Centre	Transport
N1	300	Darwin	barge **	Darwin	air *
N2	350	Darwin	barge **	Darwin	air *
N3	280	Darwin	barge **	Darwin	air *
C1	360	Alice Springs	road **	Alice Springs	air *
C2	140	Adelaide	road ***	Alice Springs	air **
C3	180	Perth	road **	Alice Springs	air *

* every week ** every fortnight *** every six weeks

Communities C1, C2, C3 and N1 were 'dry' communities where alcohol was not officially allowed. Communities N2 and N3 had limited access to alcohol, through the operation of a 'social club', which sold a maximum of six cans of beer per person per day restricted by regulation under the NT Liquor Act. Kava was used to a limited extent in community N1, but not in any other community.

I determined the population at each community by conducting a household census counting all individuals either residing at, or temporarily absent from, the community at the time of data collection.

3.4.2. Store data collection

Store data was collected meticulously in a standardised form at the six communities. All food items delivered to each store during the preceding three month period were listed from invoices, total quantities tabulated and average daily supply calculated (Addendum 1, A1.3).

Due to the limited refrigeration and storage available in all the community stores, the fact that many lines were depleted prior to the arrival of the next food delivery, and the very regular ordering patterns identified, mean daily supply was assumed to approximate mean daily store-turnover (purchases) for the extended time period covered. The mean daily store-turnover was assumed to approximate the mean daily dietary intake of the community. Apparent per capita consumption of food and nutrients per day was determined at each community by dividing the mean daily store-turnover by the population of each community at the time of data collection.

In this preliminary study, estimations of traditional bush food consumption and alcohol were not included; neither was there an attempt to apply a correction factor for food wastage.

Data was collected manually on location during the following months for the preceding 12 week period: N1, May 1986; N2, September 1986; N3, January 1987; C1, October 1986; C2, October 1986; C3, October 1986.

3.4.3. Nutrient analysis

Food data was manually entered into a Microdiet nutrient-analysis software computer package installed on an Olivetti personal computer. Data was analysed by application of the software package modified by incorporation of Australian food composition data (Australia, 1989) into the data base. Calculations adjusting for the measured proportion of fat weighed in specific meat cuts available in the communities were also entered into the data base. The proportion of visible fat in representative samples of meat cuts was measured each time store-turnover was monitored and ranged from 28% to 52% (mean 40%) of the total weight of meat, depending on the cut and the wholesale source of supply.

3.4.4. Estimation of nutritional adequacy

In this preliminary study, recommended dietary intakes for the communities as a whole were determined from recommended dietary intakes for each age and sex category in Australia (Truswell *et al*, 1990) applied across demographic data for Aboriginal groups in the Northern Territory as a whole (ABS, 1986b). The apparent mean per capita intake of nutrients as determined in each community by the store-turnover method was directly compared with the community adjusted recommended nutrient intake.

3.4.5. Comparison with dietary data for wider Australia¹

Two sources of dietary data of the Australian population were used for comparison with the results of the store-turnover method; apparent consumption data (ABS, 1987a) and the national dietary survey of adults in 1983 (CDCSH, 1986, 1987a).

3.4.6. Statistical methods

Nutrient data was electronically transferred to the IBM RT system of the Menzies School of Health Research, Darwin. In this preliminary study the only statistical test applied was the use of student t-tests to compare the means of dietary intake in the two different regions using SPSSX-4.

¹ Due to the nature of the methodology applied, the dietary contribution of Aboriginal groups can be assumed to be negligible in data pertaining to 'wider' Australia.

3.5. Results

3.5.1. Foods

The apparent per capita² intake of selected store foods in three central desert communities (C1, C2, C3) and three northern coastal communities (N1, N2, N3) is represented in Table 3.2. There were similarities between central desert and northern coastal communities in the intakes of sugar, vegetables, and intakes of flour and bread combined, but desert communities appeared to consume greater quantities of store purchased meat than the northern communities. Fruit and fruit juice consumption was higher in the desert communities, but carbonated beverages appeared to be more popular in the northern communities.

Table 3.2. Apparent consumption of selected foodstuffs in remote Aboriginal communities (per capita per day)

Food	C1	C2	C3	N1	N2	N3
Flour, white (g)	125	115	69	158	116	91
Bread, wholemeal (g)	0	12	13	15	7	20
Bread, white (g)	75	73	107	55	70	84
Beef (g)	91	198	135	65	91	56
Poultry (g)	61	56	66	25	76	61
Lamb (g)	129	0	58	27	0	0
Fish (g)	0	0	0	0	20	20
Fruits (g)	78	49	146	55	43	47
Vegetables (g)	79	40	81	48	73	40
Sugar (g)	192	112	141	169	90	153
Carbonated beverages (ml)	326	148	84	400	370	407
Fruit juice (ml)	174	136	87	37	20	43
Tinned meat (g)	10	40	27	34	40	9
Pie/Pastie (g)	17	22	40	26	55	43
Snack foods** (g)	3	7	5	2	4	16

** includes potato crisps, extruded snacks

The apparent per capita consumption of selected foods in the Aboriginal communities and national figures for Australia derived from two sources are presented in Table 3.3. Generally there were major differences between the two population groups. Intakes of sugar, white flour and sweetened soft drinks were much higher in all Aboriginal communities and intakes of wholemeal bread, fruit and vegetables were much lower than in the wider Australian community.

² per man, woman, child

Table 3.3. Apparent consumption of selected foods in remote Aboriginal communities compared with the wider Australian community (kg per capita per year)

Food	Central Australian Communities	Northern Australian Communities	All Aboriginal Communities	Australia (ABS)	Australia (NDS) [‡]
Flour (white)	37.6	44.4	41.2	n/a	1.3
Bread (all)	34.1	30.5	32.3	45.5	33.6
Beef	51.6	25.8	38.7	41.4 ^{***}	21.0 ^{**}
Poultry	22.3	19.7	21.0	23.0	9.1
Lamb	22.8	3.3	13.1	16.9	9.9
Fish	0	4.8	2.4	4.0	3.6
Fruits	33.2	17.6	25.4	106.9	65.7
Vegetables	24.3	19.6	22.0	136.2	98.2
Sugar	54.1	50.3	52.2	8.2	6.2 ^{***}
Carbonated beverages	67.9	224.6	146	73.0	19.9
Fruit juice	48.3	12.8	30.6	n/a	20.6
Tinned meat	9.4	10.1	9.3	n/a	n/a
Pie/Pastie	9.6	15.1	12.4	n/a	9.7 [#]
Snack foods ^{**}	1.8	2.7	2.3	n/a	0.5

[‡] not strictly 'apparent consumption' data

^{*} includes flour used in bread making

^{**} includes beef and veal

[#] includes all non-fish take-away foods

^{***} includes sugar products (such as sugar based flavourings and syrups) ^{**} includes potato crisps, extruded snacks

Australia (ABS): Apparent Consumption of Foodstuffs and Nutrients (ABS, 1987a)

Australia (NDS): National Dietary Survey of Adults: 1983 (CDCSH, 1986)

3.5.2. Nutrients

The style of diet was reflected by the contribution of individual macronutrients to the total energy intake and varied from community to community (Figure 3.1). The contribution of individual macronutrients to the total energy intake in the desert communities, the northern coastal communities, and all the communities combined, was compared with recommended levels (CDCSH, 1987b) and dietary data for the wider Australian community (Figure 3.2).

The total energy intake in all communities was similar (Table 3.4).

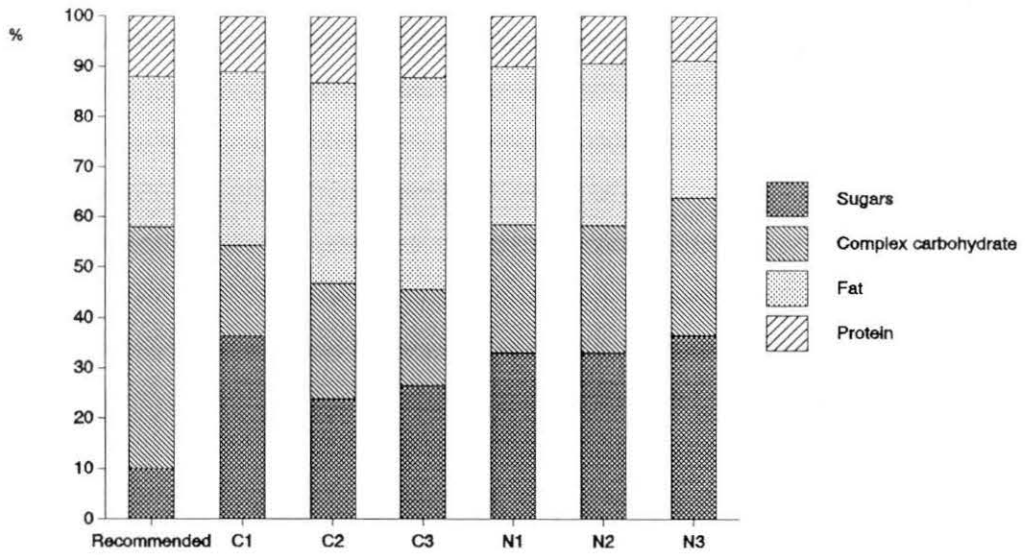
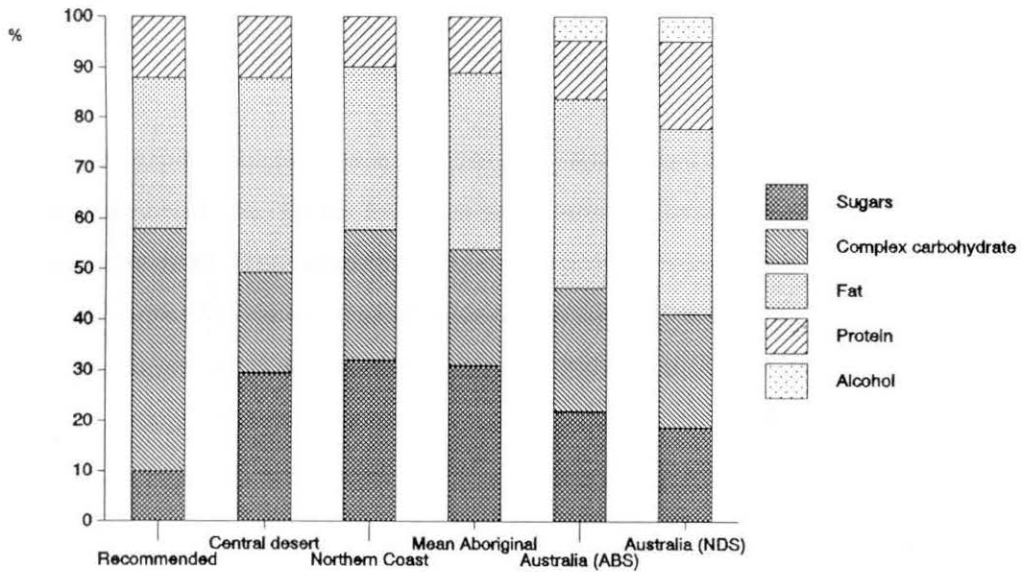


Figure 3.1. Contribution of macronutrients to total energy intake, Aboriginal communities



Alcohol data not available for Aboriginal communities
 Australia (ABS): Apparent Consumption of Foodstuffs and Nutrients (ABS, 1987a)
 Australia (NDS): National Dietary Survey of Adults: 1983 (CDCSH, 1987a)

Figure 3.2. Contribution of macronutrients to total energy intake, regional mean of Aboriginal communities, Aboriginal communities combined and apparent consumption data for the wider Australian community

Most of the energy available in Aboriginal communities appeared to be derived from total carbohydrate. This was particularly so in the northern coastal communities, where carbohydrate contributed to a mean of 58% of total energy intake. Complex carbohydrate contributed to a greater proportion of total energy intake in the northern coastal communities, (26% compared with 20%), but sugars had a similarly high profile in both environments (32% and 30% of total energy intake in northern coastal and central desert communities respectively). The ratio of complex to simple carbohydrate for northern coastal and central desert communities was 0.8 and 0.7 respectively. The proportion of total energy intake derived from sugars was higher in all Aboriginal communities than for wider Australia.

In both Aboriginal communities and the wider Australian community as determined by apparent consumption data the proportion of energy derived from protein was comparable to the recommended proportion of 10-12% (CDCSH, 1987b). However this proportion was 17% for wider Australia as determined by the national dietary survey.

The contribution of fat to the total energy intake was 38.7% in central desert and 32.7% in northern coastal communities, compared to the recommended proportion of 30% (CDCSH, 1987b). Figures for wider Australia were 36.8% for the national dietary survey and 37.5% for apparent consumption data.

Mean daily per capita macronutrient intakes for Aboriginal communities and wider Australia are presented in Table 3.4. Apparent energy intake was similar in both Aboriginal environments (approximately 13,250 kJ). However intake of fat was 18% higher ($t=2.25$, $p<0.05$), intake of protein was 23% higher ($t=4.34$, $p<0.01$) and intake of complex carbohydrate was 23% lower ($t=-5.21$, $p<0.01$) in the central desert communities.

Table 3.4. Apparent intake of energy and macronutrients in Aboriginal communities and wider Australia (per capita per day)

	Australia (ABS)	Australia (NDS) [‡]	Aboriginal Communities		
			Combined (Mean) n=6	Central Desert (Mean±se) n=3	Northern Coastal (Mean±se) n=3
Energy (kJ)	14497	9210	13254	13254±779	13254±536
Protein (g)	99	90	87	96±1.0	78±3.9
Fat (g)	147	93	125	136±4.8	115±7.8
Total carbohydrate (g)	405	233	450	414±48	486±52
Complex carbohydrate (g)	200	126	191	167±8.1	217±15
Sugars (g)	205	108	258	247±48	269±38
Alcohol (g)	23	17	n/a	n/a	n/a

Australia (ABS): Apparent Consumption of Foodstuffs and Nutrients (ABS, 1987a)

Australia (NDS): National Dietary Survey of Adults: 1983 (CDCSH, 1987a)

[‡] not strictly 'apparent consumption' data

Energy intake was higher in Aboriginal communities than the energy intake derived from the national dietary survey, but similar to that derived from apparent Australian consumption data. Apparent per capita intake of sugars in Aboriginal communities was approximately double the intake reported by the national dietary survey but closer to that described by apparent consumption data. Per capita fat intake in the central desert communities was approximately 30% higher than that measured by the national dietary survey, but slightly less than that described by apparent consumption figures for Australia.

The apparent per capita daily intake of nutrients in northern and desert communities expressed as a percentage of the community adjusted recommended intake for each nutrient was illustrated in Figure 3.3.

For all communities the apparent intake of sugars was very high while intakes of energy and fat appeared excessive and intakes of dietary fibre, some minerals (calcium and zinc) and some vitamins (retinol equivalents, riboflavin, vitamin E and folic acid) appeared low (that is, less than 66% of the RDI for the community as a whole).

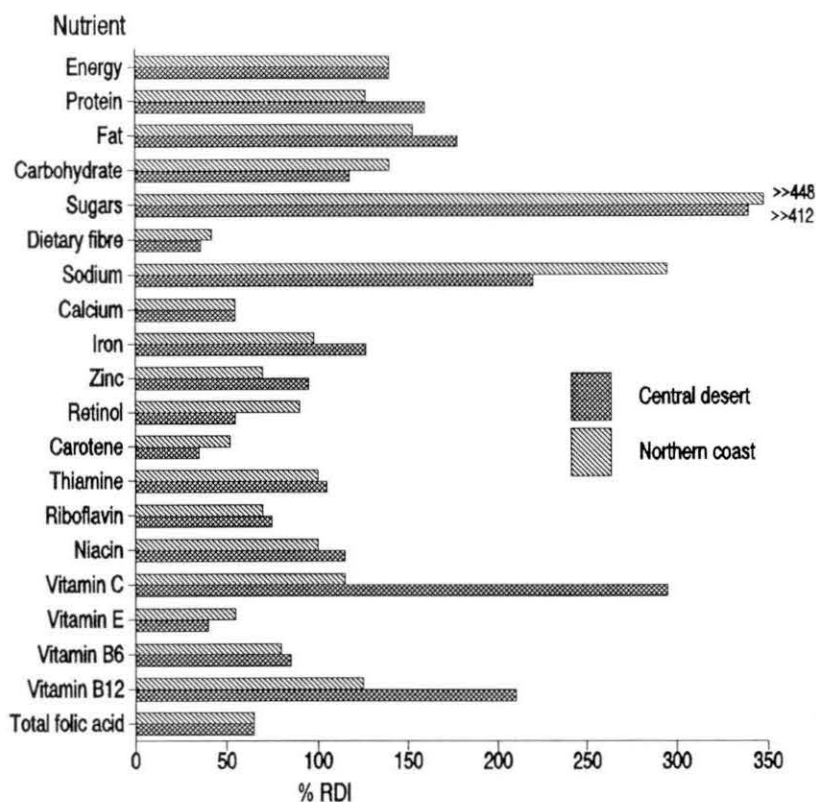


Figure 3.3. The apparent per capita daily intake of nutrients in Aboriginal communities as a percentage of community adjusted recommended intake

Only 18 types of foods each provided more than two percent of the total energy intake (Figure 3.4). Of these, sugar, meat, white flour and bread provided over 50 percent of energy in the northern coastal communities; and sugar, flour and meat alone, provided over 60 percent of energy in the central desert communities.

Fatty meat and meat products from beef, lamb and manufactured foods provided 66% and 36% of apparent total fat intake in the central desert and northern coastal communities respectively (Figure 3.5). Beef was the most popular meat providing 34% total fat in the desert and 20% total fat in the coastal communities. Lamb provided the bulk of fresh meat turnover only in the one central Australian community where sheep had been introduced earlier this century. Dairy products provided less than 8% total fat intake. The ratio of polyunsaturated fatty acids to saturated fatty acids was 0.28 in the centre and 0.43 in the northern communities. In both regions the major sources of polyunsaturated fatty acids was polyunsaturated margarine. Apparent cholesterol intake was higher in the central

desert communities (390.5 ± 24.8 mg) than the northern coastal communities (267.7 ± 14.3 mg) ($t=4.28, p < 0.01$).

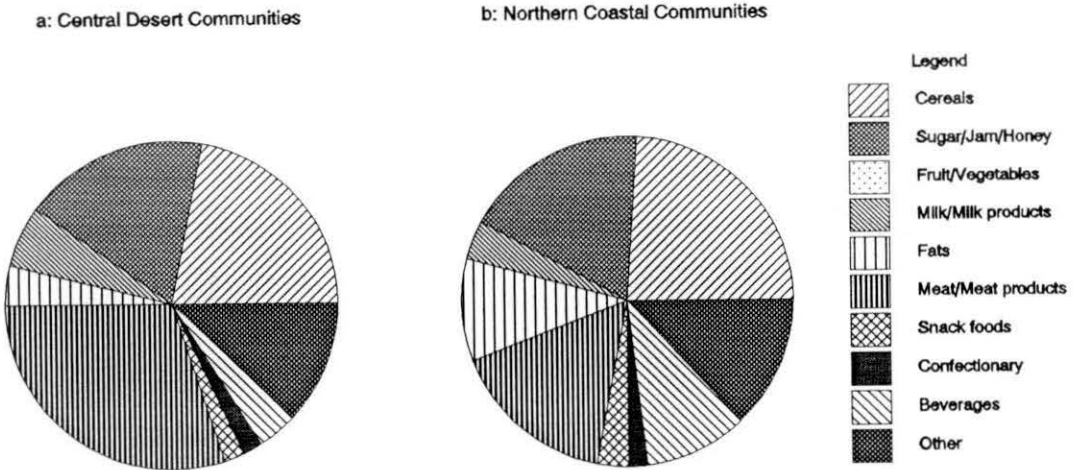


Figure 3.4. Foods contributing to total energy intake, Aboriginal communities

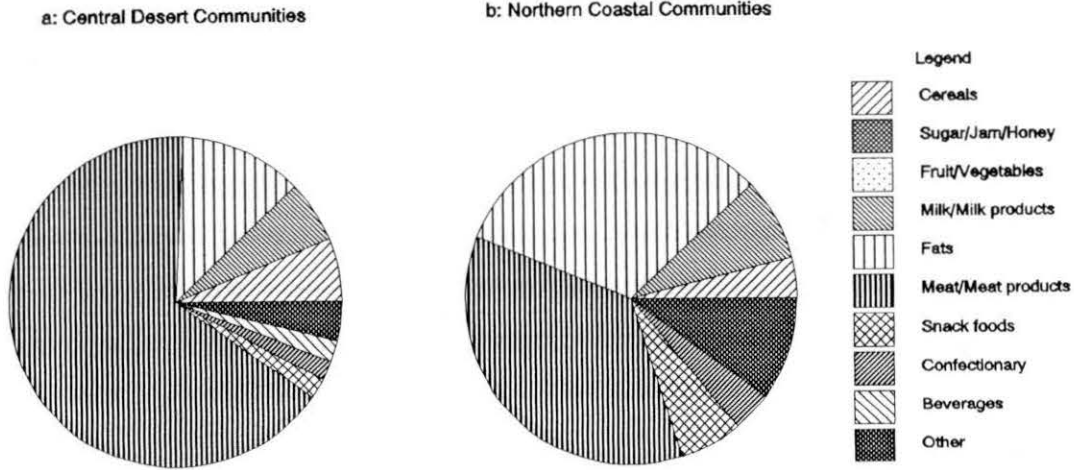


Figure 3.5. Foods contributing to total fat intake, Aboriginal communities

In both regions white sugar *per se* contributed approximately 60% of all sugars apparently consumed (Figure 3.6). The biggest difference in source of sugar between coastal and desert communities was for sweetened carbonated beverages, which provided 16% of all sugar intake in the former and 6% in the latter.

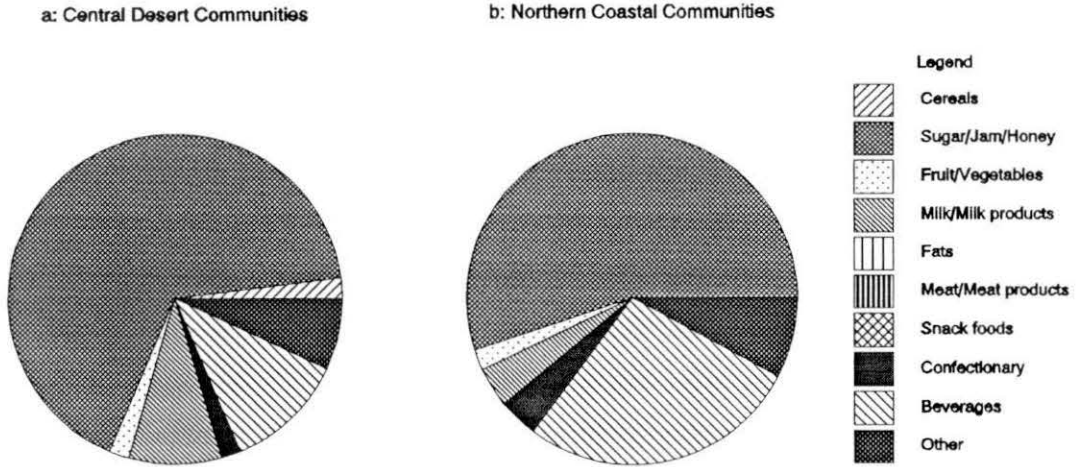


Figure 3.6. Foods contributing to total intake of sugars, Aboriginal communities

Apparent intake of total dietary fibre was low at 13.7 g in the central and 14.9 g in the northern communities. Mean total dietary fibre intake was 14.3 g per capita per day compared with recommended intake of 30-35 g per day and mean Australian adult intake of 21.5 g per capita per day (CDCSH, 1987a). Cereals were the major source of dietary fibre (Figure 3.7), and apparent intake of soluble fibre was only 2.9 g per person per day. The poor quality of the carbohydrate consumed was also reflected by the low dietary fibre index of 2.0 g of dietary fibre per 1000 kJ of total carbohydrate, compared with 5.8 g of dietary fibre per 1000 kJ of total carbohydrate for the wider Australian community, as calculated from national dietary survey data³.

³ Unfortunately apparent Australian consumption data does not include analysis for dietary fibre

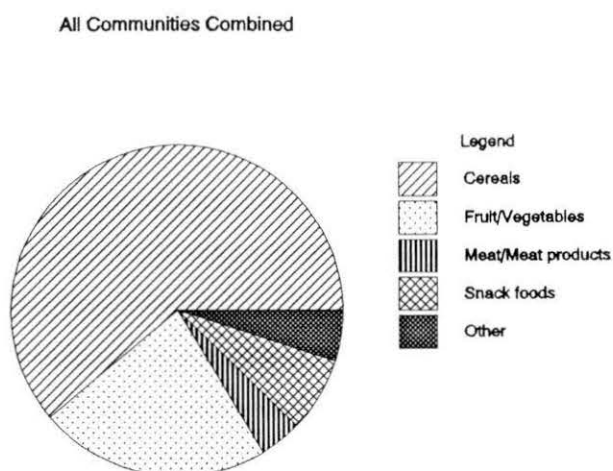


Figure 3.7. Foods contributing to intake of dietary fibre, Aboriginal communities

3.6. Discussion

3.6.1. Methodological considerations

Major advantages of the store-turnover method were that it related to the diet of the whole population, was specific to relatively small geographical areas, was relatively non-invasive, rapid, easy and inexpensive. It also did not rely on subjective assessment of diet or memory and avoided language, literacy, numeracy and cultural factors which may be a problem with direct measurement or recall of diet in Aboriginal communities. Potentially, retrospective data could also be collected.

Limitations of the store-turnover method, as applied in this preliminary study, included the lack of consideration of wastage, bulk storage and very slow stock, dietary intake from sources other than the store (including bush foods, store foods purchased from outside the community, alcohol and kava) and food distribution patterns within the community. Difficulties with determination of the population denominator and the application of appropriate nutrient composition values for foods as available in community stores were also problematic. Each possible limitation was considered in light of the results of the store-turnover method and the unique circumstances of food supply and dietary habits in centralised Aboriginal communities in remote areas. Approaches to the estimation of the

effect of enduring limitations are suggested. Aspects of the face validity of the store-turnover method are also discussed.

3.6.1.1. Wastage

It was contemplated that wastage of certain types of foods may have explained the high energy intake suggested by the results of the store-turnover method. The five main foods which contributed to energy intake were beef, sugar, flour, bread and carbonated beverages. Apparent intake of sugar alone, and flour and bread combined, was similar in both regions, despite differences in environment, climate, style of purchase and storage of these foods in each area; this suggested that spoilage due to climatic extremes may not be as high as previously estimated (section 1.8.7). Sugar appeared to have most potential for wastage, although in the pilot study of weighed dietary intake (Chapter 2) actual weighed intake of individual sugar intake was similar to that derived from store-turnover data. Sweetened soft drink was usually brought by the can (375 ml) or bottle (1.25 l), and shared from person to person with little observable wastage. Meat was a prized food which was most frequently purchased immediately before consumption with little observed wastage. The differences in meat consumption between northern and central communities may be due to greater accessibility of traditional animal foods, particularly riverine and sea foods, in the northern coastal communities.

3.6.1.2. Foods from other sources

The major source of foods other than from the community store was the 'bush'. Both the availability and accessibility of traditional foods has been compromised by the demise of nomadic lifestyle and the advent of centralised communities (section 1.5.3.2). The many methodological problems with the quantitative determination of daily bush food consumption for individuals within a community have been widely documented (section 1.8.3.1). The systematic *qualitative* estimation of the consumption of traditional foods on a community basis could be attempted in subsequent studies as an adjunct to the application of the store-turnover method.

Many communities in the Northern Territory are officially 'dry' and no alcohol is permitted. For communities where a licensed club is available turnover of alcoholic beverages may also be determined from invoices. However access to such potentially sensitive data was granted only in one community during this study; analysis of social club

invoices at community N3 indicated that mean intake of alcohol was consistent with previously available data (Watson *et al.*, 1988), being in the order of 30 g per person per day (section 1.5.4). Though not strictly a food, turnover of kava may also be available from major distributors on a community basis.

3.6.1.3. Bulk storage and slow stock

Generally storage facilities are limited in remote community stores due to restricted availability of space, high costs of refrigeration and cash flow problems. In the last decade increased efficiency and frequency of food transport has avoided the necessity to stockpile goods for an extended period in remote communities. Store managers may be able to draw attention to slow stock items, and, if necessary, store invoices can be searched over a longer time period to determine turnover for these specific items. However, it is likely that given the regular ordering patterns of major food items identified by the store-turnover method, slow stock not appearing on invoices within a three month period would also have negligible effect on the apparent dietary intake of the community as a whole.

The effects of both of these problems may be investigated by comparison of store-turnover data with food sales derived from stock take data which is generally available on a yearly basis:

$$\text{Food sold} = \text{Stock at start of Year} + \text{Food Delivered} - \text{Stock at end of Year}$$

The closer the agreement between stock held at the start and the end of the year, the less error involved in the assumption that the quantity of food sold approximates the quantity of food delivered. The larger the period addressed by the store-turnover method, the greater the potential agreement between the results of that method and stock take data. However, the store-turnover method was applied over a three month period rather than annually, to enable investigation of the effect of seasonality on dietary intake and hence to produce a potentially more sensitive measure of dietary intake. Additional stock taking procedures may also be conducted at less than twelve month intervals throughout the year to increase the sensitivity of derived nutritional data. However, relative to the application of the store-turnover method, stocktakes tend to be labour intensive, expensive, and invasive, requiring the collaboration and co-operation of store managers, staff and consumers.

3.6.1.4. Application of food composition tables

The food composition data applied were not necessarily relevant to the foods available in Aboriginal community stores. For example, the nutrient composition of perishable items may be affected by long distance transport in unfavourable conditions. However, without conducting expensive and time-consuming chemical analysis on actual food samples available for consumption, all dietary survey methods applied in remote Aboriginal communities would be subject to similar errors. The application of Australian food composition data would appear to represent the best approximation of the nutrient composition of food data derived from the store-turnover method (section 1.7.5).

3.6.1.5. Population determination

Results expressed in terms of apparent per capita intake and comparisons with community adjusted recommended dietary intakes are essentially dependent on accurate estimation of these denominators. Accurate determination of community population is notoriously difficult in Aboriginal communities (section 1.6.1). The result of a single census was unlikely to reflect the population of each community during the preceding three months. It appeared that the best method to quantify the number and demographic composition of those relying on the community store over a three month period was to conduct detailed, frequent, regular community censuses; a potentially expensive and invasive process necessitating regular community visits or training of community residents as observers (section 1.8.8).

It should be emphasised that results expressed as the proportion of macronutrient contribution to total energy intake, nutrient profiles and nutrient densities, were essentially independent of accurate estimations of numbers and the sex and age proportions of each community population and could be considered where reliable population denominators were not available.

3.6.1.6. Seasonality

It was considered that seasonality of the demand and supply of food may have explained some differences in the dietary intake between Aboriginal communities in the two regions. For example, the comparatively high turnover of sweetened carbonated beverages in community N3 may have been due to climatic considerations as the store-turnover

method was applied in that community over the wet season (the hottest and most humid period of the year). Results indicated that the effect of seasonality should be considered in subsequent studies when describing both inter- and intra- community variation in apparent dietary intake from community stores.

3.6.2. Discussion of results

Following appraisal of the apparent limitations of the application of the store-turnover method in this preliminary study, it was considered useful to contemplate the implications of the results pending validation of the method.

3.6.2.1. Quantitative results

The apparent energy intake as determined by the extended store method was very high but similar to previous quantitative estimations (section 1.8.11). If the intake of energy and component macronutrients was less in real terms, the corresponding reduction of intake of vitamins and minerals would result in such low levels of apparent intake that frank deficiencies could be expected throughout the communities. With the possible exception of folic acid, this is not the case (section 1.6.10). The high energy density may contribute to the high prevalence of obesity described in remote Aboriginal communities (section 1.6.7). The extremely high intake of refined carbohydrate measured may also help to explain the hypertriglyceridaemia frequently described in Aboriginal communities (section 1.6.6.2).

The high percentage of both energy and fat derived from meat was largely due both to the high intake of meat and the high proportion of fat in the meat. Cuts of meat available in Aboriginal communities were low in quality, with a mean of 40% by weight in samples of beef, which provided in the order of 90% of all kilojoules available from the consumption of the meat. Fat sources in the Aboriginal communities tended to be saturated, compared with traditional sources which provided much less total fat but a greater proportion of polyunsaturated fatty acids (section 1.2.2.3).

White flour and white bread are not rich sources of dietary fibre; but, due to the large quantity of these foods consumed and the comparatively low intake of fibre rich foods such as fruits and vegetables and wholegrain cereal products, they were the main sources in these Aboriginal communities. The apparent intake of soluble fibre was very low.

Traditionally the northern tropical environment provided a greater abundance and variety of foods than the central arid regions (section 1.2.2.2). It has been suggested that seafood may continue to provide the bulk of protein intake from traditional sources in coastal communities (section 1.8.10). The potentially greater reliance on central desert stores as a source of animal protein may explain the higher intake of beef in this region. Climatic differences may help to explain the higher intake of carbonated beverages in the northern coastal communities. It should be noted that in two of the three central desert communities, there has been a past attempt to promote fruit juice alternatives to sweetened carbonated beverages; dietary results may also have reflected the success of this promotion.

Compared with the wider Australian diet, fewer foods contributed significantly to the total energy intake in Aboriginal communities.

The relative proportion of total energy derived from major macronutrients in the Aboriginal communities was distorted due to the extremely high intake of refined carbohydrates. Under these circumstances (that is, when one macronutrient predominates), caution should be applied in the interpretation of direct quantitative comparisons with recommended proportional energy intakes. However marked contrasts in the 'style' of the diet were revealed by such comparisons.

The proportion of energy derived from sugars as opposed to complex carbohydrate in Aboriginal communities was much greater than that of the wider Australian community, and approximately four times that recommended (CDCSH, 1987b). The fact that these communities derived most of their sugar from refined sugar *per se* was in marked contrast to the wider Australian community, where manufactured foods provided three times the amount of sugar to the diet as sugar *per se* (ABS, 1987a). The contribution of fat to total energy in Aboriginal communities appeared lower than the national figure due to the exceptionally high intake of refined carbohydrate in these communities; however, in absolute terms, particularly in the central desert, apparent intake of fat was high. The current high contribution of meats to the total fat intake in desert communities (66%), was similar to the profile in the wider Australian community over 50 years ago (ABS, 1987a). The comparatively low proportion of fat from dairy products in Aboriginal communities apparently reflected the lack of popularity for these items; dried milk powder was the main dairy food consumed. The ratio of saturated to polyunsaturated fats in central Australia was higher than that of the wider Australian community (CDCSH, 1987a).

Protein contribution to energy appeared lower than in wider Australia due to the relatively higher intake of refined carbohydrate, but appeared adequate in absolute terms.

3.6.2.2. The limited variety of foods

The limited variety of foods contributing to total energy intake may have been due to several factors. Contemporary staples (flour, sugar and tea) were extensively used by non-Aboriginal Australians in colonial days and were the first foods introduced at ration and cattle stations (section 1.5.2). The apparent continuing popularity of these foods may have been due to practical factors such as their relative durability, low bulk, transportability, cheapness and the simple storage and cooking facilities required. Conservative dietary preferences exhibited by Aboriginal people may also be an important factor (section 1.2.3.1). With the advent of the cattle industry in the north, and refrigeration/freezer facilities in remote communities, fresh meat is more readily available and has replaced the more expensive preserved meat and meat/cereal mixes used in the past. Similarities between some store foods, such as fatty cuts of meat and white sugar, and traditionally prized foods containing fat (such as goanna and turtle fat) and sugars (such as wild honey and honey ants) may also help explain their popularity (section 1.5.3.1). As Aboriginal income has increased, a greater range of foods has become available through community stores, but perishable items, such as dairy foods, fruit and vegetables, still tend to be in short supply. These items carry the risk of high overheads which store managers may not be willing to bear; food items in community stores, particularly perishable items, are relatively expensive in remote areas (section 1.5.3.3).

Distance from major centres and poor communication are two factors of physical isolation which have a direct effect on food supply. Isolation may tempt wholesalers into passing poor quality, unwanted or out of date stock to remote stores. Assumptions may be made that Aboriginal consumers are less discriminating, but poor quality food items, particularly fruit and vegetables, may not be generally acceptable to people who traditionally had a strong preference for, and an extensive supply of, very fresh foods (section 1.2.3.1). Spoiled foods tend to remain unpurchased and fuel comments such as: "*Aboriginal people do not like fruit*" which have been used to justify lack of stock.

3.6.2.3. Implications of results for community-based nutrition education programs

Supply and demand are tightly inter-woven in Aboriginal stores, as they are generally the

only convenient retail outlet available. Past attempts to control purchases by restricting choice and manipulating pricing structure in favour of nutritious foods without community initiative or support, can be considered paternalistic and have generally been unsuccessful (section 1.9.1.2). With community concern about the increasing prevalence of 'lifestyle' diseases, in particular those affecting young adults, and with increasing Aboriginal control over community stores, opportunities now appear to exist for Aboriginal people to influence store policy and to promote healthier diets by increasing the range and variety of food. Store-turnover data could highlight potential areas for change by the community.

Full consultation and feedback of information is a very important component of any research initiated by Aboriginal communities. Nutrition information from store-turnover data was interpreted to individual communities in culturally significant ways, including visual displays of comparisons of traditional 'bush-foods' and contemporary 'store-foods' rather than discussion of nutrients. It was more meaningful, for example, to display mean sugar intake as half a cup or 39 sugar cubes, than to describe it as 155 g. Similarly fat intake was interpreted as a display of weighed fat from meat.

The dietary results were of particular relevance to nutrition programs in that they highlighted that different Aboriginal communities may have different dietary profiles.

Potentially, store data could be used to focus on dietary guidelines of particular relevance to Aboriginal communities and could be used to evaluate progress in community based nutrition programs. Although the Australian dietary guidelines (NHMRC, 1989) are of general relevance to Aboriginal communities, results of the store-turnover method indicated that the foods contributing to targeted nutrients were different in Aboriginal communities than in the wider Australian community.

In summary, culturally acceptable dietary messages suggested by store-turnover data were:

- Increase consumption of fruit and vegetables
- Increase consumption of wholemeal bread and wholegrain cereals like porridge
- Reduce consumption of refined sugar *per se*
- Choose lean meat, discarding visible fat

3.7. Major points

Results generally indicated that in all communities studied, the apparent intake of energy, sugars and fat was excessive, while the apparent intake of dietary fibre, some minerals (calcium and zinc) and some vitamins (vitamin A equivalents, riboflavin, vitamin E, vitamin B₆ and folic acid) appeared to be relatively low.

Apparent energy intake was similar in both environments and there were similarities between desert and coastal communities in the per capita intakes of refined sugar, vegetables, and intakes of flour and bread combined. However some differences between the dietary intake of central desert and northern coastal communities were apparent.

Of all foods recorded, only 18 contributed greater than two percent to the total energy intake. Of these sugar, flour, bread and meat provided over 50% of total energy intake in northern coastal communities, and sugar, flour and meat provided over 60% total energy intake in central desert communities.

Over 60% of the high fat intake was derived from fatty meats in central desert communities, and over 35% in northern coastal communities. 60% of the high sugar intake was derived from sugar *per se* in both regions.

Generally there were major differences between apparent food and nutrient intake in Aboriginal communities and national figures. Intakes of sugar, white flour and sweetened carbonated beverages were much higher, and intakes of wholemeal bread, fruit and vegetables were much lower in Aboriginal communities. In Aboriginal communities, intake of energy, total carbohydrate and sugars were much higher and protein intake was much lower than results derived from the national dietary survey, but results were more closely aligned to apparent Australian consumption data.

The results highlighted potential implications for community-based nutrition intervention programs.

CHAPTER 4: A COMMUNITY-BASED NUTRITION INTERVENTION
PROJECT: METHODS

This chapter outlines the methods applied in the initiation, planning, implementation and evaluation of a community-based nutrition intervention project in a remote, centralised Aboriginal community in northern Australia.

4.1. Introduction and study design

Food and nutrient intake, as determined by the store-turnover method, together with anthropometric, biochemical and haematological measurements of nutritional status, was used as a rational basis for the planning, implementation and evaluation of a community-based and initiated nutrition intervention project in a remote, centralised Aboriginal community in northern Australia.

For a twelve month period from June 1989, all variables were measured at three month intervals. Store-turnover was also measured in a similar community. In order to investigate the relative seasonal changes in community dietary intake, store-turnover was measured retrospectively in both the intervention and control communities over each three month period from June 1986 to June 1989.

The results of the store-turnover method were validated congruently against biological indicators of nutritional status measured at three month intervals throughout the intervention period. Aspects of face validity of the method were also considered; in particular those features arising from longitudinal application of the method.

The study design required dietary change to occur during the intervention program. Hence the practical design potentially provided advantages to the participating community and was highly ethical. In order to maximise the potential for change, several strategies were applied simultaneously, addressing two major issues; increasing motivation of community members, and the promotion of an increased variety of suitable food choices in the store.

4.2. The communities

4.2.1. Minjilang, Croker Island

Croker Island lies 240 kilometres north-east of Darwin, and is separated from the north-eastern coast of the Coburg Peninsula by the Bowen Strait which is under two kilometres wide at its narrowest point (Figure 4.1). The island covers 325 square kilometres, is 42 kilometres from north to south and 18 kilometres from east to west at the widest point. The community, Minjilang, is built on a rise on the east coast of the island at Mission Bay. The township is comprised of a large community centre/council building, health clinic, primary school, bank agency, community store, women's resource centre, church, cemetery, demountable accommodation for visitors, two ablution blocks, several workshops, sporting oval and 34 residences.

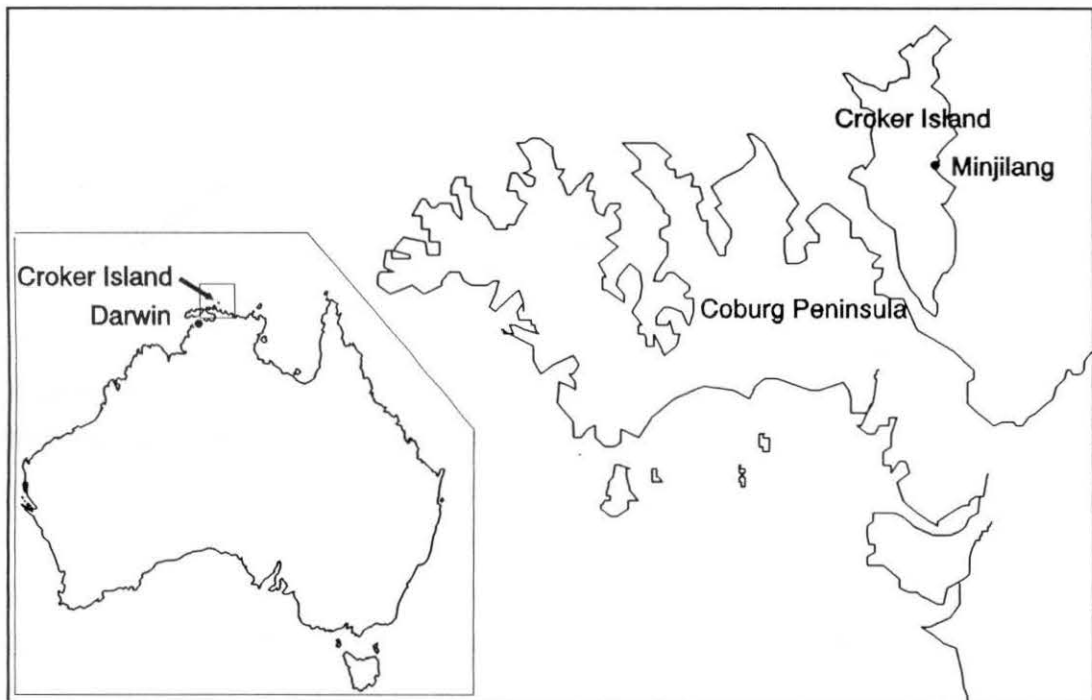


Figure 4.1. Location and Map of Croker Island

The island covers a diversity of habitats. Eucalypt forest and open woodland dominate the centre of the island, with pockets of dense rain forest in the lower lying areas. To the south-west is a flood plain with stands of paperbarks (*melaleuca sp.*) and pandanus. The plain is drained by mangrove-lined creeks which run to the south-west and the south-east. Mangrove swamps, small protected coves and vast sandy beaches surround the island.

The climate is monsoonal, being characterised by two seasons: a hot, dry period from approximately May to October (dry season) and a hot, wet period from approximately October to May (wet season) (Figure 4.2) (Bureau of Meteorology, 1982). The average rainfall is approximately 1,450 millimetres, 95% of which occurs between November and May. North-westerly winds predominate between November and March when particularly rough seas may occur, and south-easterly to easterly winds are most common during the remainder of the year. Tidal variation is high at approximately three metres. Cyclones are potentially hazardous during the Wet season.

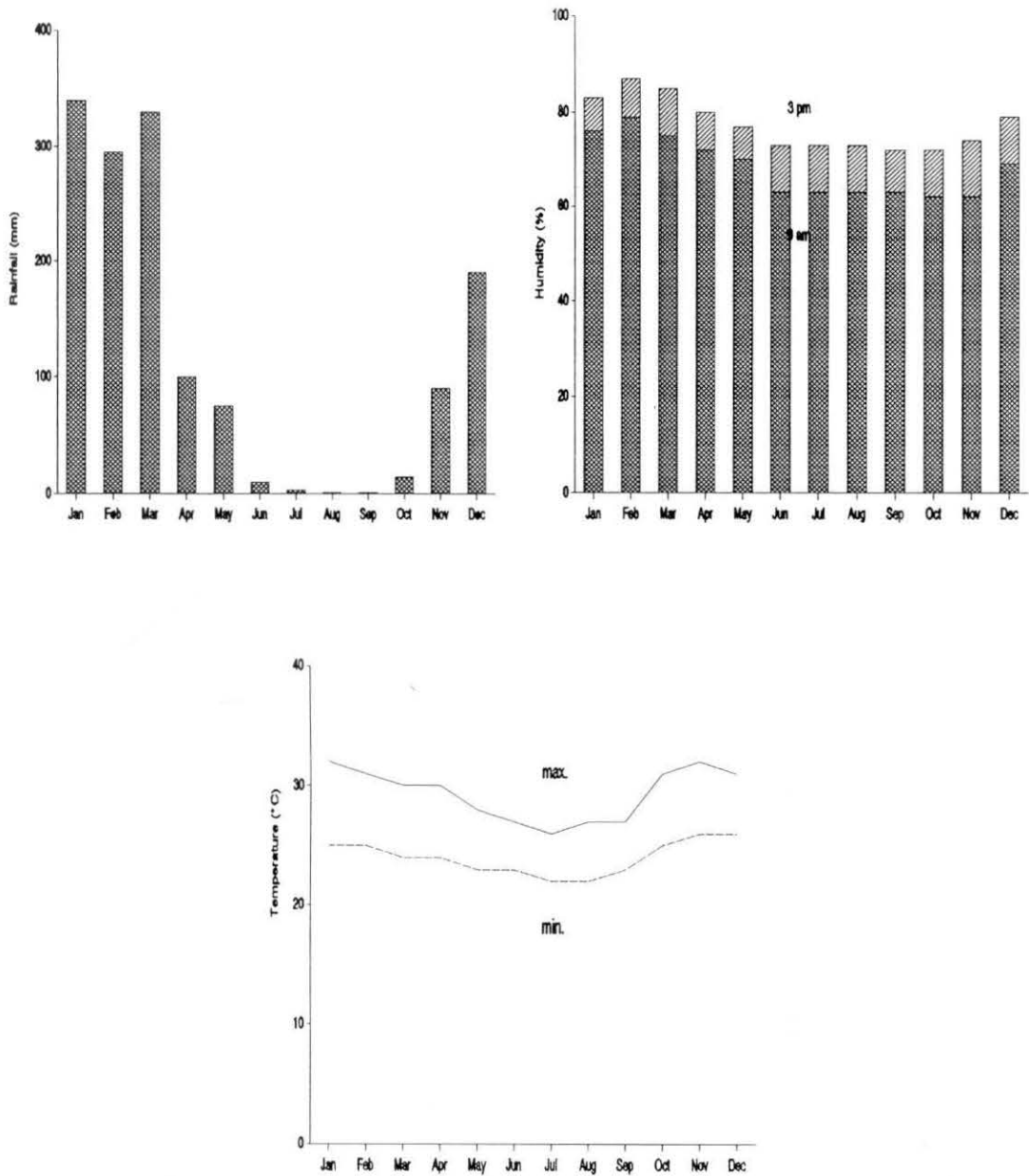
The traditional Iwadja calendar (Figure 4.3) reflects the relationship between seasonal climatic factors and cultural and natural events, including the seasonal cycle of traditional hunting and gathering activities.

The two traditional land-owning groups of Croker Island are the Iwadja to the south, and the Marrgu to the north. Both groups live together at Minjilang, and tribal affiliations do not affect the location of residences in the settlement. Relatively small numbers of Maung from Goulburn Island, and Gunwinggu from the mainland, also live at the community. There are tribal connections between Croker Island groups and the Tiwi from Bathurst and Melville Islands and also people from other Arnhem Land communities, particularly Maningrida.

It is generally believed that Aboriginal groups have inhabited the Coburg Peninsula/Croker Island area for over 40,000 years (NT Conservation Commission, 1987:71). Macassan trepangers regularly visited the area from the 17th century to the early 20th century. The local people traded with the visitors and adopted several Malay customs including the construction and use of dug-out canoes. Nearby Coburg Peninsula was the sight of the second and third European attempts to settle in Northern Australia; Fort Wellington in 1827 and Victoria settlement in 1838. Iwadja contact with Europeans dated from those early years and was likely to have pre-dated European contact at the control community by over 60 years.

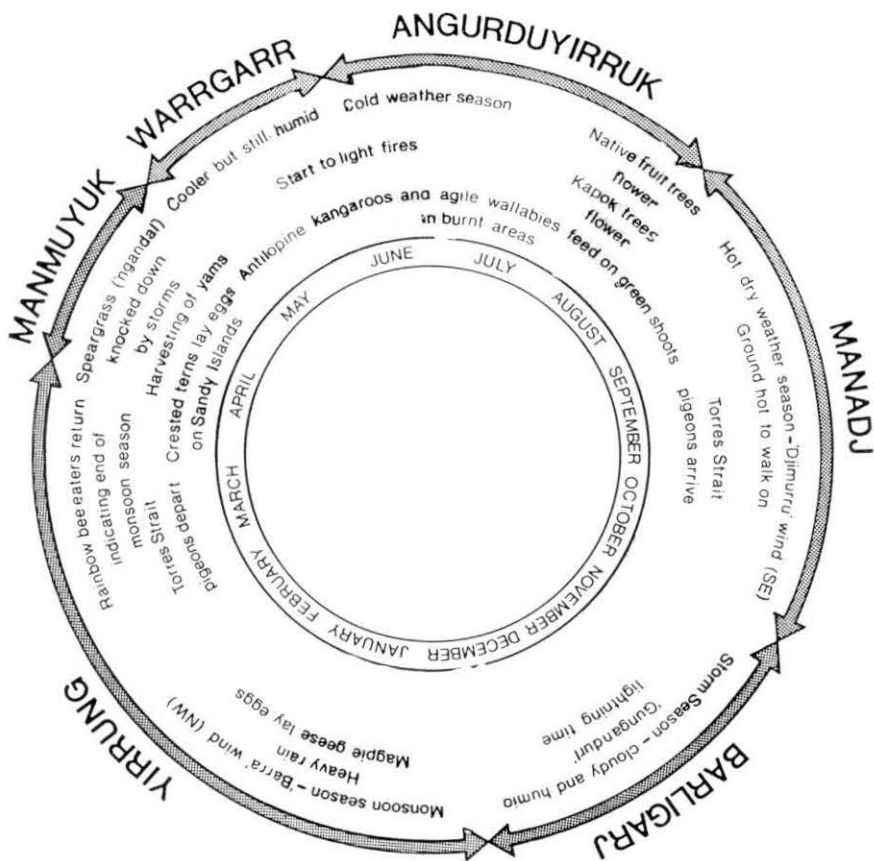
Minjilang was established by the Methodist Overseas Mission in 1940 (Cole, 1980). Forty "*part Aboriginal*" people from the Kahlin Compound in Darwin, and twenty-eight from Central Australia, were taken to the island in 1941, where the children were "*on the threshold of a new life, segregated from white man's vices and black man's degradation*" (Cole, 1980:39). Following a break during the war years, the mission continued until

1967, when the organisation and the mixed race mission children were moved to Darwin.



Source: Bureau of Meteorology, 1982

Figure 4.2. Rainfall, humidity and temperature graphs, Croker Island



Source: NT Conservation Commission, 1987

Figure 4.3. Traditional Iwadja Calendar

During the mission years the local Iwadja were separated from the mission children, and lived in corrugated iron humpies along Mission Bay beach. However several local children were permitted to attend school during the later years of the mission. As adults, several apply their literacy and numeracy skills as employees of the community council, Health and Education Departments. Although the pilot, mechanic, one school teacher, store-manager, essential services manager and book-keeper are European, there are relatively fewer European residents at Minjilang than comparable Aboriginal communities. Unemployment benefits and social security pensions are made to those residents not employed on the island. Traditional owners also receive royalty payments from commercial fishing and mainland buffalo industries, and uranium mining in the Alligator Rivers region.

Official population figures varied greatly according to source and year, and tended to be contradictory (Table 4.1). The accuracy of these population estimates has been questioned

due to seasonal fluctuations (NT Government, 1989). When scrutinised in detail, the figure provided by the Northern Territory Department of Health and Community services in 1988 was found to be a gross overestimate of the Croker Island population as it included those people who permanently resided at nearby Coburg Peninsula and who were provided with medical care from Minjilang clinic staff. More detail of the population derived from data supplied by the Australian Bureau of Statistics (ABS, 1987b) was provided (Table 4.2). Dwelling occupancy was comparatively low at approximately 5.8 persons per dwelling (NT Government, 1989).

Table 4.1. Population estimates, Minjilang

Year	Population	Source
1986	111	Department of Community Development, NT (1989)
1986	60-80	Office of Local Government, NT
1986	149	Australian Bureau of Statistics (ABS) Census (1986b)
1988	245	NT Department of Health and Community Services (Fong et al, 1988a).

Table 4.2. Demographic data Minjilang June 1986 (excluding outstations)

Age group (years)	Males	Females	Total
0-4	3	7	10
5-9	7	7	14
10-14	10	9	19
15-19	8	7	15
20-24	5	5	10
25-29	6	7	13
30-39	8	8	16
40-49	6	8	14
50-54	2	4	6
55-59	4	2	6
60-64	2	3	5
65-69	2	4	6
70+	0	0	0
TOTAL	63	71	134

Source: ABS (1987b)

At the time of the study, the community was served by an air commuter service operated six days a week by Arnhem Air Charter Pty. Ltd., which connected Minjilang with Darwin, Warrawi, Maningrida and Milingimbi. Under the aviation policy of the Northern Territory Government, Arnhem Air Charter held exclusive rights to all air charter on these routes. Arnhem Air Charter service used the major airstrip on Croker Island, which was located 10 kilometres west of Minjilang. During the wet season from October to April the road across the flood plains to this airstrip is impassible, and a detour of 30 kilometres is taken along the south coast to the settlement. A smaller airstrip is located three kilometres west of Minjilang to accommodate special charter and air medical services.

Barge Express Pty. Ltd. runs a fortnightly barge service between Darwin, Minjilang, Warrawi and Maningrida. At Croker Island the barge landing is situated three kilometres south of Minjilang in Mission bay, and is eight kilometres by road from the community.

Until May 1990 when full telephone services were commissioned, communications were restricted to a high frequency radio telephone located in the community council building and radio transceivers operating on the VJY service. Domestic television reception (ABC, Imparja) is available via satellite dish.

Water demand is adequately met by three bores, the community is fully sewered, the gravel roads are in good repair and well maintained, and the reticulated supply of electricity is sustained by three reliable diesel generators. Buildings are well maintained by council housing officers.

The community store is managed by the Aboriginal owned enterprise Arnhem Land Progress Association (ALPA), and stocks a wide variety of food, clothing and hardware. Two Aboriginal store-workers and check-out operators are employed to work with the European store manager.

The clinical health needs of the community are funded under the Grants-in-aid scheme of the Northern Territory Department of Health and Community Services. A Senior Aboriginal Health Worker is in charge of the Harry Rudbuk Health Centre and two additional Aboriginal health workers are employed. The clinic was in radio contact with a Medical Officer based in Darwin prior to connection of telephone services in May 1990. Medical visits are made fortnightly via the Aerial Medical Service from Darwin. Emergency medical evacuations are also made via this service to Royal Darwin Hospital.

The nearest police are located on the mainland at Oenpelli and can be contacted by radio or telephone.

Although the island has been considered to display excellent agricultural and pastoral potential, the small market gardening, dairying and butchering projects established during the mission years have not survived. Several large mango and tamarind trees provide seasonal fruit, but the mission citrus trees no longer bear edible fruit.

The environmental diversity of Croker Island ensures an abundance and variety of bush foods, although these tend to be hunted and procured irregularly (section 5.4). The Iwadja and Marrgu are sea-faring people; turtles, dugong, crabs, shellfish and fish are very popular foods when produced. Feral animals present on the island include pigs, cattle and Timor ponies. Marrgu elders have a dry season camp on the western coast of the island at Palm Bay. Iwadja elders tend to camp at the southern end of the island at Point David during the dry season.

4.2.2. The control community

For ethical reasons it is necessary to protect the identity of the control community which is also located on an island, approximately 300 kilometres from Darwin.

The island is substantially smaller, flatter and more arid than Croker Island and is covered in an open forest of eucalypts, ironwoods, cypress and pandanus. The island was a traditional ceremonial place, and was not permanently inhabited until a mission was established by the Methodist Overseas Mission in 1916. The community is now operated as an Association and consists of a council office, bank agency, school, women's resource centre, church, cemetery, store, health clinic and sporting oval. Feral cattle, buffalo, pigs and goats inhabit the island. The seas are as rich as Croker Island, however comparatively little traditional foods are available on the island itself.

The main residential tribal group is the traditional land owners, the Maung, with Gunwinggu, Waling and Gunavidjii from the mainland, and the Galpu from Elcho Island. Both communities have powerful cultural ties. Marriages between one group of the control community and Croker Island people have a strong traditional basis and there is some genetic admixture between the two. In both cultures descent is matrilineal, although land is inherited patrilineally.

The island has a population of approximately 300. Again official population figures vary greatly according to source and year, and tend to be contradictory (Table 4.3). The age/sex profile of the population was similar to that of Minjilang (ABS, 1987b).

Although community members are employed in similar positions to Minjilang, the rate of Aboriginal employment is much lower at the control community. The community has a slightly greater proportion of European residents than Croker Island.

Water supply is adequate for the main community centre although problems with water supply in decentralised areas have prevented the permanent establishment of outstations on the island. Several locations are nevertheless inhabited for short periods during the dry season. Diesel generators sustain the reticulated supply of electricity. The septic tank/sanitary pan sewerage system has been considered obsolete and there are plans to implement a fully sewered system. Full telephone services were installed in March 1990. The community is also serviced by Arnhem Air and Barge Express under a similar schedule as Minjilang. The aerodrome is much closer to the community centre than at Minjilang and roads on the island are rarely impassible, even at the height of the wet season.

Table 4.3. Population estimates, Control community

Year	Population	Source
1960	200	Australian Bureau of Statistics
1974	250	Australian Bureau of Statistics
1976	230	Department NT (Water Resources Branch)
1980	267	Department of Community Development, NT
1981	277	Australian Bureau of Statistics
1985	258	Department of Community Development, NT
1985	311	Department of Aboriginal Affairs
1986	221	Department of Aboriginal Affairs
1986	321	Australian Bureau of Statistics, (1987b).
1988	300	Office of Local Government, NT

Clinical health services are also funded under the Grants-in-aid scheme of the Northern Territory Department of Health and Community Services, and the community employs a European nursing sister and three Aboriginal health workers. Other aspects of the health service operate under the same conditions as Minjilang.

The council acquired ownership of the community store in July 1977 and now employs ALPA to manage the business. There is a European store manager and several community members are employed as store-workers. A large variety of food, clothing and hardware is stocked.

None of the enterprises initiated during the mission years remain. A small oyster industry operated during the late 1970s but no longer continues.

4.3. Initiation of the intervention project at Minjilang

The project was initiated in response to a direct request from the Senior Aboriginal Health Worker at Minjilang (Mrs Daisy Yarmirr), on behalf of those adults living at the community who wished to be screened for serum lipid concentrations, particularly serum cholesterol. The entire intervention project developed from this initial request and aspects of that process are recorded in detail (section 8.3.1.1) due to the many public health and ethical implications.

4.4. Sampling methods

Due to the relatively small number of adults in the community, the sample aimed to include all available adults. For ethical reasons screening was offered to those available adults (18 years or over) who presented on a voluntary basis only.

4.5. Determination of demographic data

A list of the name and date of birth of all individuals who had resided at both Minjilang and the control community for any period since June 1988 was obtained from records of the Northern Territory Department of Health and Community Services (Fong et al, 1988a, 1988b). These lists were updated by cross-referencing against the current medical records held at the Harry Rudbuk clinic at Minjilang and, by non-Aboriginal medical staff, at the clinic in the control community.

At Minjilang, using the resulting list as an initial guide, a household census naming all individuals either residing at, or temporarily absent from, the community was conducted during the last week of May 1989. Date of birth was obtained from medical records filed at Minjilang clinic and at Royal Darwin Hospital. Where the actual date of birth was unknown, age was determined from the first day of July in the year of recorded birth, (a likely guesstimate in the case of several individuals over the age of 45 years). Population was determined by named census on a household basis for all age and sex groups every three months throughout the intervention period coinciding with each community survey.

A 'roll call' was also completed each fortnight from April 1989 throughout the year with the assistance of Aboriginal Health Workers and key informants where necessary. All new arrivals to the community and visitors were also recorded fortnightly. The mean population determined by 'roll call' for each three month period was used as the population denominator for analysis of the store-turnover data.

At the control community it was necessary to maintain negligible intrusion and therefore it was not possible to conduct a regular named census. A total population tally was supplied by Health Centre staff at the control community every three months to coincide with collation of store-turnover data. It is unlikely that this data was of comparable quality to that obtained at Minjilang, where more detailed data was required in order to evaluate the representativeness of the sample of volunteers at each survey. Where relevant, the concept of nutrient density was employed in analysis of the store-turnover data to minimise the effect of potentially inaccurate population data.

4.6. The community survey process

At each survey all necessary equipment (Addendum 1, A1.5) was flown to Minjilang where it was checked, and tubes and data sheets were numbered.

Biological surveys were conducted at the Harry Rudbuk Clinic, Minjilang, from approximately 7.00 am to 11.45 am for seven to ten days each survey period. Screening was restricted to the morning of week days in order that the samples collected for haematological assessment were measured at Royal Darwin Hospital on the same day.

In all surveys every effort was made to control for individual observer in order to minimise observer bias.

4.6.1. Consent

A consent form (Addendum 1, A1.4) was read and explained in simple English and, where necessary, translated into Iwadja by either an Aboriginal Health Worker or another community member, and then signed/marked by each volunteer.

4.6.2. Anthropometric measurement

Anthropometric measurements made included height, weight, hip, waist and mid-arm circumference. Measurements were recorded on individual data sheets (Addendum 1, A1.6).

Weight was determined using tared Avery beam scales using standard methods to the nearest kilogram. Subjects were weighed following an overnight fast in light clothing without shoes and socks. The accuracy of the scales was checked in the field at each survey using multiples of known weights (500 g margarine containers). The same trained observer measured weight at all surveys.

Height was determined using a fixed Avery stadiometer to the nearest centimetre using standard methods (Truswell, 1981:60). The accuracy of the stadiometer was checked using a fibreglass tape. The same trained observer measured height at all surveys.

Waist, hip and mid-arm circumferences were measured to the nearest centimetre using a fibreglass tape. The 'waist' was identified as being the circumference at the level of the umbilicus which each subject located for the observer. The 'hips' were identified as being the greatest circumference of the hips and included the buttocks within this girth. Mid-arm circumference was measured at the mid point between the tip of the elbow and the head of the humerus.

4.6.3. Blood pressure and pulse

Blood pressure and pulse were measured automatically using a calibrated Dynamap (Criticon Incorporated, Florida, USA) after at least 10 minutes seated rest. The reliability of the Dynamap in Aboriginal community work had been previously established (Mathews *et al*, 1988). In an attempt to control for the marked level of anxiety displayed by some volunteers pending venipuncture, blood pressure and pulse were always taken at least ten

minutes following that procedure. The machine was operated by a trained community member employed specifically to assist with the screening process. Prior to recording, the final readings were checked by either an Aboriginal Health Worker, myself or the Aboriginal research assistant (Ms Annie Bonson). Where cultural beliefs prohibited the measurement of a participant by the operator, the machine was operated by the research assistant or myself. It was originally planned to repeat blood pressure measurement after a five minute interval but, as measurement of blood pressure was the final procedure of each survey, subjects were generally unwilling to remain in the clinic after completion of the initial reading. Therefore only one reading was made for each subject during each survey.

4.6.4. Status of substance use/abuse

The use of cigarettes, alcohol (when accessible) and kava was determined by direct questioning (Addendum 1, A1.6) and confirmed using the method of consensus ranking (Mathews *et al*, 1988). It was also possible to directly observe individual use of these substances while residing in the community.

4.6.5. Collection of blood samples

Blood samples were collected as summarised in Table 4.4. At each survey blood was collected by a certified Aboriginal Health Worker or phlebotomist. In the first screening the District Medical Officer also assisted with venipuncture of the post glucose load sample. In the fourth screening, at the request of the community, only Aboriginal Health Workers and other Minjilang residents worked with the Aboriginal research assistant and myself. In other surveys assistance with collection and handling of blood samples in the field was also provided by a skilled laboratory technician employed by either Menzies School of Health Research or Royal Darwin Hospital.

During the first (June 1989) and last (June 1990) surveys, a total of 20 ml of venous blood was collected from volunteer subjects following an overnight fast of 10 to 12 hours for determination of fasting plasma glucose concentration, fasting serum lipid concentrations, fasting serum fructosamine and insulin concentration, serum and red blood cell folate and thiamine concentration, serum vitamin B₆ and vitamin B₁₂ concentration and serum gamma-glutamyl transferase. After collection of the fasting blood sample subjects drank 75 g glucose monohydrate dissolved in water. A second venous blood sample of 7.5

ml was taken two hours following the administration of the glucose load for measurement of plasma glucose, insulin, ascorbic acid, α -tocopherol, retinol, β -carotene and α -carotene concentration.

During the second (September 1989), third (December 1989) and fourth (March 1990) surveys, only one collection of a total of 22.5 ml of venous blood was made from subjects following an overnight fast of 10 to 12 hours. Fasting serum lipid concentration, serum and red blood cell folate and thiamine concentration, serum gamma-glutamyl transferase, vitamin B₆ and vitamin B₁₂ concentration, plasma ascorbic acid, α -tocopherol, retinol, β -carotene and α -carotene concentration were determined from this sample. In December 1989 serum samples were also analysed for fasting fructosamine concentration.

Table 4.4.a.i.

Collection and handling of blood samples June 1989 and June 1990: Fasting sample (total 25 ml)

Collection	Processing	Storage in the field	Transport to Darwin	Storage in Darwin	Analysed
14 ml (2 x 7 ml) Plain tubes (Yellow top)	Centrifuged				
	4 ml Serum (Lipids,LFT,Ferritin) Plain glass tube (White top)	Chilled(-4°C) overnight	Light aircraft Following day	Nil required	Biochemistry Department Royal Darwin Hospital (RDH)
	1 ml Serum (fructosamine) Plain glass tube (White Top)	Frozen(-30°C)	Light aircraft End of week	Frozen (-70°C)	Department of Human Nutrition Deakin University, Vic.
	2ml Serum (Serum vitamin B) Plain plastic tube (Yellow Top)	Frozen(-30°C)	Light aircraft End of week	Frozen (-30°C) transported ASAP	Biochemistry Department Royal Perth Hospital
1 ml EDTA tube (Pink top)	(Full blood count) Nil Further Handling Required	Chilled(4°C)	Light aircraft Same day:12 noon	Nil required	Haematology Department Royal Darwin Hospital
2.5 ml EDTA tube (Pink top)	(Whole blood B vitamins) Nil Further Handling Required	Frozen(-30°C)	Light aircraft End of week	Frozen (-30°C) transported ASAP	Biochemistry Department Royal Perth Hospital
2.5 ml fluoride (Red top)	Centrifuged (Plasma glucose) (insulin)	Chilled (4°C)	Light aircraft Following day	Nil required	Biochemistry Department, RDH Department of Human Nutrition, Deakin University, Vic.

Table 4.4.a.ii.

Collection and handling of blood samples June 1989 and June 1990: Two hour post glucose load sample (total 7.5 ml)

Collection	Processing	Storage in the field	Transport to Darwin	Storage in Darwin	Analysed
2.5 ml fluoride (Red top)	Centrifuged (Plasma glucose) (insulin)	Chilled(4°C)	Light aircraft Following day	Nil required	Biochemistry Department RDH Department of Human Nutrition Deakin University, Vic.
5 ml Heparin Alfoiled (Orange top)	Centrifuged immediately > 1 ml plasma (Fat soluble vitamins) Alfoiled (Yellow top)	Frozen(-30°C)	Light aircraft End of week	Frozen (-70°C) 6 months	Biochemistry Department RDH/MSHR
	1/2 ml plasma add 1.5 ml cold 6% TCA acid Precipitate/Centrifuge 1 ml supernatant (Vitamin C) (Yellow Top)	Frozen(-30°C) End of week	Light aircraft max 12 months	Frozen (-70°C)	Human Nutrition Unit University of Sydney

Table 4.4. b.

Collection and handling of fasting blood samples (total 22.5 ml) September 1989, December 1989 and March 1990

Collection	Processing	Storage in the field	Transport to Darwin	Storage in Darwin	Analysed
14 ml (2 x 7 ml) Plain tubes (Yellow top)	Centrifuged 4 ml Serum (Lipids, LFT, Ferritin) Plain glass tube (White top) 1 ml Serum (fructosamine) Plain glass tube (White Top) 2ml Serum (Serum vitamins B) Plain plastic tube (Yellow Top)	Chilled(-4°C) overnight Frozen(-30°C) Frozen(-30°C)	Light aircraft Following day Light aircraft End of week Light aircraft End of week	Nil required Frozen (-70°C) Frozen (-30°C) transported ASAP	Biochemistry Department Royal Darwin Hospital (RDH) Department of Human Nutrition, Deakin University, Vic. Biochemistry Department Royal Perth Hospital
1 ml EDTA tube (Pink top)	(Full blood count) Nil Further Handling Required	Chilled (4°C)	Light aircraft Same day: 12 noon	Nil required	Haematology Department Royal Darwin Hospital
2.5 ml EDTA tube (Pink top)	(Whole blood B vitamins) Nil Further Handling Required	Frozen(-30°C)	Light aircraft End of week	Frozen (-30°C) transported ASAP	Biochemistry Department Royal Perth Hospital
5 ml Heparinised tubes Alfoiled (Orange top)	Centrifuged immediately > 1 ml plasma (Fat soluble vitamins) Alfoiled (Yellow top) 1/2 ml plasma add 1.5 ml cold 6% TCA acid Precipitate/Centrifuge 1 ml supernatant (Vitamin C) (Yellow Top)	Frozen(-30°C) Frozen(-30°C) End of week	Light aircraft End of week Light aircraft max 12 months	Frozen (-70°C) 6 months Frozen (-70°C)	Biochemistry Department RDH/MSHR Human Nutrition Unit University of Sydney

4.6.6. Handling of blood samples

Blood samples were handled as summarised in Table 4.4.

Samples to be analysed at Royal Darwin Hospital were flown to Darwin packed in 'eskies' with freezer blocks, collected from the airport by a courier from the Menzies School of Health Research and transferred immediately to the Biochemistry or Haematology department.

Other frozen serum, plasma and whole blood samples were sent packed in dry ice via overnight courier to the relevant southern laboratories.

4.7. Measurement of biological indicators of nutritional status

4.7.1. Lipids

Fasting cholesterol and triglyceride concentrations were measured at the Biochemistry Department of the Royal Darwin Hospital after enzymatic hydrolysis with a commercially available kit (Boehringer Mannheim, FRG) and measured using an automative KONE colorimetric reaction at 500 nm. Serum HDL-cholesterol concentration was measured as for determination of total serum cholesterol concentration following precipitation of chylomicrons, low density lipoprotein and very low density lipoprotein by phosphotungstic acid. The department adheres to strict validity and reliability protocols and participates in national quality assurance programs.

4.7.2. Serum ferritin, albumin, protein and plasma glucose concentrations

Serum ferritin, albumin and protein and plasma glucose concentrations of samples were measured at the Biochemistry Department of the Royal Darwin Hospital.

Serum albumin, protein and glucose concentrations were determined colorimetrically using standardised Kodak Ektachem dry clinical chemistry methods. The intra-assay co-efficient of variation was less than 5% for duplicate analysis.

Serum ferritin concentration was measured by Bioclone IRMA kit.

4.7.3. Serum fructosamine and insulin concentrations

Immunoreactive insulin concentrations in fluoride heparin plasma were measured at the Department of Human Nutrition at Deakin University by Ms Kathy Traianedes with kits purchased from Pharmacia (Uppsala, Sweden) with human insulin standard. The range of the assay was 5-240 m μ /l with an inter-assay coefficient of variation of less than 5%. Concentration of fasting serum fructosamine concentration in samples collected in June 1989, December 1989 and June 1990 were also analysed colorimetrically at the Department of Human Nutrition at Deakin University using kits from Roche Diagnostica (Basel, Switzerland).

4.7.4. Serum and red blood cell folate and thiamine and serum pyridoxal and cobalamin

The vitamins, folate, pyridoxal and thiamine were measured at the Haematology department, Royal Perth Hospital using automated microbiological methods with *Lactobacillus casei* as the test organism for folate and pyridoxal (Davis and Kelly, 1973; Davis et al, 1973, Hunter 1990) and *Lactobacillus fermenti* for thiamine (Davis and Icke, 1983). Cobalamin was measured using *Euglena gracilis* as the test organism (Nicolas and Pitney, 1958). The limits of detection of these methods were 0.5 ug/l for thiamine and folate, 5.98 nmol/l for pyridoxal and 50 ng/l for cobalamin. The test organisms were sensitive to various forms of folate and thiamine present in serum and red blood cells but not to their metabolites. In the assay for thiamine the pyrophosphate was used to prepare the standards. Pyridoxal was dephosphorylated before being assayed using a potato phosphatase. A lyophilised control sample was reconstituted and assayed at ten minute intervals throughout each batch. Mean values of the control sample were calculated and plotted. The co-efficient of variation was less than 5% for duplicate analysis.

All the above B-group vitamins were measured in samples collected during each survey except March 1990 when, due to budgetary constraints, only red blood cell folate concentration was assayed.

4.7.5. Plasma concentration of retinol, α -tocopherol, β -carotene and α -carotene

Plasma concentrations of retinol, α -tocopherol, β -carotene and α -carotene were determined using HPLC methods. The analysis was conducted at the Royal Darwin Hospital on a

Waters Model 600E System Controller with a multisolvent delivery system, a U6K Universal injector, a Model 990 Photodiode Array Detector and dedicated recording software. Methods were established by Mr Malcolm Riley of the Menzies School of Health Research (MSHR). Fat soluble vitamin concentrations were measured in two discrete batches; in November 1989 those samples from the first screening in June 1989 were measured by Mr Malcolm Riley, and in November 1990 samples collected during December 1989 and June 1990 were measured by Ms Kathy Abbott of MSHR (with assistance from myself and staff of the biochemistry department, Royal Darwin Hospital). In order to reduce costs, the samples collected during March 1990, and the remainder of the samples collected during August 1989 were not assayed.

In subdued light 0.5 ml of sample was vortexed with 0.5 ml retinyl acetate internal standard (approximately 1 mg/l in ethanol) and 0.4 ml hexane, then centrifuged at 800 x g (60 secs). 0.25 of the hexane layer was evaporated under nitrogen, and the lipid residue was redissolved in 0.1 ml ethanol. 50 μ l of prepared sample was injected onto the chromatography column (Waters Associates reversed-phase Nova-Pak c18 stainless steel column, 3.9 mm i.d. x 15 cm protected by a Resolve C18 guard column). Peak height of α -tocopherol was measured at 292 nm, retinol and retinyl acetate (internal standard) at 325 nm, and the two carotenes at 460 nm. Approximate retention times were 1.9 minutes for retinol, 2.2 minutes for retinyl acetate, 3.7 minutes for α -tocopherol, 9.8 minutes for α -carotene and 10.5 minutes for β -carotene. Each sample was analysed twice to obtain the mean concentration of each vitamin and pro-vitamin.

Standard curves were produced using spectrophotometrically measured concentrations of standard compounds. Working standards of retinyl acetate were prepared weekly. Replicate pooled plasma samples from volunteers were measured at least twice on each assay day as quality control standards. The within-run variation was 1.7% for retinol, 2.6% for α -tocopherol, 12.1% for α -carotene and 10.6% for β -carotene. Between-run variation was 6.3% for retinol, 4.0% for α -tocopherol, 13.6% for β -carotene and 14.7% for α -carotene.

4.7.8. Ascorbic acid

Ascorbic acid concentrations of supernatant produced from blood collected at June 1989, December 1989 and June 1989, were determined by Mr Zia Ahamad at the Human Nutrition Unit, University of Sydney using HPLC methods. Samples received from

Darwin were stored at -80°C until assayed. Standards were prepared using L-ascorbic acid treated in the same way as samples. Pooled blood was prepared in the same manner as the samples, and stored, thawed and analysed with samples as quality control standards. HPLC was conducted using a LKB 2150 HPLC pump, ETP-Kortek K65B HPLC autoinjector, ETP-Kortek K95 XR UV detector and Shimadzu C-R3A integrator. The HPLC column was C18 Spherisorb ODS 4.6 cm x 25 cm and the mobile phase used was water brought to pH 2.5 by 10% metaphosphoric acid. HPLC conditions included a mobile phase flow rate of 1 ml/minute, a sample volume injected of 25 μl and detection at a wavelength of 245 μm . The area integrated from a single ascorbic acid peak was used to determine the concentration of vitamin C in the sample.

Due to budgetary constraints, the samples collected in August 1989 and March 1990 were not assayed for ascorbic acid.

4.7.9. Intra- and inter-batch variation

In order to investigate the potential effect of intra-batch measurement error, duplicate serum samples from 10 subjects were analysed blindly for fasting serum triglyceride, total serum cholesterol and HDL cholesterol concentration in December 1989. In order to investigate the potential effect of inter-batch measurement error, the analysis of serum samples originally collected from 10 subjects in June 1989 was repeated blindly for fasting serum triglyceride, total serum cholesterol and HDL cholesterol concentration in June 1990. In the interim, samples had been stored frozen at -30°C . In both cases results were compared by calculation of the inter-class correlation coefficient (Bartko, 1966).

To investigate potential inter-batch measurement error of fat soluble vitamins, samples collected during the first survey in June 1990, with sufficient plasma remaining after determination of fat-soluble vitamin concentration in November 1989, were stored at minus 70°C and remeasured in November 1990.

Intra-batch variation in anthropometric measurements was investigated by repeated measurement in 10 subjects in September 1989. The potential effect of observer error on intra- and inter-batch variation was minimised by ensuring that the same observer was responsible for anthropometric measurements over all surveys. Automatic blood pressure measurements were also employed for the same reason.

4.7.10. Confirmation of fasting status

Fasting status was checked in June 1989 and June 1990 by comparison of insulin concentrations in serum collected before and two hours after the 75 g glucose load administered as a glucose tolerance test. Although there was no objective test of fasting status at other surveys, care was taken to collect blood as early as possible in the morning, often from subjects in their homes before they had risen from bed (signed consent having been obtained during the previous day). All subjects were asked if they were fasting prior to the collection of samples. Where available family members also participated as informants. If a subject was not considered to be fasting he/she was given the option of re-presenting the following day, or blood was subsequently collected for only those tests where fasting status was known to be inconsequential.

4.8. Dietary intake

4.8.1. Store-turnover

Apparent community dietary intake was measured every three months from June 1989 by the store-turnover method as previously described (section 3.4.2), in both Minjilang and the control community.

Longitudinal dietary intake was determined retrospectively in three month blocks from store invoices dating from March 1986 using the store-turnover method for both Minjilang and the control community. The relevant invoices were located at both community stores and checked for completeness against copies stored in the archives of the Darwin offices of Arnhem Land Progress Association.

To investigate the nature of dietary intake and change over both the intervention period and the previous four year period, target foods were separated into major categories of interest and the quantity of foods of each category supplied to both Minjilang and the control community store for each three month period from March 1986 was calculated. As accurate retrospective population figures were not available, data prior to June 1989 could not be presented in terms of apparent per capita intake. For comparative purposes results were therefore expressed in terms of the total quantity of foods of each specific category supplied to each community every three months over the five year period.

Food data was analysed for nutrients as previously described (section 3.4.3). Calculations adjusting for the measured proportion of fat weighed in specific meat cuts available in the communities were also entered into the data base. To determine the proportion of visible fat contained in cuts of meat at Minjilang store, two samples of rump steak were randomly selected from the meat freezer at the Minjilang store each week throughout the year, thawed and all visible fat was carefully removed using a scalpel and weighed.

4.8.2. Presentation of results of store-turnover

In an attempt to control for total energy intake and enable comparison of dietary intake between the intervention and control communities, results of nutrient intake were expressed in terms of nutrient density. As reliable, retrospective population data was not available for either community, or for the control community from June 1989, calculation of nutrient density was also necessary in order to compare longitudinal dietary intake. Therefore results were expressed, in the case of macronutrients, in terms of the proportion of total energy intake derived from each nutrient, and, in the case of micronutrients in terms of the amount of each specific nutrient provided per 1000 kJ.

4.8.3. Calculation of recommended nutrient density for Minjilang

The recommended nutrient density for Minjilang was determined by extension of demographic data and recommended nutrient intakes for each major age/sex group derived from current recommended dietary intakes for use in Australia (Truswell *et al*, 1990:13-14,16-17). The mean height of each adult group was determined from anthropometric data. The daily energy requirements of Minjilang adults were estimated to be 1.5 x BMR for both sexes (Truswell *et al*, 1990:15). The demographic breakdown of the Minjilang community was determined by the quarterly household named census.

4.8.4. Use of traditional foods at Minjilang

Qualitative data concerning contemporary use of vegetable and animal foods at Minjilang between June 1989 and June 1990 was obtained by direct observation, reporting of local informants and supplemented where unavoidable by direct questioning. Information concerning the contemporary use of traditional foods by Iwadja groups living at Gurig National Park (Coburg Peninsula) (NT Conservation Commission, 1987:32, 42) was used as a basis to collect information about the knowledge/availability/use of similar foods by

people at Minjilang from key informants (particularly Mrs Janita Mapurduwu, sister of traditional elder, Mr Bill Neidje ('Kakadu Man'), and a respected authority on traditional foods and medicines within the community). Although the numbers of discrete, large animals such as turtle and dugong were tallied during the year, there was no attempt to quantify the limited procurement of vegetable foods for reasons previously outlined (section 1.8.3.1).

The frequency of hunting/gathering activities at Minjilang was determined every fortnight by direct observation. Referring to the list of names used for the fortnightly 'roll call', individuals were classified into five categories of frequency of hunting/gathering activities: nearly every day; at least five times per fortnight; at least twice per fortnight; at least once per fortnight; rarely/never.

4.8.5. Nutrient analysis of traditional foods

Frozen samples of magpie goose, collected in August, turtle flesh and fat and dugong flesh and fat, both collected in November, and barramundi, collected in March 1990, were analysed for fat content and fatty acid composition at the Department of Human Nutrition, Deakin University, Victoria. Samples were immediately frozen (-30°C) on collection, and transferred to Deakin University packed in dry ice via overnight courier.

4.9. Intervention strategies

Intervention strategies were developed with the community on an ongoing basis throughout the project. The strategies were constantly revised and modified according to the results of the three monthly monitoring of both the dietary intake of the community and individual biological indicators of health and nutritional status. Specific strategies devised and applied included:

- 1: Increasing motivation of community members:
 - a) Direction of the project by the community elders
 - b) The development of personal relationships with community members and all family groups
 - c) The identification of both traditional and contemporary beliefs and attitudes related to health and nutrition; subsequent strategies were

designed to reinforce and build on existing food and nutrition knowledge

- d) The employment of an Aboriginal research assistant/nutrition worker
- e) Employment of community members during the screening and monitoring process
- f) Prompt return, in an appropriate form, of the individual results of all biochemical, anthropometric and haematological monitoring, including individual and family group discussion of the significance of all results
- g) Prompt display and discussion of community results following each clinical and dietary survey
- h) Involvement of children through practical nutrition sessions and activities at school
- i) The use of store-turnover data to devise simple, culturally appropriate dietary messages highlighting 'target' foods:
 - i) increase consumption of fruit and vegetables
 - ii) increase consumption of whole grain cereals and bread
 - iii) reduce sugar consumption
 - iv) reduce consumption of sweetened carbonated beverages
 - v) choose lean meat, discarding visible fat
 - vi) eat sandwiches instead of pies

2: Promoting an increased variety of food choice:

- a) The provision of a wide variety of foods in the store, including continuous availability of good quality 'target' foods
- b) Visual display, at the entrance to the store, of the sugar and fat content of the most commonly consumed foods
- c) The use of 'shelf-talkers' to aid recognition of 'target' foods
- d) The preparation and tasting of a wide variety of 'target' foods and suitable recipes in the store
- e) Maintenance of good relationships with the store manager and all employees

Examples of 'feed back' booklets used to return the results of individual biological measurements and the community survey are included at Addendum 3, A3.1.

4.10. Methods of statistical analysis

Biological data were entered onto the UNIX system at Menzies School of Health Research, Darwin, using Oracle software, and analysed using SPSSX-4 and GLIM statistical software.

In statistical analysis of results, the pre-intervention survey (June 1989) was nominated as survey number one. Subsequent surveys, conducted at three month intervals, were numbered sequentially to survey number five (June 1990).

Results were expressed in terms of the mean and standard error of each variable measured at each survey. The difference between the mean of the pre- and post-intervention variables measured in all eligible individuals participating in both relevant surveys were compared by paired t-test using SPSSX-4. Additional analysis considered the change in the mean results over the intervention period according to age and sex groups using SPSSX-4.

Analysis of variance of variables measured in all eligible individuals over the intervention period was also investigated using SPSSX-4. Where necessary, when the distribution was skewed, the analysis of variance was based on the logarithm of the relevant variable.

From a statistical perspective, the more sensitive analysis to detect changes in variables over time first controlled for inter-individual variance; the greater power arising from this analysis being analogous to the increased power apparent in paired t-tests compared with unpaired t-tests. This more sensitive test was applied for key variables using GLIM; analysis of variance tested whether the inter-individual and/or intra-individual variance was greater than the residual variance (which measured the interaction between individual and occasion). However, due to the non-orthogonal (unbalanced) nature of this study, arising from the fact that only a proportion of subjects were seen at all surveys, it was also necessary to ensure that the inter-individual variation was not confounded with the intra-individual variation. Therefore the subset of subjects attending on all occasions (where these effects were orthogonal), was used to investigate the effect of intra- and inter- individual change of major variables of interest over the intervention period. Under these circumstances, subject identification code, when fitted as a 21 level factor, necessarily explained all of the inter-individual variation in the variable under consideration, while survey number, when fitted as a five-level factor, necessarily explained all of the intra-individual variation in the variable under consideration. To

measure the proportion of intra-individual variation that reflected any linear change of the variable under consideration over the intervention period, survey number was fitted, not as a factor, but as an ordinal (linear) variable.

However restriction of analysis to those 21 individuals for whom a complete data set was available severely reduced the total information available. This was particularly so in respect of comparison of biological and store-turnover data. For this purpose, the best estimate of the nutritional status of the whole community at any time included as many individuals as possible at each survey. Moreover there was evidence that the subset was biased compared to the total population. Compared with the community as a whole it contained a greater proportion of women, younger people, diabetics, those who abstained from alcohol, and those who travelled less from the community. Therefore analysis was also conducted for all available data over the five surveys; that is, for all subjects attending at any survey during the intervention period.

Results ultimately revealed that, for variables of major interest, there was no substantial confounding between intra-individual and inter-individual variance when all subjects attending at any survey during the intervention period were considered. Therefore, survey number, when fitted as a five-level factor across all available (non-orthogonal) data, closely approximated the intra-individual variation in the variable under consideration, and subject identification code, when fitted as a 94 level factor across all available (non-orthogonal) data, closely approximated the inter-individual variation in the variable under consideration. An estimation of the proportion of intra-individual variation that reflected any linear change over the period of the intervention, was obtained by fitting survey number, not as a factor, but as an ordinal (linear) variable across all available data.

In order to investigate the determinants of variables of interest, the relationships between biochemical, anthropometric and haematological parameters and other variables including demographic factors and substance use/abuse at each three month period, were investigated by analysis of (approximate) inter-individual variance, (that is, after fitting survey number as both as ordinal (linear) variable and as a five level factor across all available data) using GLIM.

To validate the store-turnover method, the relationship between change in nutrient intake over the intervention period and change in biochemical, anthropometric and haematological indicators of nutritional status was investigated by analysis of

(approximate) intra-individual variance, (that is, after fitting subject identification code as a 94 level factor across all available data) using GLIM¹.

Potential confounding factors such as change in individual status of cigarette smoking, kava and alcohol consumption (as identified in sections 6.6, 6.8 and 6.10) were examined together with dietary intake.

Dietary variables were chosen on a rational basis following consideration of the relevant literature (section 1.7.4). In an attempt to control for confounding due to total energy intake in those models investigating the relationship between vitamin status and vitamin intake, the density of intake of the respective vitamin was usually considered. Actual vitamin intake was employed in the relevant statistical models only where it was shown to be independent of total energy intake. In those models investigating the relationship between change in lipid profile, blood pressure and anthropometric variables during the intervention period and change in dietary intake, the percentage of total dietary energy intake derived from fat was chosen to best approximate the style of diet as measured over the intervention period by the store-turnover method. This variable was chosen as it was highly correlated both with total energy intake ($r=0.92$, $p<0.0001$) and the percentage of total energy derived from saturated fat ($r= 0.96$, $p<0.0001$), and negatively correlated with the density of intake of dietary fibre ($r= -0.55$, $P<0.0001$).

¹ Naturally, subjects attending only once did not contribute any information on the effects of the intervention.

CHAPTER 5: A COMMUNITY-BASED NUTRITION INTERVENTION PROJECT: DIETARY RESULTS AND COMMENTS.

This chapter presents results of the apparent food and nutrient intake at Minjilang and the control community as measured by the store-turnover method. Dietary results are presented for the period of the community-based nutrition intervention project at Minjilang from June 1989 to June 1990 (when store-turnover was determined every quarter for the previous three month period) and for the preceding four years from June 1986 to June 1989 (when store-turnover was determined retrospectively every quarter for the preceding three month period). The qualitative intake of traditional foods at Minjilang from June 1989 to June 1990 is also considered here.

5.1. Population

Prior to being able to calculate the absolute dietary intake (intake expressed as *per capita per day*) using the store-turnover method, it was necessary to determine the size of the population of both Minjilang and the control community. The population at Minjilang over the intervention period was 139 ± 13 (mean \pm sd) when measured by three monthly census, and 154 ± 10 when measured by fortnightly 'roll call' (Addendum 2: Table A2.1a, Table A2.1b). The mean estimated population of the control community was 310 ± 25 over the intervention period (Addendum 2: Table A2.1c).

Dietary data presented in terms of nutrient density were independent of the population denominator.

5.2. Dietary intake: Foods

The total weight of key food items (fruit and vegetables, bread, sugar, beverages, take-away foods and meat) supplied during the previous three month period from June 1986 to June 1990 at both Minjilang and the control community is presented graphically.

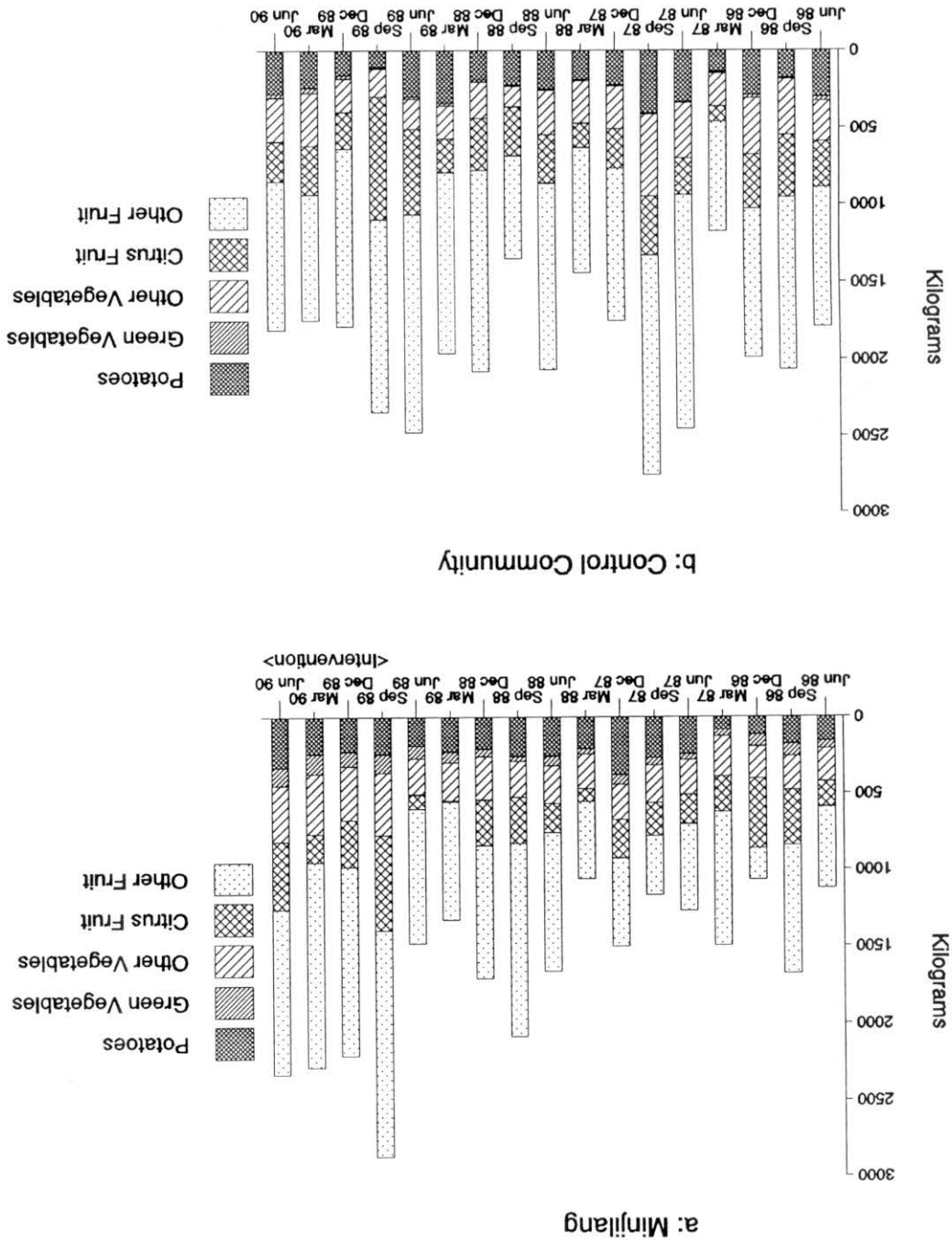
5.2.1. Fruit and Vegetables

Graphical depiction of the total weight of fruit and vegetables supplied during the previous three month period from June 1986 to June 1990 (Figure 5.1) clearly demonstrated the relative increase in fruit and vegetable turnover at Minjilang from June 1989; total fruit and vegetable turnover approximately doubled during the intervention period compared to the previous four years in Minjilang, while remaining relatively constant in the control community throughout the entire five year period.

When the size of the population was taken into account, the intake of fruit and vegetables at Minjilang increased from 83 g per person per day in June 1989 to 183 g per person per day in June 1990; the per capita intake of fruit and vegetables remained similar in the control community at 77 g per person per day in June 1989 and 73 g per person per day in June 1990.

Figure 5.1. The total weight of fruit and vegetables supplied during the previous three months from June 1986 to June 1990

Estimated populations: Mingjiang approximately 150 and Control Community approximately 300



5.2.2. Sugar

Graphical depiction of the total weight of sugar supplied during the previous three month period from June 1986 to June 1990 (Figure 5.2) suggested a trend for declining turnover of sugar at Minjilang over the five year period, even prior to the initiation of the intervention project. The intake of sugar had remained relatively constant at the control community.

When the size of the population was taken into account, the intake of sugar tended to decrease slightly at Minjilang over the intervention project, from 102 g to 89 g per person per day. At the control community the intake of sugar remained relatively high at 162 g per person per day in June 1989 and 188 g per person per day in June 1990.

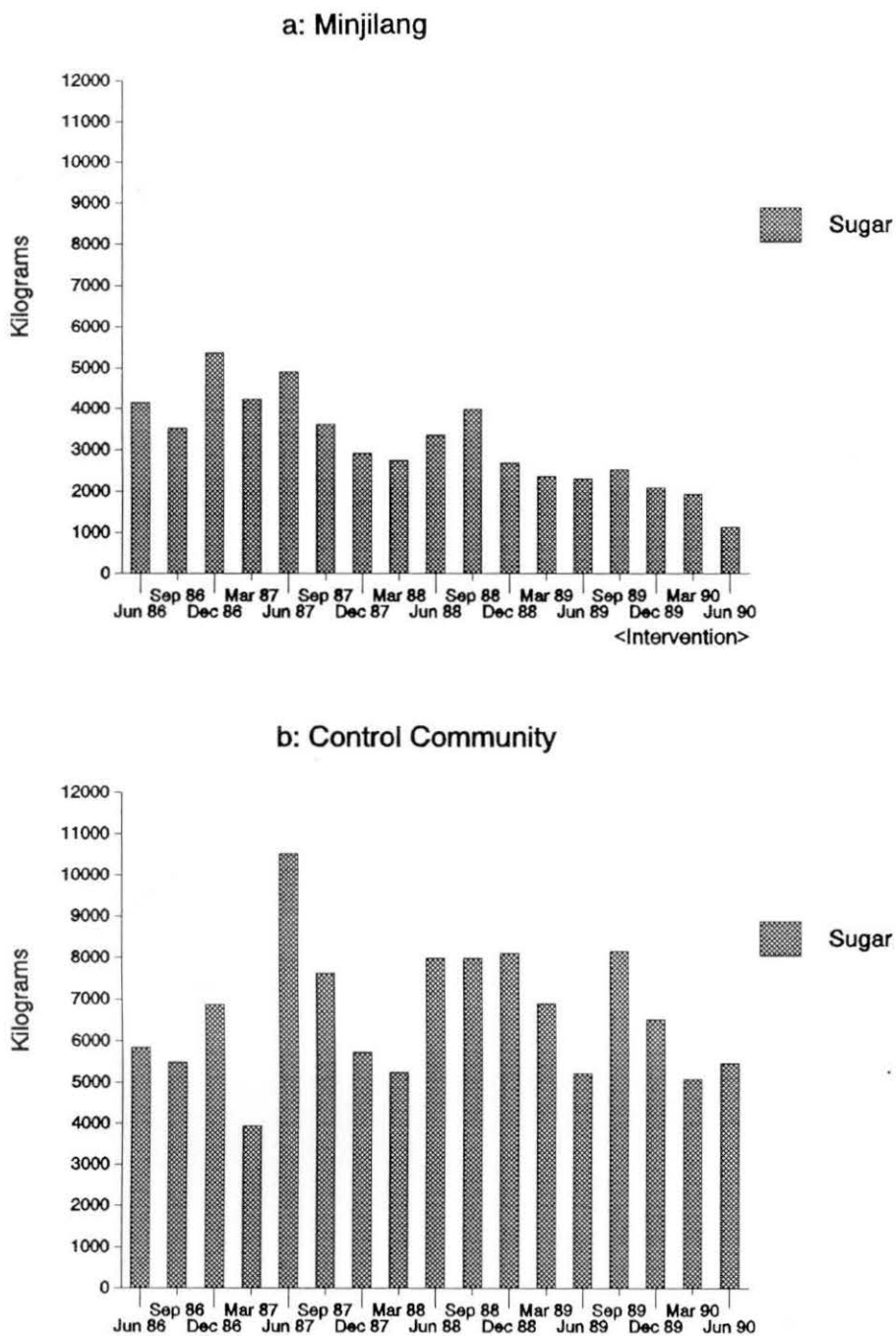


Figure 5.2. The total weight of sugar supplied during the previous three months from June 1986 to June 1990

Guesstimated populations: Minjilang approximately 150 and Control Community approximately 300

5.2.3. Bread

Graphical depiction of the total weight of bread supplied during the previous three month period from June 1986 to June 1990 (Figure 5.3) suggested a trend for bread turnover to increase at Minjilang over the five year period. The intake of bread had remained relatively stable at the control community. There was a tendency for the turnover of bread to increase during the wet season months. The total quantity of bread supplied at Minjilang throughout the intervention period increased from 104 g per person per day in June 1989 to 137 g per person per day in June 1990. Per capita consumption of bread at the control community tended to decrease over the year but was also more variable during this period than at Minjilang.

When the size of the population was taken into account, the intake of both wholemeal and mixed grain breads had increased from 14 g to 33 g per person per day at Minjilang, that is, to over 20% of the total bread turnover. At the control community the proportion of wholemeal bread was consistently less than 8% throughout this period.

At the time of the study, flour used for bread baking in Australia was not fortified with thiamine.

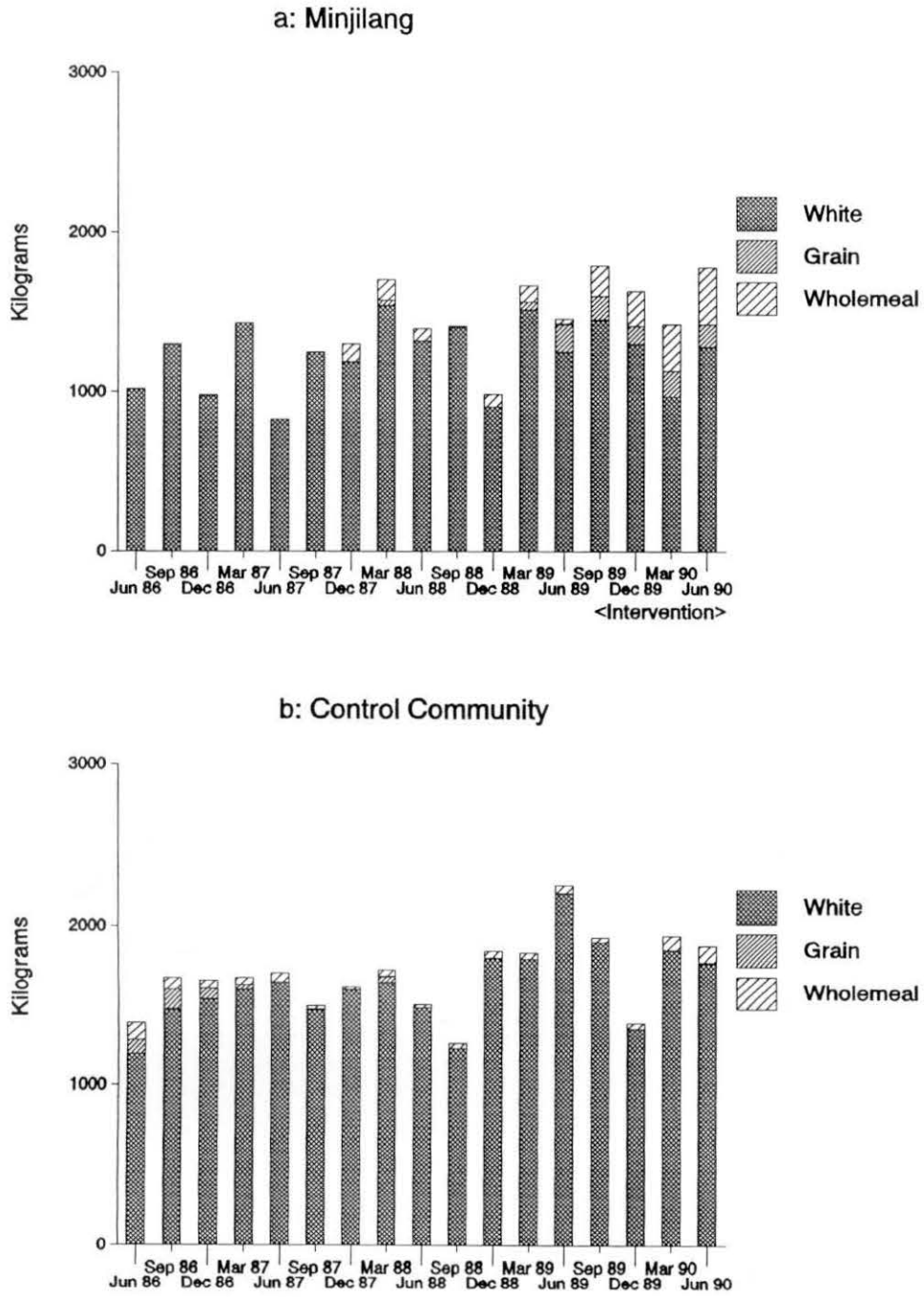


Figure 5.3. The total weight of bread supplied during the previous three months from June 1986 to June 1990

Guesstimated populations: Minjilang approximately 150 and Control Community approximately 300

5.2.4. Flour

The turnover of flour was relatively constant at both communities from June 1986 to June 1990 (Figure 5.4). The turnover of wholemeal flour was negligible. There appeared to be a small seasonal effect, with the turnover of flour being relatively higher at both communities during the dry season months. The total turnover of flour appeared to decrease slightly at both communities over the intervention year. When the size of the population was taken into account, the mean per capita turnover of flour remained similar at both communities.

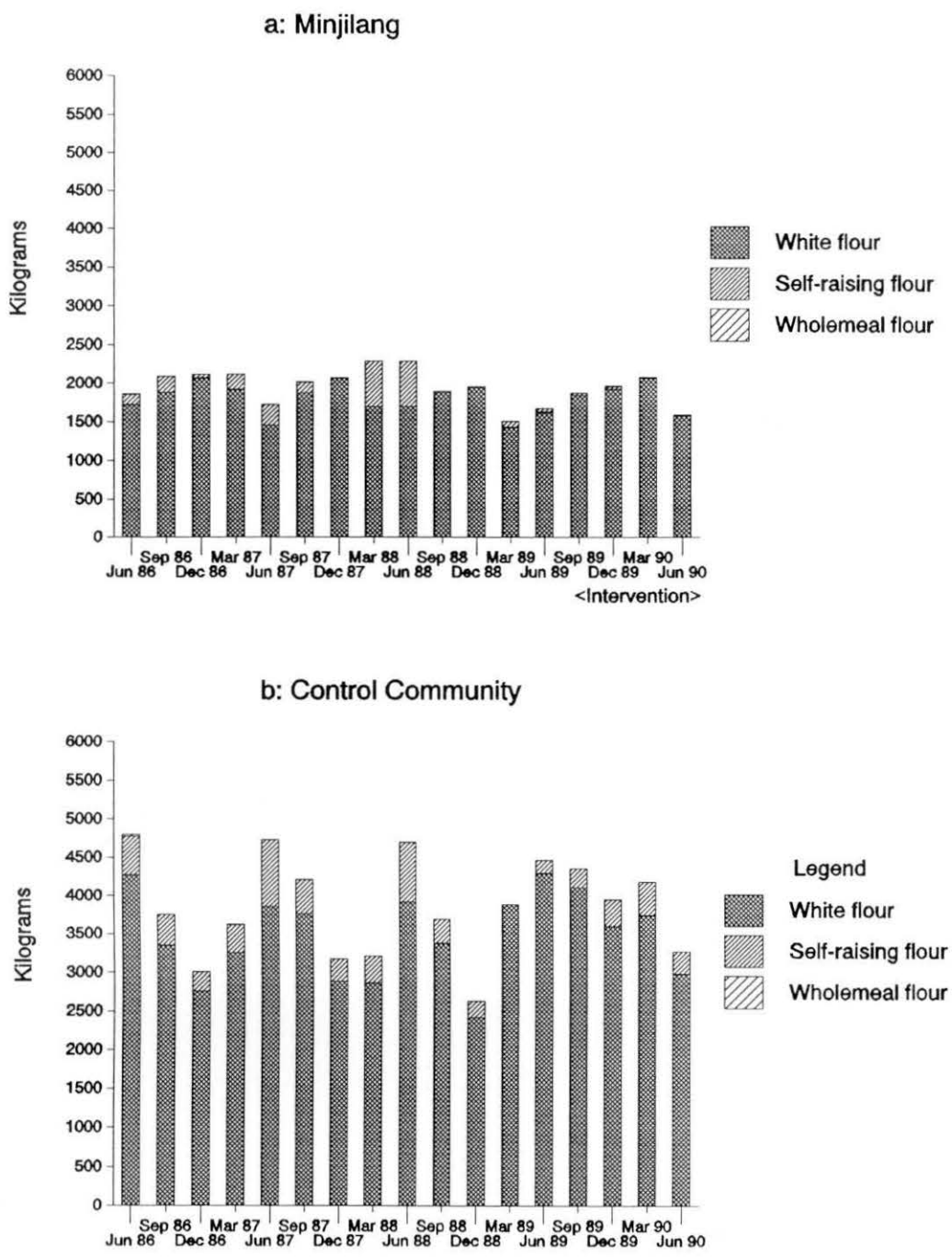


Figure 5.4. The total weight of flour supplied during the previous three months from June 1986 to June 1990

Guesstimated populations: Minjilang approximately 150 and Control Community approximately 300

5.2.5. 'Take-away' foods

Graphical depiction of the total weight of 'take-away' foods supplied during the previous three month period from June 1986 to June 1990 (Figure 5.5) suggested a seasonal effect, with increased turnover, particularly of hot meat pies, during the cooler months of the year. There was a clear demonstration of the relative decrease in 'take-away' foods at Minjilang from June 1989.

When the size of the population was taken into account, the intake of 'take-away' foods decreased at Minjilang from 116 g per person per day in June 1989 to 40 g per person per day in June 1990, but did remain higher than at the control community where the per capita intake of take-away foods reduced from 49 g per person per day in June 1989 to 29 g per person per day in June 1990.

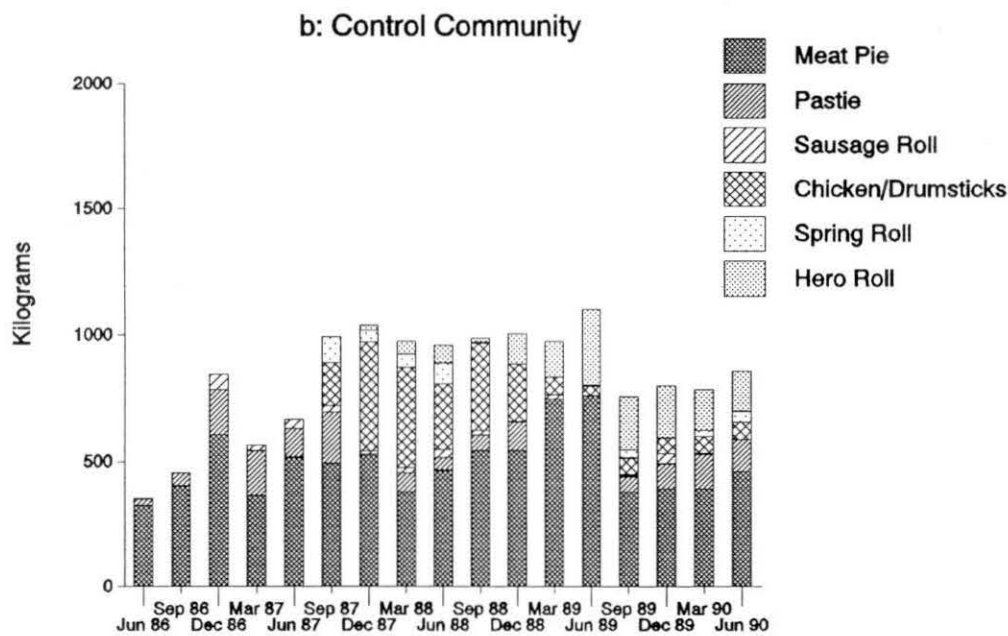
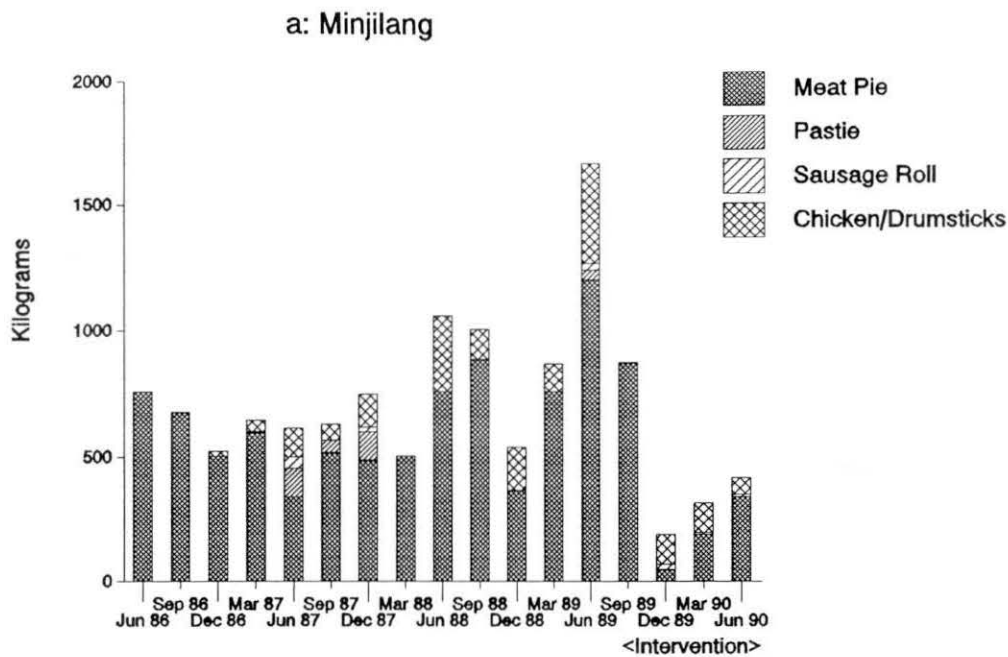


Figure 5.5. The total weight of 'take-away' foods supplied during the previous three months from June 1986 to June 1990

Guesstimated populations: Minjilang approximately 150 and Control Community approximately 300

5.2.6. Meats

Graphical depiction of the total turnover of fresh meat suggested that there was a tendency for the turnover of fresh meat to increase gradually in both communities from June 1986 to June 1990 (Figure 5.6). There appeared to be a seasonal effect, with the turnover of fresh meats being relatively higher at both communities during the wet season months.

During the intervention period the total turnover of fresh meats remained relatively stable at both communities. Although the turnover of chicken was similar in both communities, the total turnover of fresh meats was consistently much higher at the control community than at Minjilang. When the size of the population was taken into account, the mean per capita turnover of fresh meat was invariably twice as high at the control community than at Minjilang.

The proportion of fat found in the cuts of meat supplied to the Minjilang store reduced by 37% over the intervention period (Table 5.1). Although the store manager, on behalf of the community, constantly requested lean cuts of meat from the wholesale butchers, the most dramatic reduction occurred after the supplier was changed in early December 1989.

Table 5.1. Percentage visible fat of total meat in randomly selected cuts of rump steak supplied to Minjilang store (mean±sd)

Period	June 1989	September 1989	December 1989	March 1990	June 1990
n	24	24	24	24	24
% fat	35.4±3.5	33.1±3.3	26.5±3.1	24.7±3.1	22.4±2.8

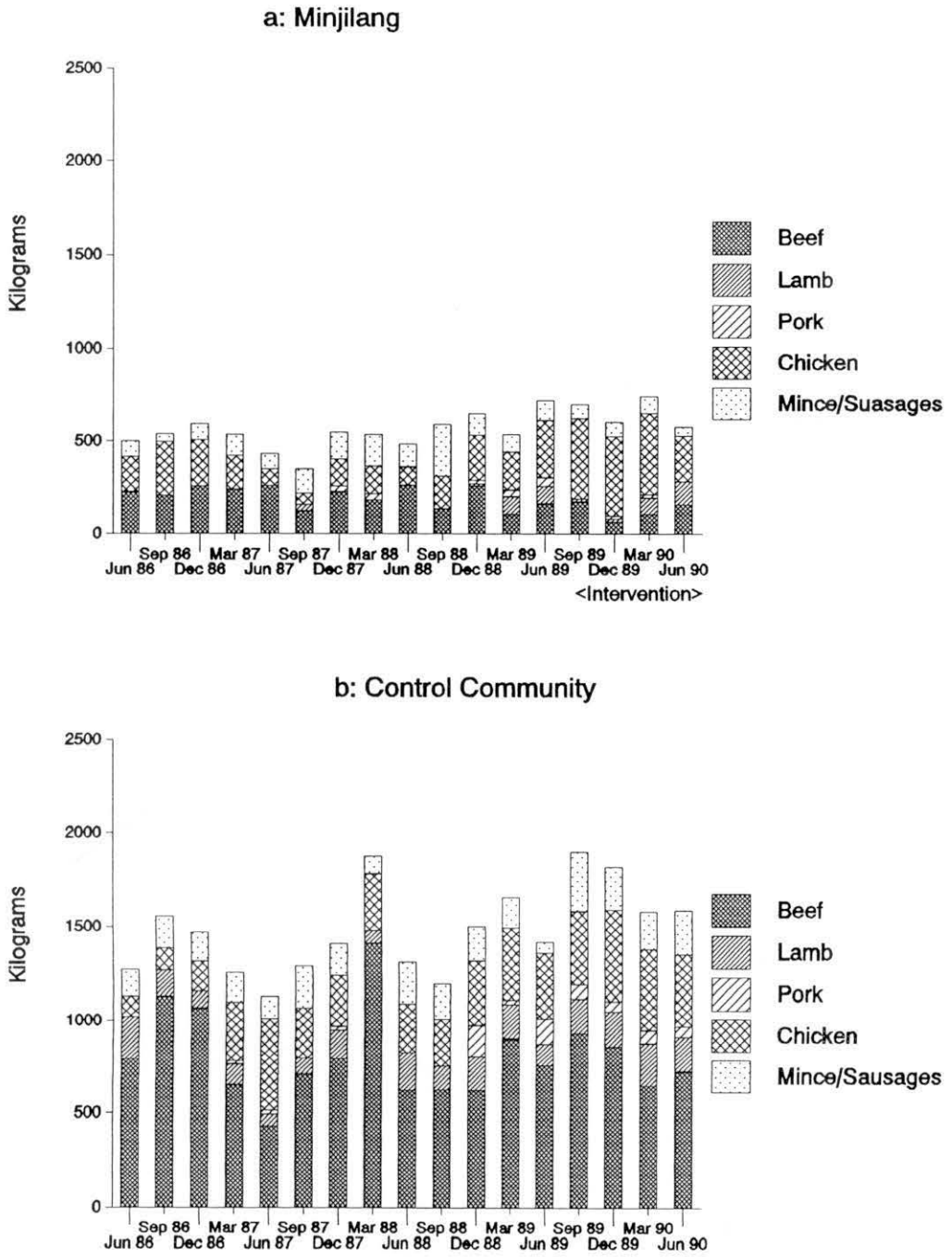


Figure 5.6. The total weight of meats supplied during the previous three months from June 1986 to June 1990

Guestimated populations: Minjilang approximately 150 and Control Community approximately 300

5.2.7. Store-purchased beverages

Graphical depiction of the total volume of beverages supplied during the previous three month period from June 1986 to June 1990 (Figure 5.7) suggested a seasonal pattern, particularly at Minjilang; the turnover of beverages tended to increase during the warmest and most humid months of the year (September to May), and decrease during the cooler dry season months (June, July and August). The total quantity of beverages supplied at Minjilang pre- and post-intervention remained relatively unchanged. However there was an increased proportion of unsweetened 'diet' beverages to approximately 12% and fruit juices to 17% of the total beverages supplied. In September, October and November 1989, 'diet' beverages comprised over 30% of the total beverage turnover. At the control community the proportion of 'diet' beverages and fruit juice was consistently less than 2% and 6% of total beverage turnover respectively throughout this period.

When the size of the population was taken into account, per capita consumption of store-purchased beverages was comparable in both communities for similar seasons; the total consumption of store-purchased beverages at Minjilang was 275 ml per person per day in June 1989 and 280 ml per person per day in June 1990 and at the control community total beverage intake was 319 ml per person per day in June 1989 and 283 ml per person per day in June 1990.

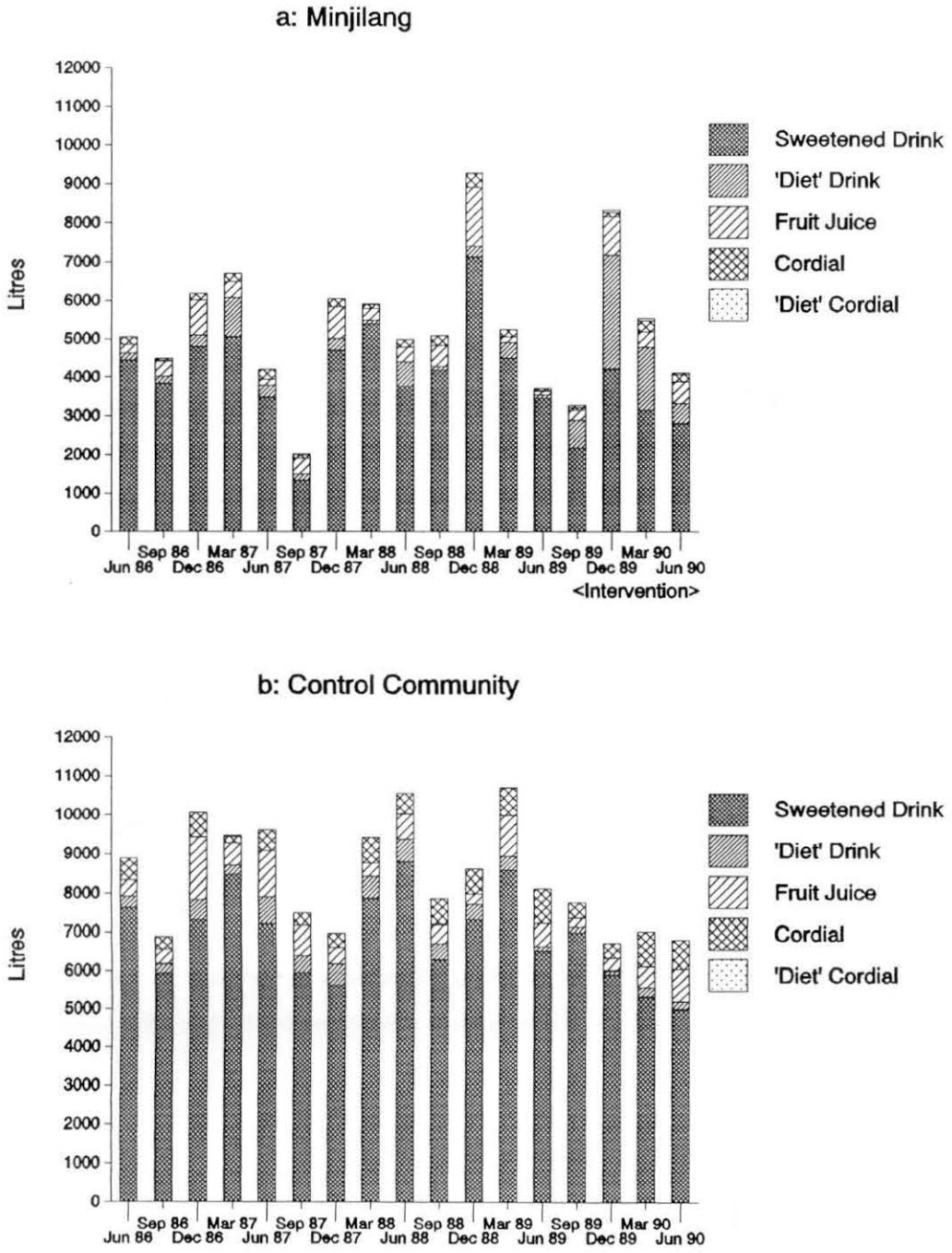


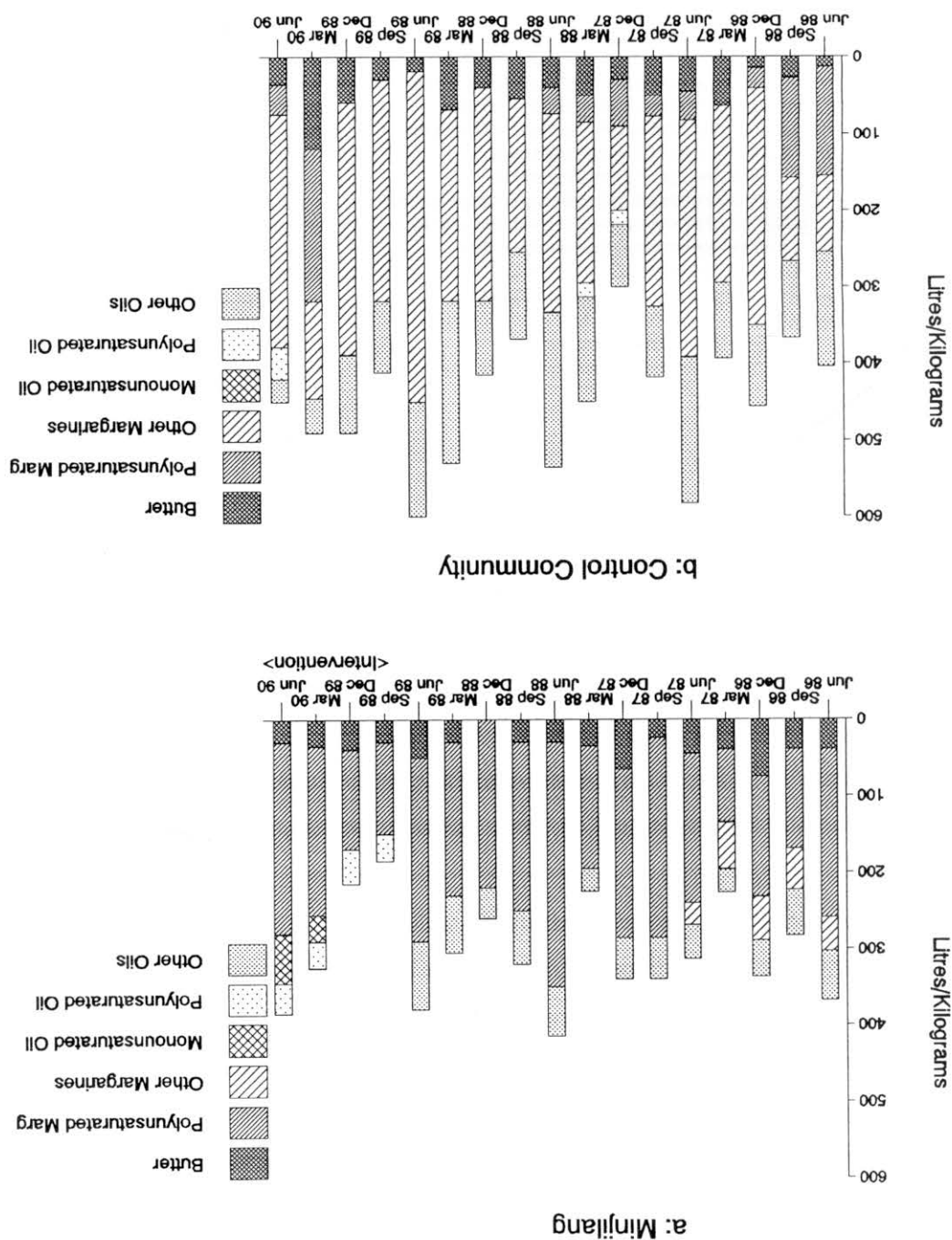
Figure 5.7. The total volume of beverages supplied during the previous three months from June 1986 to June 1990

Estimated populations: Minjilang approximately 150 and Control Community approximately 300

Graphical depiction of the total weight/volume of fats and oils supplied during the previous three month period from June 1986 to June 1990 (Figure 5.8) again suggested a seasonal pattern; the turnover of fats and oils tended to increase during March, April and May and decrease during the warmest and most humid months of the year (September to May). There were some noticeable differences in the types of fats provided at the two communities; polyunsaturated margarine appeared to be popular at Minjilang throughout the entire five year period, whereas non-polyunsaturated margarine had been more consistently available at the control community. The total quantity of fats and oils supplied at Minjilang pre- and post-intervention remained relatively unchanged, although monounsaturated and polyunsaturated vegetable oils had completely replaced the original cooking oils by June 1990. The turnover of polyunsaturated margarine was similar at comparable seasons throughout this period. At the control community polyunsaturated margarine was re-introduced in January 1990, although butter and non-polyunsaturated margarine remained popular; there was little change in the total turnover of fats and oils throughout the year.

When the size of the population was taken into account, per capita consumption of total fats and oils was comparable in both communities for similar seasons; the total intake of fats and oils at Minjilang was approximately 25 ml per person per day in both June 1989 and June 1990 and at the control community total fat and oil intake was 21 ml per person per day in June 1989 and 18 ml per person per day in June 1990.

Figure 5.8. The total weight of fats and oils supplied during the previous three months from June 1986 to June 1990. Guesstimated populations: Minjilang approximately 150 and Control Community approximately 300



5.3. Dietary intake: Nutrients

The results of nutrient analysis of store-turnover measured every three months from June 1989 to June 1990 revealed a marked improvement in nutrient density in Minjilang over the intervention period (Table 5.2a). In comparison the nutrient intake at the control community remained relatively stable during this period (Table 5.2b). There was also a reduction in the apparent per capita energy intake at Minjilang over the intervention year; this was not observed at the control community.

From a seasonal perspective, results of the application of the store-turnover method at Minjilang in June 1990 were directly comparable to the results in June 1989 (Figure 5.9). Results indicated a relative 30% decrease in the percentage of total energy intake derived from saturated fat, a slight decrease in the proportion of total energy derived from sugars, a relative 20% increase in density of dietary fibre and marked relative increases in the density of intake of most vitamins and minerals over the intervention period, including ascorbic acid (135%), folate (60%), β -carotene (40%), thiamine (50%) and calcium (60%).

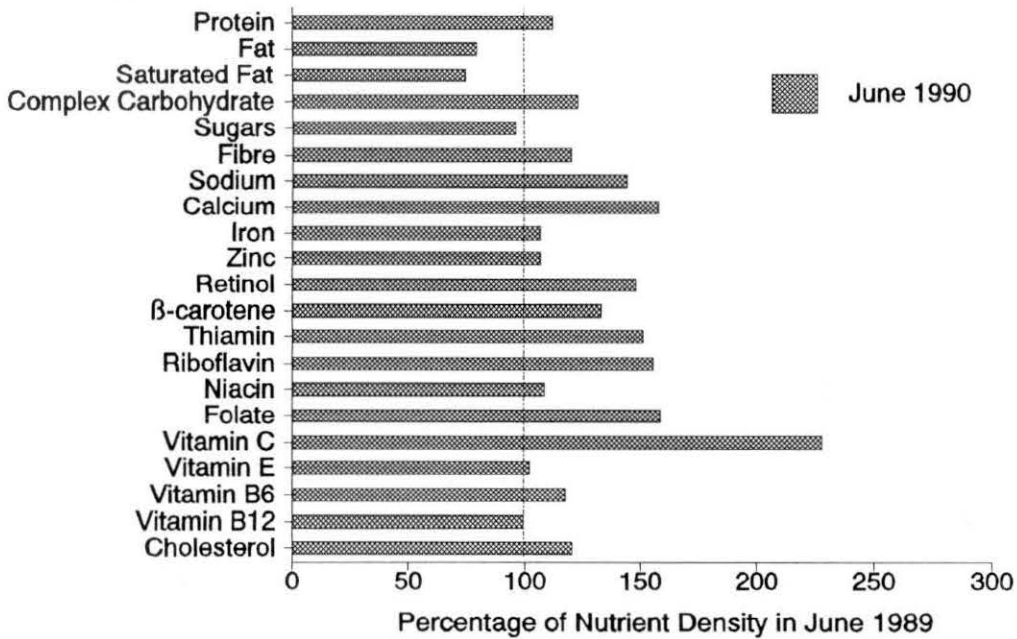


Figure 5.9. Change in nutrient density over the intervention period, Minjilang

Nutrient analysis of longitudinal store-turnover data further supported the relative seasonal

change in nutrient density in Minjilang over the intervention period, both compared to the preceding four years and to the control community throughout the entire five year period (Figure 5.10 to Figure 5.32).

Recommended nutrient densities calculated for the Minjilang community as a whole are included in the relevant sections below and are also presented in tabular form (Addendum 2: Table A2.2b).

Table 5.2a. Dietary intake as measured by the store-turnover method (per capita per day): Minjilang

Period	June 1989	Sept 1989	Dec 1989	March 1989	June 1990
Energy (kJ)	12408	11387	10875	11321	10963
Energy (kCal)	2963	2706	2575	2687	2626
Protein (g)	74.0	80.8	71.0	74.9	78.3
% Energy protein (%)	10.2	11.5	11.1	11.3	11.4
Fat (g)	110	83.3	73.5	73.3	78.9
Saturated fat (g)	45	32	31	28	30
Monounsaturated fat (g)	34	29	25	23	26
Polyunsaturated fat (g)	21	14	12	14	16
% Energy fat (%)	34	28	26	25	27
P:S ratio	0.47	0.45	0.38	0.49	0.54
Total carbohydrate (g)	445	438	434	460	428
Complex carbohydrate (g)	212	204	200	225	229
Sugars (g)	233	235	234	235	199
% Energy carbohydrate (%)	56	61	63	64	61
% Energy complex carbohydrate (%)	27	28	29	31	33
% Energy sugars (%)	30	33	34	33	29
Calcium (mg)	677	790	776	850	945
Iron (mg)	11	10	8.9	9.5	11
Density iron (mg/1000 kJ)	0.86	0.89	0.82	0.84	0.97
Zinc (mg)	9.1	9.3	8.6	8.8	8.6
Vitamin A eq. (μ g)	435	408	450	544	551
Retinol (μ g)	296	235	290	324	388
Carotene (μ g)	829	1037	959	1325	976
Density vitamin A eq. (μ g/1000kJ)	35.0	35.8	41.7	48.1	50.2
Density retinol (μ g/1000kJ)	23.9	20.6	26.7	28.6	35.4
Density β -carotene (μ g/1000kJ)	66.8	91.1	88.2	117.0	89.0
Folate (μ g)	131	176	173	170	183
Density folate (μ g/1000kJ)	10.5	15.5	16.0	15.0	16.7
Thiamine (mg)	0.97	1.1	1.2	1.4	1.3
Density thiamine (mg/1000kJ)	0.08	0.10	0.11	0.12	0.12
Riboflavin (mg)	1.3	1.5	1.7	1.7	1.8
Niacin (mg)	14.7	16.8	14.0	15.1	14.1
Vitamin C (mg)	29.6	52.6	58.5	42.0	59.7
Density vitamin C (mg/1000kJ)	2.39	4.62	5.38	3.71	5.45
Vitamin E (mg)	6.1	5.1	4.4	4.6	5.5
Density vitamin E (mg/1000kJ)	0.49	0.45	0.40	0.41	0.50
Vitamin B ₆ (mg)	1.2	1.2	1.2	1.2	1.2
Density vitamin B ₆ (mg/1000kJ)	0.10	0.11	0.11	0.10	0.11
Vitamin B ₁₂ (μ g)	3.0	2.2	1.8	2.1	2.6
Density vitamin B ₁₂ (μ g/1000kJ)	0.24	0.19	0.17	0.18	0.24
Sodium (mg)	3564	3684	3219	3521	4546
Cholesterol (mg)	223	233	171	149	238
Dietary fibre (g)	16	16	14	17	17
Density fibre (g/1000kJ)	1.29	1.41	1.31	1.50	1.55

Table 5.2b. Dietary intake as measured by the store-turnover method (per capita per day): Control Community

Period	June 1989	Sept 1989	Dec 1989	March 1989	June 1990
Energy (kJ)	12169	12953	12117	12581	12358
Energy (kCal)	2892	3066	2872	2978	2925
Protein (g)	75.6	67.9	67.2	68.7	62.2
% Energy protein (%)	10.5	8.8	9.3	9.2	8.5
Fat (g)	76.8	72.8	74.5	71.6	74.6
Saturated fat (g)	37	35	36	33	33
Monounsaturated fat(g)	28	27	27	27	27
Polyunsaturated fat(g)	12	9.8	11	11	13
% Energy fat (%)	24	21	23	22	23
P:S ratio	0.33	0.28	0.30	0.33	0.39
Total carbohydrate (g)	506	571	515	549	534
Complex carbohydrate (g)	236	190	184	220	180
Sugars (g)	270	381	331	329	354
% Energy carbohydrate (%)	66	70	67	69	69
% Energy complex carbohydrate (%)	31	23	24	28	23
% Energy sugars (%)	35	47	43	41	45
Calcium (mg)	936	774	800	658	767
Iron (mg)	11	9.6	9.0	9.6	9.0
Density iron (mg/1000 kJ)	0.88	0.74	0.74	0.76	0.73
Zinc (mg)	9.2	8.3	8.6	8.1	7.7
Folate (μ g)	124	104	96.4	105	102
Density folate (μ g/1000kJ)	10.2	8.1	8.0	8.3	8.3
Vitamin A eq. (μ g)	486	362	431	365	427
Retinol (μ g)	331	258	305	258	321
Carotene (μ g)	933	627	755	642	640
Density vitamin A eq. (μ g/1000kJ)	39.9	28.0	35.5	29.0	34.6
Density retinol (μ g/1000kJ)	27.2	19.9	25.1	20.5	25.9
Density β -carotene (μ g/1000kJ)	76.6	48.4	62.3	51.0	51.8
Thiamine (mg)	1.0	0.90	0.87	0.86	0.85
Density thiamine (mg/1000kJ)	0.086	0.070	0.072	0.068	0.069
Riboflavin (mg)	1.5	1.3	1.3	1.1	1.3
Niacin (mg)	12.3	11.2	11.0	11.4	10.5
Vitamin C (mg)	30.2	21.4	14.5	24.4	35.0
Density vitamin C (mg/1000kJ)	2.48	1.65	1.20	1.94	2.83
Vitamin E (mg)	3.3	3.1	3.3	3.5	4.2
Density vitamin E (mg/1000kJ)	0.27	0.24	0.27	0.28	0.34
Vitamin B ₆ (mg)	0.92	0.79	0.79	0.88	0.77
Density vitamin B ₆ (mg/1000kJ)	0.076	0.061	0.065	0.070	0.062
Vitamin B ₁₂ (μ g)	3.1	2.5	2.5	2.0	2.5
Density vitamin B ₁₂ (μ g/1000kJ)	0.258	0.196	0.206	0.161	0.202
Cholesterol (mg)	207	226	220	202	202
Sodium (mg)	3735	2957	2824	3824	2955
Dietary fibre (g)	13	11	12	12	11
Density fibre (g/1000 kJ)	1.08	0.85	0.97	0.92	0.86

5.3.1. Longitudinal macronutrient profile

At Minjilang, retrospective analysis revealed that from June 1987 until the initiation of the intervention project, there had already been a 37% decrease in the percentage of energy derived from sugars (Figure 5.10). Most of the decrease over this period could be explained by the reduction in intake of sugar *per se* (Figure 5.2). However there was also potential confounding due to an increase in the corresponding proportional dietary intake of fat from June 1988 to June 1989 (Figure 5.11). The proportion of energy derived from protein gradually increased from March 1986 to June 1989, however there was little increase in the absolute intake of protein during this period until June 1988 (Figure 5.12). Conversely, the percentage of energy derived from complex carbohydrate remained relatively stable over this period (Figure 5.13).

In contrast to changes in the style of diet apparent at Minjilang prior to June 1989, the proportion of energy derived from all macronutrients remained relatively stable at the control community.

Although the percentage of energy derived from sugars was consistently higher at the control community, there was a slight decrease over the five year period, with levels being particularly low during the three month pre-intervention period. There was evidence of a seasonal effect on the proportion of energy derived from sugars at both communities, with higher levels during the warmest and most humid months of the year, corresponding to an increased turnover of sweetened, chilled beverages.

In addition to the unusually low percentage of energy derived from sugar at the control community in June 1989, there was also a correspondingly high percentage of energy derived from complex carbohydrate; the total contribution of carbohydrate to the energy intake remained relatively constant. There was no suggestion of a seasonal effect on the proportion of energy derived from complex carbohydrate at either community.

Although similar prior to June 1988, retrospective analysis of longitudinal store-turnover in both communities, revealed that the percentage of energy from fat increased at Minjilang relative to the control community from that time (Figure 5.11). This increase was associated with both the decreasing percentage of energy derived from sugar and the increase in the absolute intake of fat from June 1988 at Minjilang. From June 1988 to the pre-intervention high (33.5%) at June 1989, the percentage of energy derived from fat

increased at Minjilang, and was 40% higher than that at the control community. There was evidence of a relative seasonal increase in the proportion of energy intake derived from total fat during the cooler months of the year (from June to August) in both communities.

The pattern of energy contribution from total fat in both communities throughout the four year pre-intervention period was paralleled by that of saturated fat (Figure 5.14). However the percentage of energy derived from saturated fat at the control community also tended to rise gradually over this time. The percentage of energy derived from saturated fat at Minjilang in June 1989 was 15% higher than the rate at the control community.

The ratio of polyunsaturated fatty acids to saturated fatty acids (P:S ratio) had varied at Minjilang prior to the intervention (Figure 5.15). It decreased by 34% from June 1986 to September 1987, then increased by 65% over the following year when the only spread available at the store was polyunsaturated margarine. Changes in the P:S ratio at both communities also appeared to be related to the introduction of specialised store equipment, such as bain-maries (section 7.2.2.3). With the introduction of a 'take-away' section in the Minjilang store in May 1988 (new pie-warmer and rotisserie) and the increased turnover of meat and meat products from June 1988, there was another relative decrease in the P:S ratio until the commencement of the intervention project. The P:S ratio at the control community was usually lower than at Minjilang, particularly following the installation of a bain-marie for display of deep fried foods at the community store in July 1987. Butter (a rich source of saturated fatty acids) had also been more regularly available/popular at the control community than at Minjilang (Figure 5.8).

The percentage of energy derived from protein was higher at the control community until June 1988. The proportion rose at Minjilang due to a relative increase in the absolute intake of protein, (predominantly from the increased turnover of chicken and meat/meat products) (Figure 5.6), but also due to the reducing percentage of energy derived from sugars. At June 1989 the percentage of energy derived from protein at both communities was high relative to the levels of the previous year (Figure 5.12).

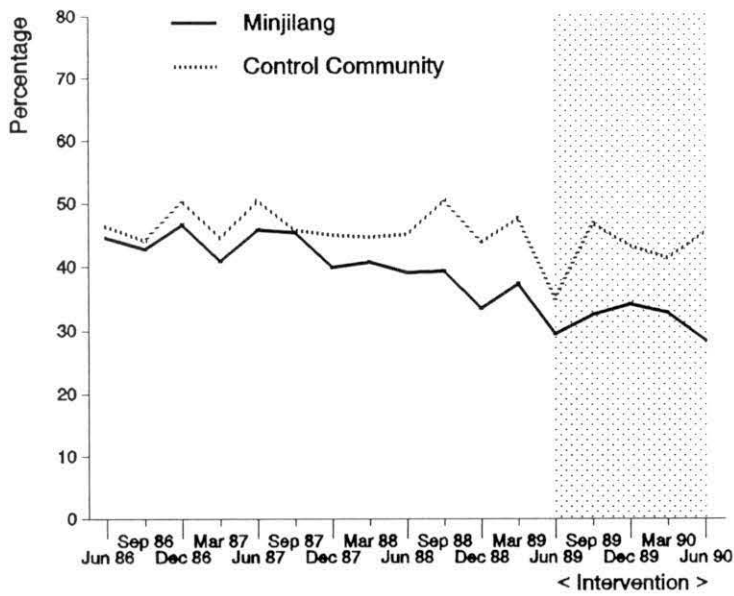


Figure 5.10. Percentage of total energy derived from sugars during the previous three month period from June 1986 to June 1990

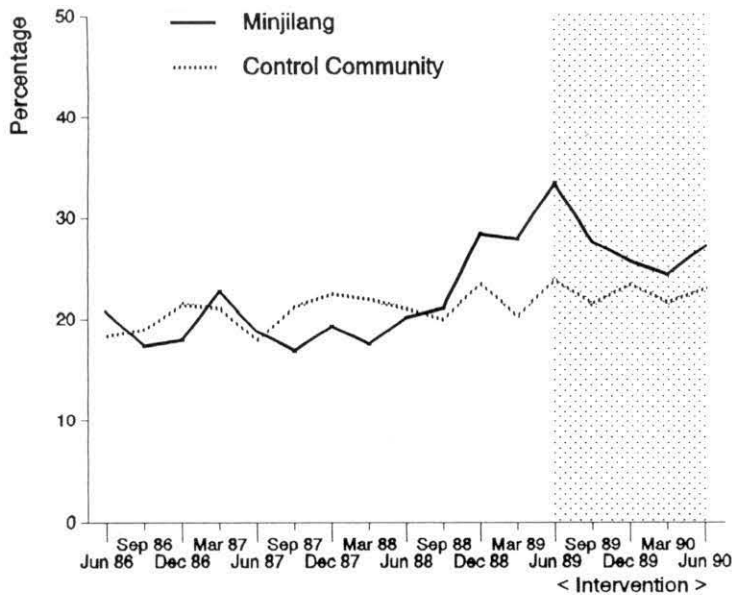


Figure 5.11. Percentage of total energy derived from fat during the previous three month period from June 1986 to June 1990

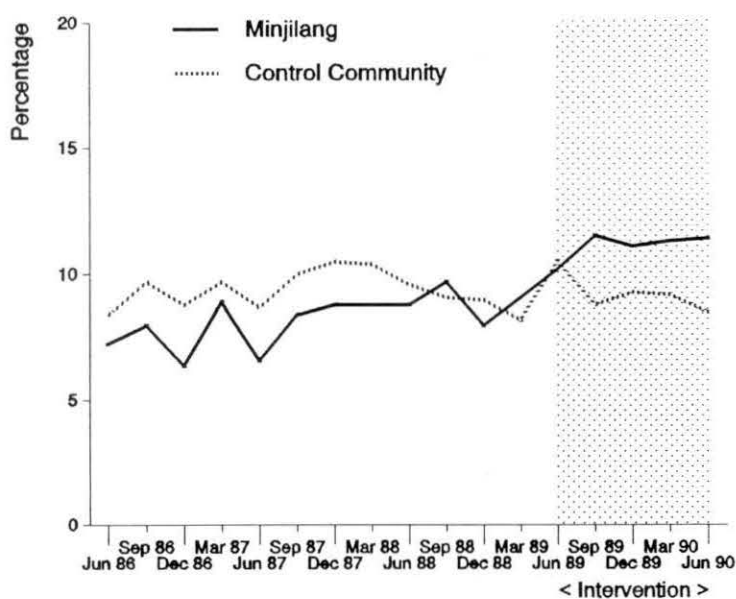


Figure 5.12. Percentage of total energy derived from protein during the previous three month period from June 1986 to June 1990

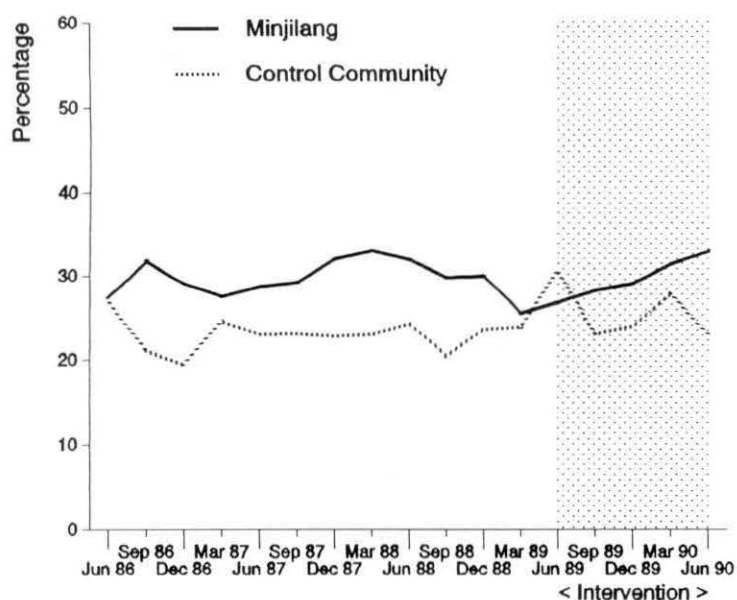


Figure 5.13. Percentage of total energy intake derived from complex carbohydrate during the previous three month period from June 1986 to June 1990

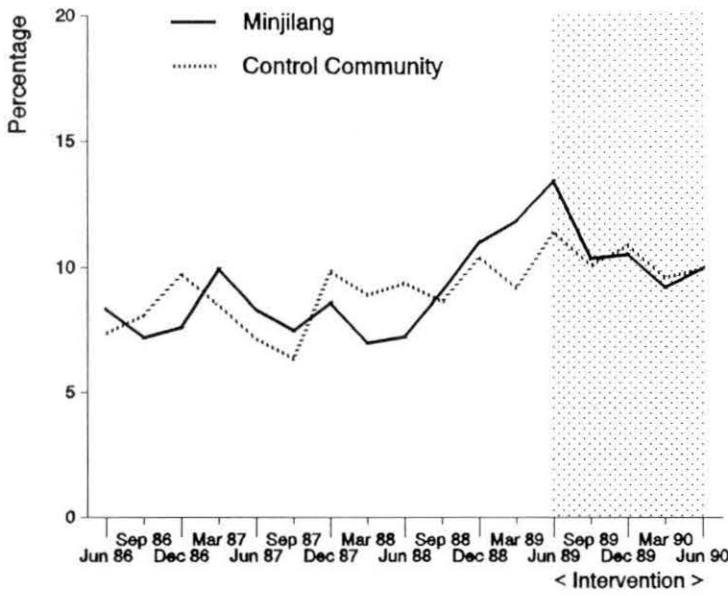


Figure 5.14. Percentage of total energy derived from saturated fat during the previous three month period from June 1986 to June 1990

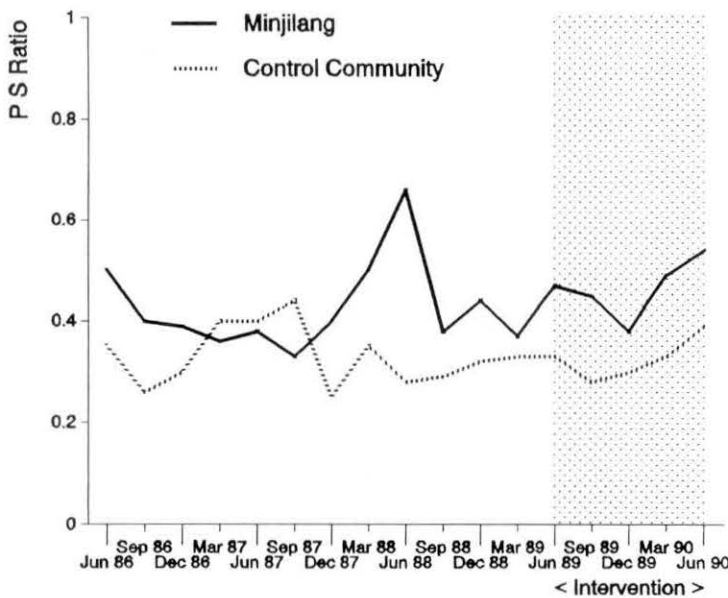


Figure 5.15. The ratio of polyunsaturated to saturated fatty acids during the previous three month period from June 1986 to June 1990

5.3.2. Change in macronutrient profile during the intervention period

In June 1989 prior to initiation of the intervention project at Minjilang, the macronutrient profile of both communities was comparable to other Aboriginal communities in that intake of energy, sugars and saturated fat were excessive. However the diet at Minjilang in June 1989 was relatively lower in sugars and higher in fats than the control community and tended more towards the style of diet of wider (non-Aboriginal) Australia (section 3.5.2).

Investigation of the types of foods¹ contributing to total energy intake (Table 5.3a), disclosed the proportional increase in cereal and cereal products, fruit and vegetables, milk and milk products, and the proportional decrease in snack foods and take-away foods at Minjilang over the intervention period.

In both June 1989 and June 1990 at the control community 18 types of foods each provided more than two percent of the total energy intake and sugar, meat, flour and bread together contributed over 50% of the total energy intake. These results were similar to those described by store-turnover data in other Aboriginal communities (section 3.5.2). However at Minjilang in June 1989, 20 types of foods each provided more than two percent of the total energy intake, and 'only' 38.4% of the total energy intake was derived from those four foods (sugar (14.2%), fresh meat (4.6%), flour (11.2%) and bread (8.8%)); manufactured meat products provided 15.1% of the total energy intake at Minjilang in June 1989. After the intervention project at Minjilang, 23 types of food provided more than two percent of the total energy intake; 45.3% of the total energy intake was derived from different proportions of the same four foods (sugar (13.0%), fresh meat (4.5%), flour (14.8%) and bread (13.1%)), and 6.7% of total energy intake was derived from manufactured meat products.

During the intervention period at Minjilang the percentage of energy intake derived from sugars rose slightly during the first nine months before reducing to a level slightly lower than the pre-intervention measure (Figure 5.10). Conversely the percentage of energy derived from sugars appeared to increase over the intervention period at the control community. During the intervention period at Minjilang the proportion of sugars derived

¹ Food categories were based on groupings applied in analysis of the national dietary survey of adults, 1983 (CDCSH, 1986)

from fruit and vegetables almost tripled while the proportion of sugars derived from sweetened carbonated beverages decreased by more than 50% (Table 5.3b).

During the intervention period there was a continuous increase in the proportion of energy intake derived from complex carbohydrate at Minjilang (Figure 5.13).

During the intervention period the percentage of total energy intake derived from fat decreased by almost 30% in the first nine months at Minjilang, and approached the relatively constant level at the control community (Figure 5.11). Although the absolute intake of fat continued to decline during the last three months of intervention, the proportion of energy intake derived from fat increased due to the decrease in both the absolute and proportional intake of sugar during those months. The proportion of energy derived from saturated fat decreased by 31% in the first nine months with most of the reduction occurring during the first three months. During the intervention period the percentage of energy derived from saturated fat decreased more at Minjilang than at the control community (Figure 5.14). Although the P:S ratio remained lower at the control community than at Minjilang over the intervention period (Figure 5.15), the P:S ratio actually increased at the control community. This was due primarily to the substitution of polyunsaturated cooking oil in all stores managed by the responsible store co-operative association after November 1989 (Figure 5.8). The production of wholemeal salad sandwiches as an alternate to high fat 'take-away' foods, the supply of leaner meat at Minjilang over the intervention period (Table 5.1), and the reduction in intake of 'snack foods', contributed to the reduction in the intake of both total and saturated fatty acids over the year (Table 5.3c). The provision of choice of cooking oils, polyunsaturated from June 1989 and monounsaturated (brand name: *Canola*) from February 1990, also contributed to the changing profile of fatty acid intake at Minjilang (Figure 5.8).

The percentage of energy derived from protein rose by 13% at Minjilang during the first three months of the intervention project, and stayed at that level for the rest of the year (Figure 5.12). At the control community, the percentage of energy derived from protein stabilised after the first three months at approximately 84% of the pre-intervention level.

Table 5.3a. Major food groups contributing to total energy intake (%)

Food group	Minjilang			Control Community		
	June 1989	December 1989	June 1990	June 1989	December 1989	June 1990
Cereals and cereal products	26.9	32.7	37.7	35.3	26.2	25.4
Fruits and Vegetables	2.0	3.4	4.8	2.5	1.4	1.7
Milk and Milk products	8.3	13.7	13.3	13.5	14.6	12.7
Meat and Meat products	8.3	11.6	7.2	10.3	11.6	9.2
Sugar, Jams, Honey and Syrups	14.4	19.8	14.4	23.2	31.4	31.7
Fats	10.3	5.4	9.7	5.4	5.9	7.1
Snack foods	6.5	0.7	0.6	0.5	0.7	0.7
Condiments	0.1	0.3	0.3	0.3	0.2	0.3
Beverages	7.8	5.6	5.1	5.1	3.7	4.8
Confectionery	3.5	2.6	3.6	1.9	2.2	4.2
Take-away foods	11.9	4.3	3.7	2.1	2.0	2.3

Table 5.3b. Major food groups contributing to total intake of sugars (%)

Food group	Minjilang			Control Community		
	June 1989	December 1989	June 1990	June 1989	December 1989	June 1990
Cereals and cereal products	6.1	4.8	6.7	5.7	2.6	2.8
Fruits and Vegetables	3.0	5.9	8.5	3.7	1.6	2.0
Milk and Milk products	7.3	11.1	8.5	8.11	10.8	7.8
Meat and Meat products	0.1	0.1	0.1	0.4	0.2	0.1
Sugar, Jams, Honey and Syrups	47.8	57.3	49.7	65.3	71.8	69.1
Fats	0.1	0.1	0.1	0.0	0.0	0.0
Snack foods	0.4	0.1	0.0	0.0	0.0	0.0
Condiments	0.2	0.04	0.2	0.3	0.3	0.4
Beverages	25.0	15.0	16.2	12.7	7.9	9.5
Confectionery	9.4	5.5	9.9	3.7	4.5	8.3
Take-Away foods	0.7	0.0	0.2	0.1	0.2	0.1

Table 5.3c. Major food groups contributing to total intake of fat (%)

Food group	Minjilang			Control Community		
	June 1989	December 1989	June 1990	June 1989	December 1989	June 1990
Cereals and Cereal products	8.7	11.6	12.0	13.4	7.1	9.5
Fruits and Vegetables	0.4	1.0	1.5	0.8	0.3	0.2
Milk and Milk products	10.7	22.2	21.4	24.3	25.2	22.8
Meat and Meat products	18.3	31.8	17.1	30.7	35.4	28.3
Sugar, Jams, Honey and Syrups	0.0	0.0	0.0	0.0	0.0	0.0
Fats	30.0	20.9	34.5	22.1	24.4	30.3
Snack foods	10.6	1.5	2.2	1.2	1.7	1.6
Condiments	0.1	0.2	0.4	0.4	0.2	0.2
Beverages	0.1	0.3	0.2	0.3	0.1	0.2
Confectionery	1.5	2.1	2.1	1.9	0.9	1.5
Take-Away foods	20.3	8.5	8.6	4.8	4.6	5.0

Table 5.3d. Major food groups contributing to total intake of folate (%)

Food group	Minjilang			Control Community		
	June 1989	December 1989	June 1990	June 1989	December 1989	June 1990
Cereals and Cereal Products	19.1	40.4	42.8	54.7	55.6	52.8
Fruits and Vegetables	21.0	26.5	34.4	16.0	12.0	14.9
Milk and Milk products	8.5	9.0	10.2	17.0	16.8	14.3
Meat and Meat Products	5.7	4.7	3.1	4.9	6.8	0.7
Sugar, Jams, Honey and Syrups	0.0	0.0	0.0	0.0	0.0	0.0
Fats	4.5	1.3	1.4	2.7	5.3	4.3
Snack foods	5.7	0.4	0.3	0.5	0.8	0.7
Condiments	2.1	10.0	5.7	2.0	0.2	1.5
Beverages	0.9	2.3	1.5	1.1	1.1	2.4
Confectionery	0.7	0.5	0.5	0.6	0.4	0.6
Take-Away foods	4.9	4.9	1.2	0.9	1.1	0.9

5.3.3. Folate

Prior to the intervention project at Minjilang, the mean density of folate intake at both Minjilang and the control community was less than 50% of the recommended intake of 21 $\mu\text{g}/1000 \text{ kJ}^2$ (Figure 5.16). Retrospective analysis of longitudinal store-turnover in both communities revealed no evidence of a seasonal pattern in density of folate intake over the four years preceding the intervention project. From March 1986 to September 1987 the density of folate intake was similar in both communities. However the density of folate intake tended to be somewhat higher in Minjilang from September 1987 to September 1988 and in the three months prior to June 1989. These periods corresponded to the appointment of specific store managers (section 7.2.2.4).

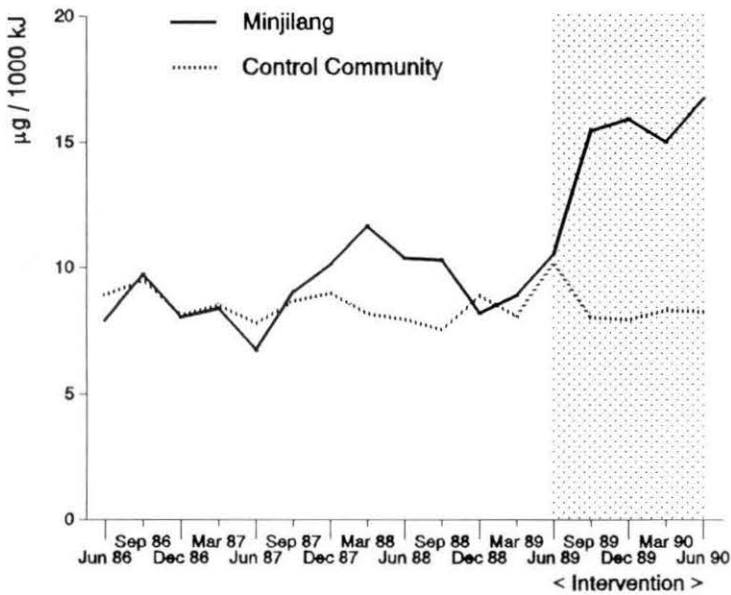


Figure 5.16. Density of folate intake during the previous three month period from June 1986 to June 1990

Comparison of store-turnover data between Minjilang and the control community over the intervention period indicated a marked increase in density of folate intake in Minjilang (Figure 5.16). The pre-intervention density of folate intake was approximately 10.5 $\mu\text{g}/1000 \text{ kJ}$ in both communities and density of folate intake increased by over 50% from

² Current recommended levels of folic acid are believed to reflect realistically both physiological requirements and 'usual' dietary intakes and the recommendation does not encompass the large 'safety margins' applied to recommended dietary intakes of other nutrients (Truswell, 1990).

the first three months in Minjilang, while remaining relatively constant in the control community. The changing source of folate in the Minjilang diet from June 1989 to June 1990 reflected a proportional increase in bread and cereals, particularly wholegrain varieties, fruit and vegetables and 'vegemite' (included as condiment), all of which were targeted during the intervention period (Table 5.3d). Comparatively little change was observed in the folate profile in the control community.

5.3.4. Thiamine, Riboflavin, Niacin and Pyridoxine

Retrospective analysis of longitudinal store-turnover in both communities prior to the intervention project at Minjilang (Figure 5.17, Figure 5.18, Figure 5.19, Figure 5.20), confirmed that the density of intake of most B-group vitamins was low relative to the recommended intake. In both communities during this period the mean density of both thiamine and riboflavin intake was approximately 67% of the recommended intake of 0.10 mg/1000 kJ and 0.16 mg/1000 kJ respectively, whereas the mean density of both niacin and vitamin B₆ were both less than 50% of the recommended intake of 1.77 mg/1000 kJ and 0.15 mg/1000 kJ respectively.

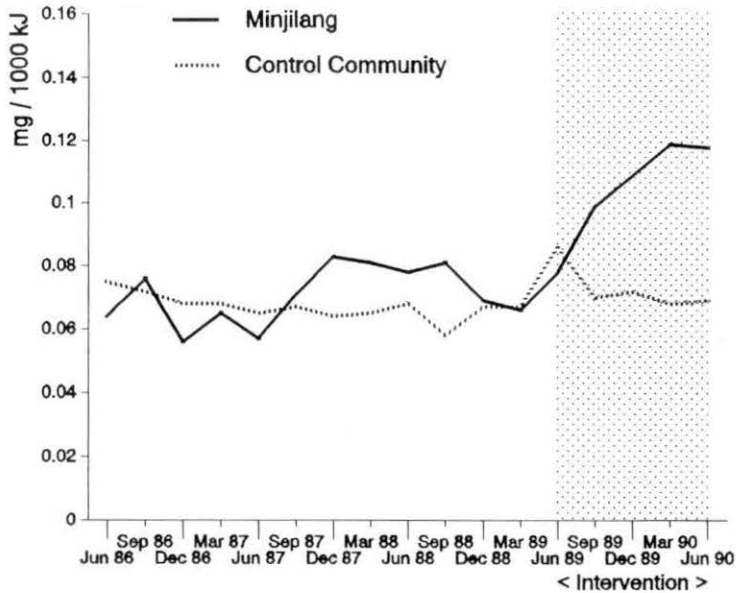


Figure 5.17. Density of thiamine intake during the previous three month period from June 1986 to June 1990

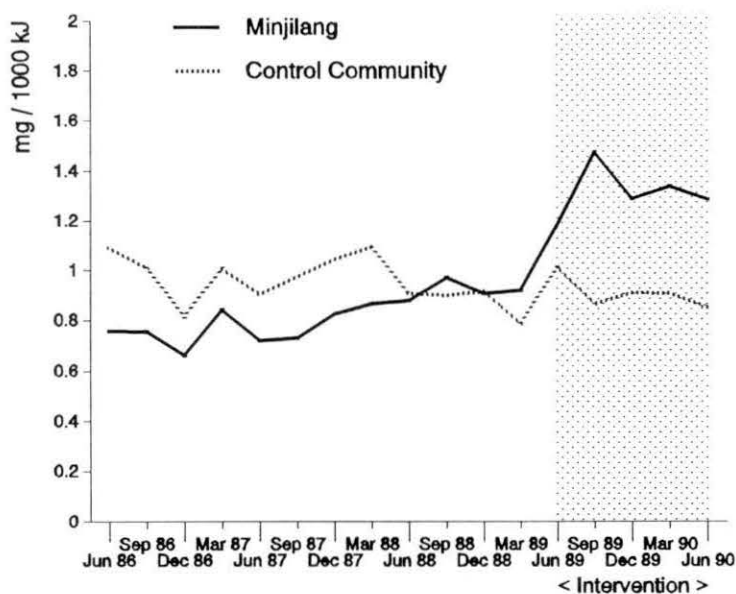


Figure 5.18. Density of niacin intake during the previous three month period from June 1986 to June 1990

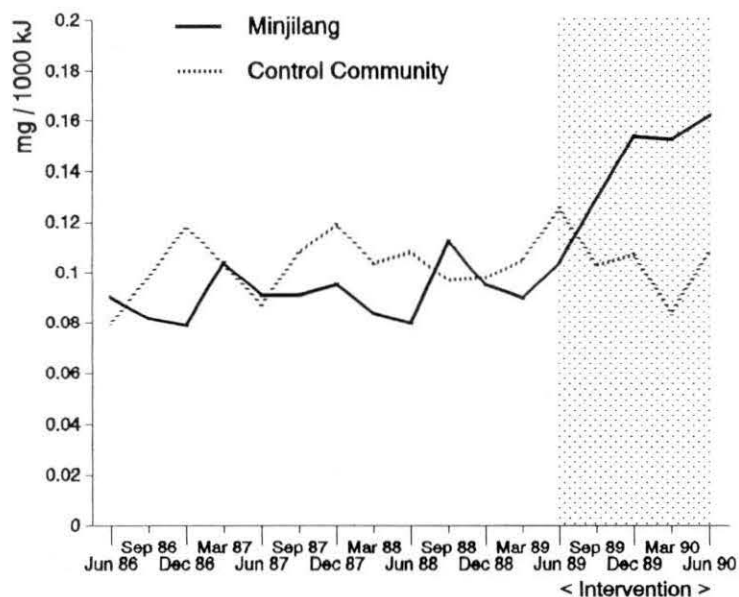


Figure 5.19. Density of riboflavin intake during the previous three month period from June 1986 to June 1990

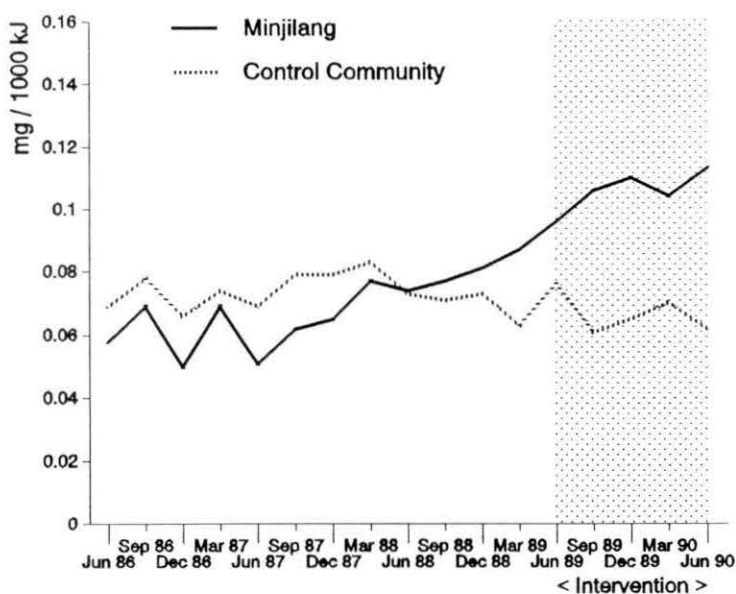


Figure 5.20. Density of vitamin B₆ intake during the previous three month period from June 1986 to June 1990

There was no evidence of a seasonal pattern in density of B-group vitamin intake over the four years preceding the intervention project.

The density of intake of most B-group vitamins appeared to be relatively constant in the control community during the total five year period, being approximately 0.07 mg/1000 kJ, 0.10 mg/1000 kJ, 0.9 mg/1000 kJ and 0.07 mg/1000 kJ respectively for thiamine, riboflavin, niacin and pyridoxine. However the density of intake of both thiamine and riboflavin in the control community tended to be somewhat higher during the three month period prior to June 1989 than at any other period, due to an increased turnover of wholemeal cereals and the popular introduction of fresh milk at that time. This period also corresponded to the appointment of a specific (nutritionally-aware) relief store manager at the control community (section 7.2.2.4).

The density of intake of most B-group vitamins tended to be slightly lower at Minjilang than at the control community until approximately June 1988. However the density of thiamine intake tended to be higher at Minjilang from June 1987 to September 1988 (Figure 5.17) due to an increased turnover of wholegrain cereals during that period; again this period corresponded to the appointment of a specific store manager (section 7.2.2.4). Density of pyridoxal intake (Figure 5.20), also began to increase at Minjilang relative to

the control community from June 1987. The major source of pyridoxal (approximately 20%) in the Minjilang diet was 'snack foods' at that time.

Comparison of store-turnover data between Minjilang and the control community over the intervention period, indicated that the pre-intervention density of thiamine, riboflavin and niacin was similar in both communities. In the first three months, density of intake of these B-group vitamins tended to decrease at the control community, while increasing at Minjilang. Thereafter intake of these B-group vitamins remained relatively constant in the control community, while continuing to increase at Minjilang. In June 1990, the densities of intake of thiamine, riboflavin, niacin and pyridoxal had increased respectively by 51%, 56%, 9% and 18% compared with the pre-intervention levels, and were respectively 71%, 50%, 51% and 82% higher at Minjilang than at the control community. At the end of the 12 month intervention period, the density of intake of both thiamine and riboflavin at Minjilang were comparable with recommended levels, and the density of both niacin and pyridoxal intake had increased to over 70% of the recommended level.

Major foods promoted at Minjilang which contributed to the increased density of intake of these nutrients during the intervention project included wholemeal bread, wholegrain breakfast cereals, yeast extract (brand name: *Vegemite*) and bananas (a rich source of vitamin B₆).

5.3.5. Vitamin B₁₂

Retrospective analysis of store-turnover data suggested that the density of intake of vitamin B₁₂ at Minjilang tended to be lower than that of the control community prior to December 1988 (Figure 5.21). There tended to be a seasonal pattern in density of vitamin B₁₂ intake, with vitamin B₁₂ intake from both stores tending to be higher during the wet season months between December and April. This effect was more marked at Minjilang than at the control community, particularly prior to 1989.

During the intervention project at Minjilang there was no nett increase in density of vitamin B₁₂ intake over the year in contrast to other B-group vitamins. Both pre- and post-intervention density of vitamin B₁₂ intake at both communities was comparable to the recommended intake of 0.21 µg/1000 kJ. The density of vitamin B₁₂ intake initially reduced in the first six months of intervention and then increased to a similar level as the preceding year. Neither did the density of vitamin B₁₂ intake at Minjilang differ

substantially from that measured every three months at the control community during this period, and both communities exhibited similar seasonal fluctuations of vitamin B₁₂ intake. Meat and meat products were consistently the major source of vitamin B₁₂ at both communities.

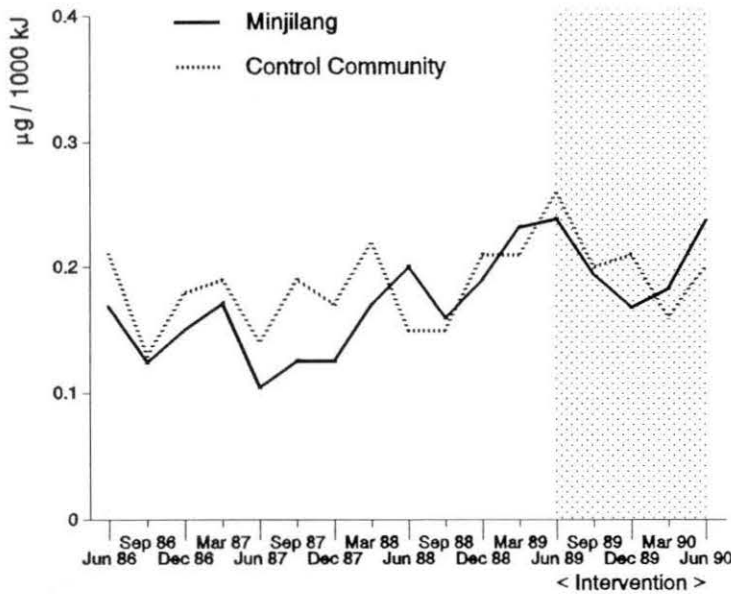


Figure 5.21. Density of vitamin B₁₂ intake during the previous three month period from June 1986 to June 1990

5.3.6. Ascorbic Acid

Retrospective analysis of longitudinal store-turnover in both communities, revealed a marked contrast between the pattern of dietary intake of ascorbic acid between Minjilang and the control community (Figure 5.22). The density of intake of ascorbic acid at the control community was frequently less than 50% of the recommended intake of 3.7 mg/1000 kJ. Although the density of ascorbic acid intake remained relatively constant in the control community, there was marked seasonal variation in this intake at Minjilang, where density of ascorbic acid intake tended to be lowest in the first half of the year and highest during the second half of the year. As with folate, the density of vitamin C intake was relatively high at both communities when the respective stores were managed by a specific store manager (section 7.2.2.4).

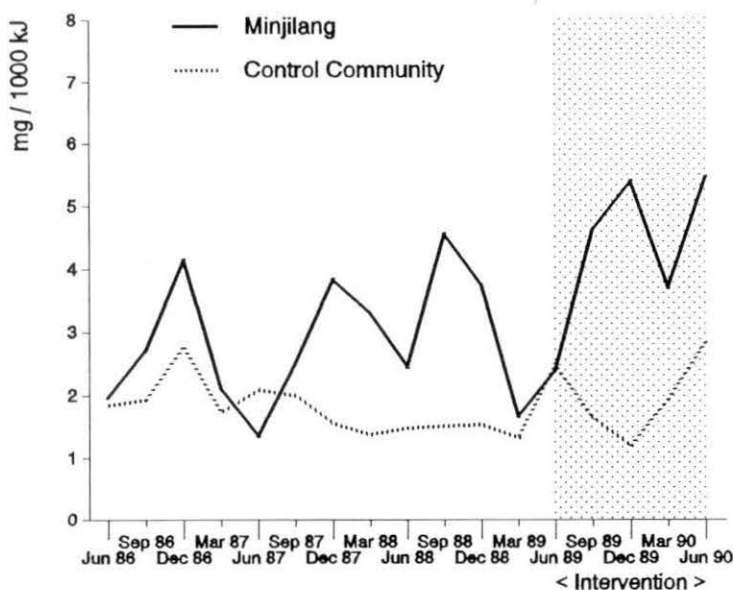


Figure 5.22. Density of ascorbic acid intake during the previous three month period from June 1986 to June 1990

Comparison of store-turnover data between Minjilang and the control community during the intervention period indicated that the density of ascorbic acid intake at Minjilang tended to remain at a higher level relative to the previous four years and almost doubled over the year. The pre-intervention density of ascorbic acid intake was similar in both communities at approximately 2.4 mg/1000 kJ. In the first six months density of ascorbic acid intake decreased by almost 50% in the control community but recovered during the second half of the year when the turnover of vitamin C enriched fruit juices increased at the control community (Figure 5.7). During the intervention project the density of ascorbic acid intake at Minjilang was consistently higher than the recommended level of 3.74 mg/1000 kJ.

5.3.7. Vitamin A

5.3.7.1. Retinol

Retrospective analysis of longitudinal store-turnover in both communities, revealed that there was a tendency for density of intake of retinol to gradually increase during the four years preceding the intervention project (Figure 5.23). In both communities the intake of retinol tended to be associated with the intake of energy derived from fat, and there was a

slight tendency for the density of retinol intake to increase during the coolest months of the year in line with the increase in the proportion of total energy intake derived from fat at that time. In both communities the major source (up to 50%) of retinol was from vitamin A supplemented margarine.

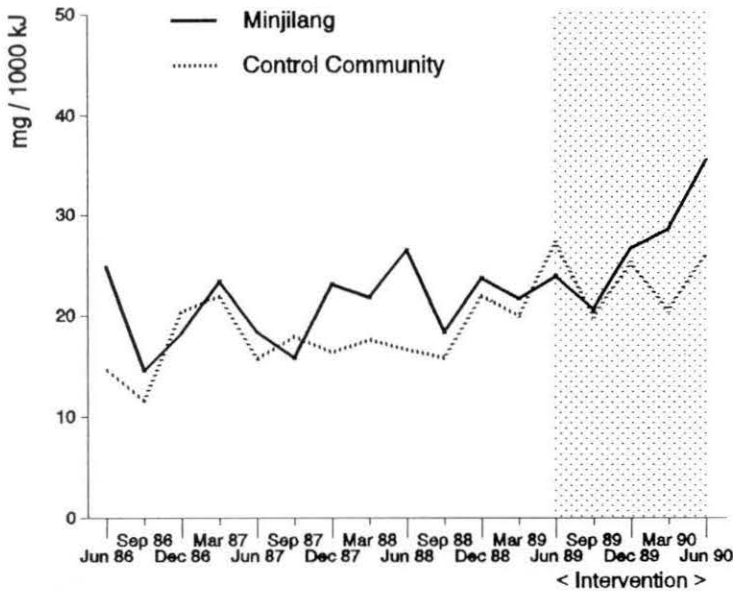


Figure 5.23. Density of retinol intake during the previous three month period from June 1986 to June 1990

Comparison of store-turnover data between Minjilang and the control community over the intervention period, indicated a consistent increase in density of retinol intake in Minjilang, to over 50% of the pre-intervention intake. The changing source of retinol in the Minjilang diet from June 1989 to June 1990 reflected a relative increase in milk and milk products. No such serial change was observed in the control community where the density of intake of retinol continued to fluctuate throughout the twelve month period.

5.3.7.2. β -carotene

Of all vitamins the intake of β -carotene tended to be most variable in both communities (Figure 5.24). At Minjilang there appeared to be a seasonal effect on intake of β -carotene, with intake being highest during the months of December, January and February. At all times intake of β -carotene tended to be lower at the control community than at Minjilang.

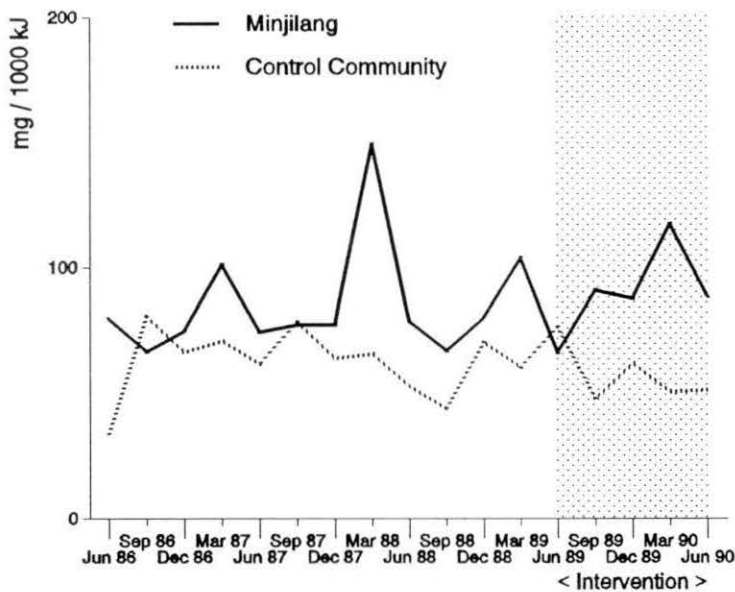


Figure 5.24. Density of β -carotene intake during the previous three month period from June 1986 to June 1990

Comparison of store-turnover data between Minjilang and the control community over the intervention period, indicated a increase in density of β -carotene intake at Minjilang while density β -carotene remained relatively constant at the control community. However, the specific effect of the intervention project on β -carotene is unclear in the light of prior fluctuations in the density of that pro-vitamin at Minjilang.

The changing source of retinol in the Minjilang diet from June 1989 to June 1990 reflected a relative increase in β -carotene containing yellow vegetables and fruits which were targeted during the intervention period (Figure 5.1). No such change was observed in the control community.

5.3.7.3. Vitamin A Equivalents

Density of intake of vitamin A equivalent reflected the pattern of both retinol and β -carotene intake (Figure 5.25); marked fluctuations were again apparent. In both communities the density of intake of vitamin A equivalent was consistently in excess of the recommended intake of 25 IU/1000 kJ (74.8 retinol equivalents per 1000 kJ). The density of intake of vitamin A equivalent was particularly high at Minjilang during the intervention period, but also from June 1987 to September 1988. Again the density of

intake of this nutrient tended to be relatively high at both communities when the respective stores were managed by a specific store manager (section 7.2.2.4); at that time the proportion of total retinol equivalent derived from carotenoid sources increased.

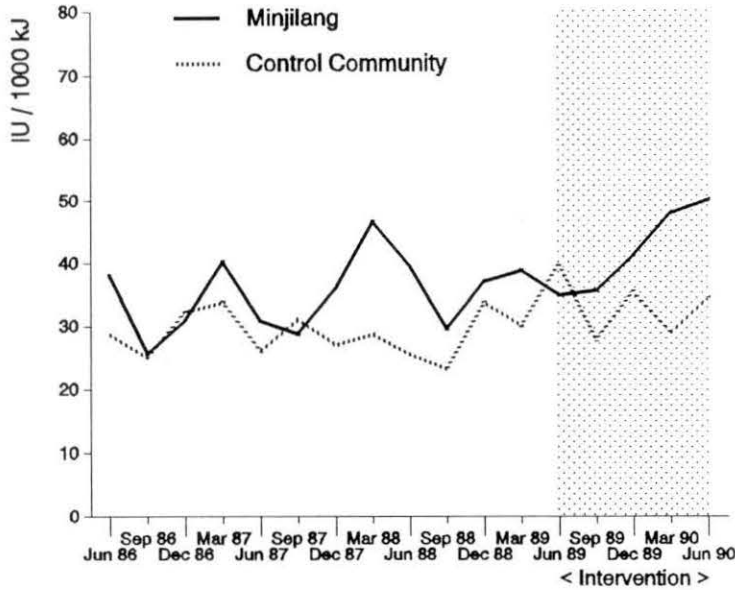


Figure 5.25. Density of intake of vitamin A equivalents during the previous three month period from June 1986 to June 1990

5.3.8. Vitamin E

Retrospective analysis of longitudinal store-turnover in both communities, revealed that density of intake of α -tocopherol increased dramatically at Minjilang from December 1987 relative to the control community, although it tended to increase slowly in the control community from June 1989 (Figure 5.26). This increase was associated with the increasing intake of energy derived from fat in both communities, and, as with retinol, there was also evidence of a relative seasonal increase in the intake of this fat soluble vitamin during the coolest months of the year. The density of intake of vitamin E was consistently much lower than the calculated recommended level of 0.92 mg/1000 kJ.

Throughout the entire intervention period, density of vitamin E intake was higher at Minjilang than at the control community. Prior to the last three month period, density of α -tocopherol intake tended to decrease at Minjilang while increasing at the control community. This effect was associated with the pattern of fat intake.

lowest during the dry season months. As with vitamin B₁₂ the density of intake of both iron and zinc tended to be At both communities the major dietary source of both iron and zinc was meat and meat

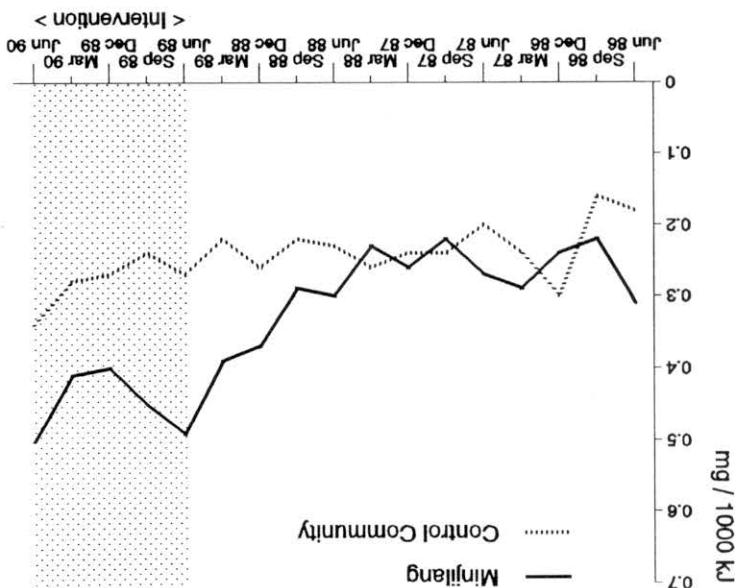
1.2 mg/1000 kJ.

intake at both communities was consistently less than 60% of the recommended level of zinc tended to be lower at Minjilang until early 1989 (Figure 5.28). The density of intake of Although tending to follow a similar pattern in both communities, the density of intake of

approximately 66% of the recommended level of 1.2 mg/1000 kJ. Retrospective analysis of longitudinal store-turnover in both communities revealed a similar pattern of dietary iron intake in both Minjilang and the control community, particularly up to March 1988 when the intake of iron tended to rise at Minjilang (Figure 5.27). Prior to June 1989 the mean density of iron intake at both communities was

5.3.9. Iron and Zinc

Figure 5.26. Density of intake of vitamin E during the previous three month period from June 1986 to June 1990



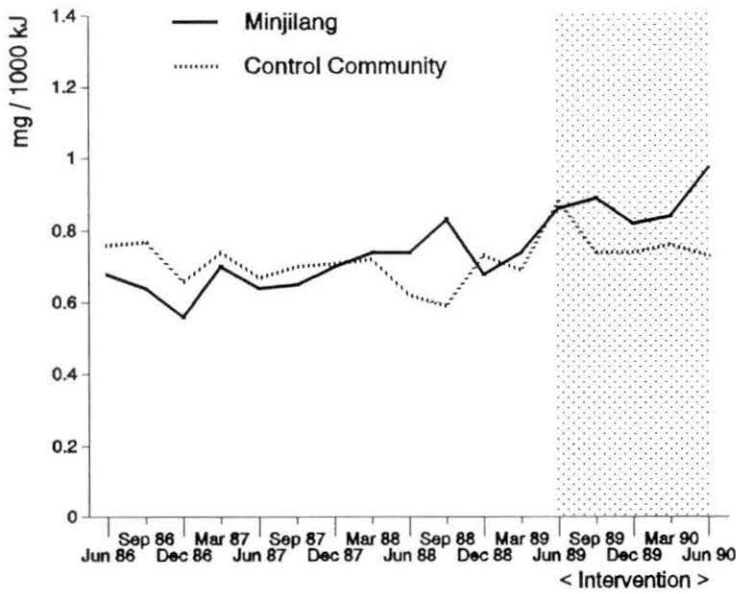


Figure 5.27. Density of iron intake during the previous three month period from June 1986 to June 1990

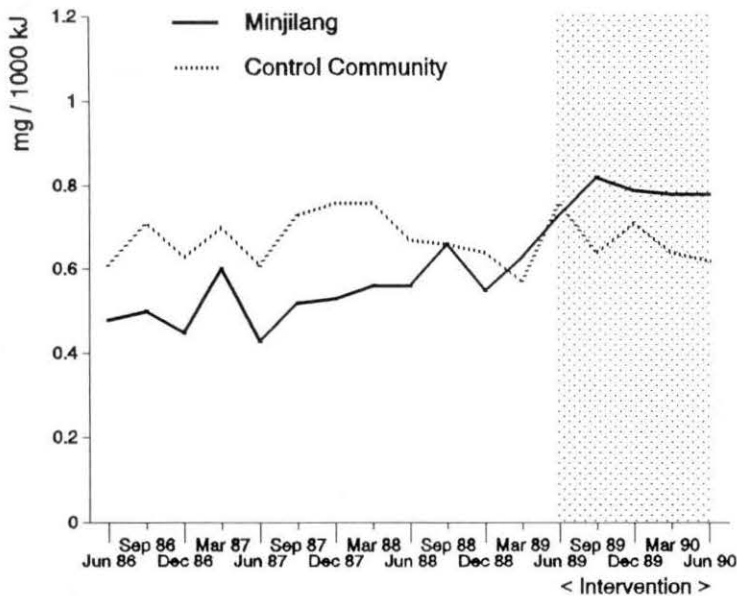


Figure 5.28. Density of zinc intake during the previous three month period from June 1986 to June 1990

Comparison of store-turnover data between Minjilang and the control community indicated that the density of both iron and zinc intake was similar in June 1989. At Minjilang, the

density of both minerals remained relatively high throughout the intervention period in continuation of a clear pre-intervention trend. Following the intervention project, the density of both iron and zinc intake was approximately 30% higher at Minjilang than at the control community; apparent intake of iron was more than 80% of the recommended level and the apparent intake of zinc was more than 66% of the recommended level.

5.3.10. Calcium

Retrospective analysis of longitudinal store-turnover in both communities, revealed a similar pattern of dietary calcium intake in both Minjilang and the control community, particularly up to September 1988. Up until this time the density of intake of calcium was approximately 60% of the recommended level of 102 mg/1000 kJ. At that time calcium intake rose comparatively at the control community mainly due to the introduction of flavoured milk drinks which appeared to reduce in demand/supply after May 1989 (Figure 5.29). There was no evidence of a seasonal effect on calcium intake.

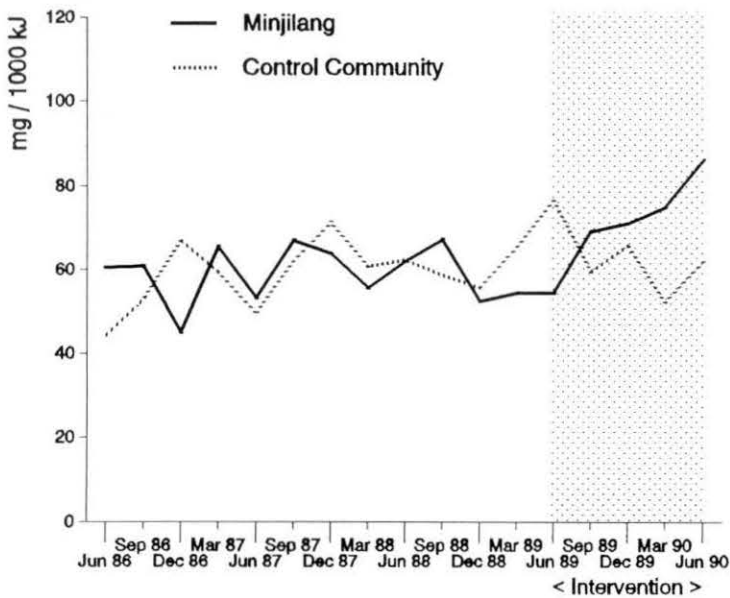


Figure 5.29. Density of calcium intake during the previous three month period from June 1986 to June 1990

Comparison of store-turnover data between Minjilang and the control community during the intervention period indicated that the density of calcium intake tended to increase in Minjilang throughout the intervention period but fluctuated in the control community.

Throughout the intervention period the density of calcium intake increased by 58% at Minjilang, and reached almost 90% of the recommended level. The major dietary source of calcium at Minjilang throughout the intervention period was dried milk.

5.3.11. Dietary Fibre

Density of dietary fibre intake was much higher at Minjilang than at the control community throughout most of the period considered, and was particularly high from June 1987 to September 1988 (corresponding to an increased turnover of fruit, vegetables and wholegrain cereals (Figure 5.30). Density of fibre intake tended to be less variable in the control community, but was again relatively higher when a specific store manager was temporarily employed at the community (section 7.2.2.4). Over the intervention period the density of intake of dietary fibre tended to increase at Minjilang but decrease at the control community.

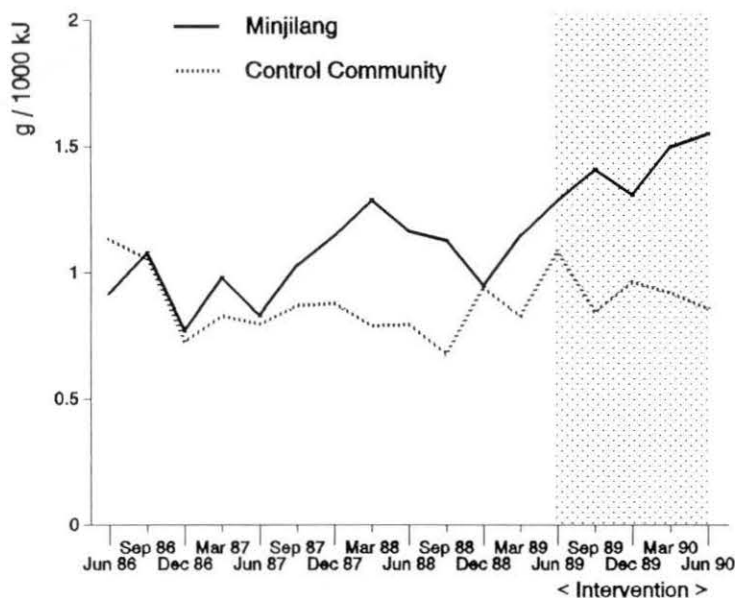


Figure 5.30. Density of dietary fibre during the previous three month period from June 1986 to June 1990

5.3.12. Dietary Cholesterol

Longitudinal analysis of store-turnover data indicated that density of intake of cholesterol tended to be more consistent at the control community, but more erratic and, prior to

December 1989, lower at Minjilang (Figure 5.31). The variation in the density of cholesterol intake measured at Minjilang could be partially explained by the fluctuation in the supply of hens' eggs at that community; in the last three months of the intervention period fresh eggs were consistently available at the store and turnover increased by 20%. The density of cholesterol intake remained relatively constant at the control community throughout the year.

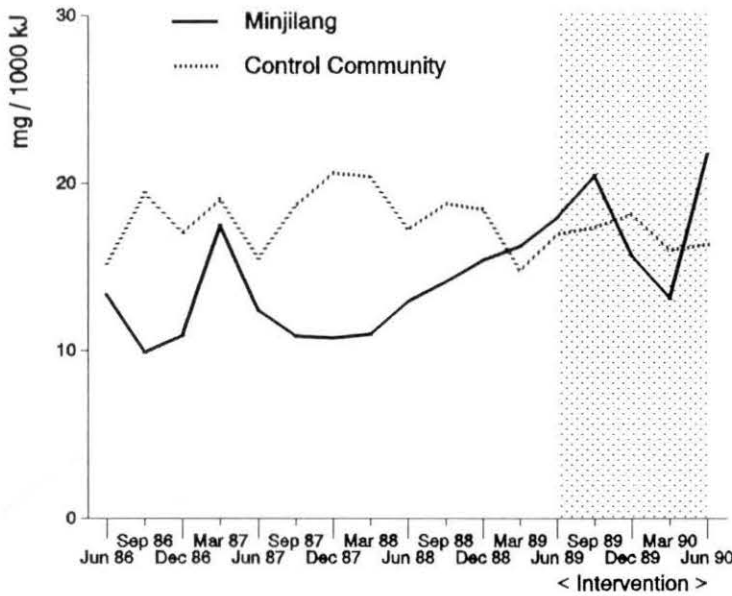


Figure 5.31. Density of dietary cholesterol during the previous three month period from June 1986 to June 1990

5.4. Contemporary use of traditional foods at Minjilang

5.4.1. Frequency of hunting/gathering activities at Minjilang

Throughout the year most adults attempted to procure traditional bush foods on less than one day per fortnight (Table 5.4); most activity occurred at weekends. The highest frequency of gathering bush food for women occurred during July 1989, when three women temporarily resided at a coastal outstation during the dry 'bush holiday' season for two one-week periods. Conversely, men most frequently sought bush foods during the late dry season ('magpie geese time') when four men went shooting three or four evenings per week. Older people tended to procure traditional foods at a greater frequency than

younger adults. The three months prior to March 1990 were the quietest; most people tended to relax and/or play cards at Minjilang rather than forage, fish or hunt during the height of the wet season.

5.4.2. Descriptive intake of traditional foods at Minjilang June 1989 to June 1990

Those traditional vegetable foods (Addendum 2, Table A2.3a) and animal foods (Addendum 2, Table A2.3b) obtained at least once between June 1989 and June 1989 were listed. Although the lists appeared extensive, they represented only a small proportion of the traditional food resources available from the rich and diverse environments on the island.

There was a strongly stated preference for traditionally favoured foods. These included animal foods which provided a rich source of fat (turtle, dugong and magpie geese) and sweet foods ("*kakurl*"). In contrast, some foods were collected once only for demonstration purposes ("*kanirr*", "*kurldi*" and "*garbiyoyo*").

Although several foods were constantly available throughout the year, accessibility was not necessarily assured. In particular, access to a boat or car was usually required for coastal or riverine fishing. Only one family group had the traditional right to fish within walking distance of the community at Mission Bay. During the year a total of two out-board powered dinghies and nine motor vehicles were operated by community members, including those vehicles owned by the community council. The boats and motor vehicles were often non-serviceable.

During the wet season relatively treacherous seas and unpleasant conditions discouraged fishing. With the exception of barramundi caught by two families at Palm Beach Creek on the western side of the island, few fresh fish were consumed by the Minjilang community during the wet season.

Of the small number of fruits collected during the dry season, most were distributed to children. They tended to be collected on route to a fishing spot, rather than being the primary aim of a collecting trip. Occasionally some varieties of yam, particularly "*lungun*", were brought back to the community and shared with family members.

Table 5.4. Frequency of hunting/gathering activities during the previous three month period: adults residing at Minjilang

	June 1989 n (%)	September 1989 n (%)	December 1989 n (%)	March 1990 n (%)	June 1990 n (%)
Nearly every day	0 (0)	1 (1.5)	1 (1.5)	0 (0)	0 (0)
At least five days per fortnight	3 (4.2)	10 (14.9)	11 (16.4)	0 (0)	2 (2.9)
At least three days per fortnight	18 (25.0)	12 (17.9)	6 (9.0)	2 (3.0)	22 (31.9)
At least one day per fortnight	22 (30.6)	18 (26.8)	24 (35.8)	13 (19.7)	22 (31.9)
Rarely/never	28 (38.8)	26 (38.8)	25 (37.3)	41 (62.1)	23 (33.3)
Total number	72	67	67	66	69

The intake of traditional foods varied during the year. During the late dry season there was an increased availability of magpie geese. During this period four residents of the control community also visited Croker island to hunt the geese. In excess of thirty birds were bagged and transported to the control community during this season. When the store freezer required maintenance and frozen meat was temporarily unavailable at Minjilang, eleven green-back turtles were hunted in a single period from 4/11/1989 to 18/11/1989.

A very rough guesstimate of the proportion of total energy intake derived from bush foods averaged over the population at Minjilang would be less than eight percent during the dry season and less than half this proportion during the peak of the wet season.

Fatty acid analysis of traditional animal foods collected at Minjilang revealed that the lipid fraction of turtle fat was unusually high in lauric acid (C12:0) (Addendum 2, Table A2.4). Dugong fat and turtle fat were also relatively high in myristic acid (C14:0). As in all other meats, the lipid fraction of goose and dugong flesh was relatively high in oleic acid (C18:1).

5.5. Major points

The initial application of the store-turnover method in June 1989 at both Minjilang and the control community revealed similarities to other Aboriginal communities in that intake of energy, sugars and saturated fat were excessive and nutrient density was low. However, longitudinal store-turnover data suggested that there had been a gradual decrease in the turnover of sugar *per se* at Minjilang over the four years prior to the commencement of the intervention project; at the commencement of the intervention project the diet at Minjilang was relatively lower in sugars and higher in fats than the control community and tended more towards the style of diet of wider (non-Aboriginal) Australia.

Longitudinal store-turnover data disclosed the relative seasonal changes in the turnover of targeted foods during the intervention period in Minjilang compared to the preceding four years and also compared with the control community. Over the intervention period, changes in the apparent consumption of targeted foods in Minjilang included a marked increase in the consumption of fresh fruit and vegetables and wholemeal bread, and a decrease in the turnover of take-away foods and sugar. There was also an increased proportion of nutritionally-preferred varieties of carbonated beverages (artificially sweetened) and cooking oils (polyunsaturated and monounsaturated) at Minjilang over the

intervention period.

Nutrient analysis of store-turnover data as measured every three months from June 1989 to June 1990 at Minjilang suggested a decrease in intake of saturated fat, a small decrease in intake of sugars, a increase in dietary fibre and marked improvement in the density of intake of most vitamins over the intervention period.

These changes were further supported by nutrient analysis of longitudinal store-turnover data, which demonstrated the relative seasonal variation in nutrient density in Minjilang over the intervention period compared with the preceding four years and also compared with the control community throughout the entire five year period.

Consideration of bush food intake from June 1989 to June 1990 suggested that the mean per capita intake of traditional foods was low throughout the Minjilang community as a whole.

CHAPTER 6: A COMMUNITY-BASED NUTRITION INTERVENTION PROJECT: BIOCHEMICAL, ANTHROPOMETRIC AND HAEMATOLOGICAL RESULTS AND COMMENTS.

In this chapter the results of the biochemical, anthropometric and haematological variables measured at three month intervals throughout the intervention period are presented. Findings are presented in three specific clusters; anthropometric and metabolic results, haematological results (incorporating folate data), and other vitamin results. The results of analysis less central to the major thesis are presented in a smaller font.

6.1. Sample

Details of the sample screened at each three month period are provided in Table 6.1. Analysis of variance indicated that there was no significant difference in either the age or sex of those subjects monitored at different surveys (Table 6.7).

Ninety-four percent of all adults residing at Minjilang in June 1989 participated in the initial screening process. Of those four Minjilang residents originally declining to participate in June 1989, two were an elderly couple over the age of sixty years, one was an elderly, incapacitated woman who died in November 1989 and one was an overweight man in his late forties who stated that he was not interested in the project. The major reason that subjects from the original sample did not participate in subsequent surveys was temporary absence from the community (Table 6.1a). The number of adults available for survey was lowest in September 1989 when several Minjilang residents remained for a short period in a neighbouring community following traditional ceremonies, and in March 1990 when a group of young adults went to Darwin for an Aboriginal rock concert. Of those present in the community at the time of subsequent surveys but refusing to participate, six older subjects stated that they were "*weak*" from giving blood during previous surveys. These subjects believed, in line with local traditional conviction, that blood was not restored in the body after removal. In addition, four younger subjects declined to participate in subsequent surveys because they were "*afraid of the needle*". The number of available subjects choosing to participate was lowest in March 1990 as several older residents decided to "*save their blood*" for the final survey in June 1990.

Two 'control' subjects, (the Aboriginal research assistant and myself) participated in all surveys. Several residents from other communities travelled to Minjilang at survey time in order to be "*checked right through*". These data were excluded from analysis which was confined to Minjilang residents only.

To investigate possible sample bias, the subset of subjects attending on all occasions was compared to the total population. In the first screening there was no significant difference between those who would subsequently participate in all surveys and the community in general (except for serum thiamine; $t=-2.31$, $p<0.05$). However over all surveys this subset contained a greater proportion of women ($t=3.58$, $p<0.001$), those of higher BMI ($t=2.67$, $p<0.01$) and diabetics ($t=2.55$, $p<0.05$) than the community as a whole. Over all the surveys there was no significant difference between the two groups in terms of either mean age ($t=0.53$), systolic blood pressure ($t=-1.36$), diastolic blood pressure ($t=-1.56$), serum cholesterol ($t=1.74$), fasting serum triglyceride ($t=-0.96$), red blood cell folate ($t=1.59$), retinol ($t=0.13$), α -tocopherol ($t=-0.43$), red blood cell thiamine ($t=0.28$), serum thiamine ($t=-0.29$), vitamin B₆ ($t=-1.44$), vitamin B₁₂ ($t=-1.44$) or ferritin ($t=1.70$) concentrations, or between kava consumption ($t=1.85$), cigarette smoking ($t=1.39$) or consumption of alcohol ($t=1.37$). However, over all surveys, the subset did have a significantly lower mean γ gt concentration ($t=-3.26$, $p<0.001$), but significantly higher β -carotene ($t=2.95$, $p<0.005$), ascorbic acid ($t=2.38$, $p<0.05$) and serum folate ($t=2.41$, $p<0.05$) concentrations than the community as a whole. Differences in characteristics of the subset of the original sample screened at each three month period and the community as a whole, may have arisen due to the increased effect of the intervention program in those who presented at every survey. However, subsequent analysis of variance after controlling for inter-individual variation (section 6.3), suggested that there was no significant difference in the change of these variables over time between the two groups (Table 6.4b).

Of those who stated that they were fasting, insulin concentrations supported fasting status in all but one subject; all relevant results for this subject were subsequently excluded from analysis over all surveys. Using WHO criteria (section 1.6.7.1), the glucose tolerance of one diabetic subject diagnosed in June 1989 had improved to the extent that she was no longer classified as a diabetic in June 1990; relevant results for this subject were included with those of non-diabetic subjects in longitudinal analysis.

Table 6.1a. Total sample screened at each three month period

	6/89	9/89	12/89	3/90	6/90
Total adults (≥ 18 years) living at Minjilang	72	67	67	66	69
Deceased since last survey	-	-	1	-	-
Number of people permanently left Minjilang since last survey	-	9	2	2	1
Number of new residents in Minjilang since last survey	-	4	3	1	4
Residents temporarily absent	1	11	5	10	4
Number of residents available for survey	71	56	62	56	65
Number of original population available for survey	71	55	60	53	58
Number of original sample available for survey	68	52	58	51	56
New residents declining to participate (accumulative)	-	2	4	5	8
Number of original sample declining to participate in subsequent surveys (accumulative)	3	5	6	10	10
Number of original sample temporarily declining to participate	-	2	2	9	-
Total number of people participating in survey	68	52	58	33	48
'Control' subjects	2	2	2	2	2
Number of Minjilang residents participating (%)	68 (94.4)	45 (67.1)	51 (76.1)	32 (48.5)	47 (68.1)
Number of original population participating (%)	68 (95.8)	43 (60.6)	48 (67.6)	29 (40.8)	44 (62.0)
Number of original sample participating (%)	68 (100)	43 (63.2)	48 (70.6)	29 (42.6)	44 (64.7)
Number of original sample available participating (%)	68 (100)	43 (82.7)	48 (82.8)	29 (56.9)	44 (78.6)

Table 6.1b. Characteristics of sample screened at each three month period

	6/89	9/89	12/89	3/90	6/90
Men < 35 years	16	7	9	5	11
Men ≥ 35 years	17	13	15	8	11
Women < 35 years	14	10	9	7	11
Women ≥ 35 years	21	15	18	12	14

Table 6.1c. Characteristics of sub-sample screened at June 1989 and June 1990

	Both surveys
Men < 35 years	10
Men ≥ 35 years	10
Women < 35 years	11
Women ≥ 35 years	13

Table 6.1d. Characteristics of sub-sample screened at every three month period

	All surveys
Men < 35 years	2
Men ≥ 35 years	6
Women < 35 years	5
Women ≥ 35 years	8

6.2. Anthropometric and metabolic data

6.2.1. Repeated intra- and inter-batch measurement

As determined by the intra-class correlation coefficient, results suggested good agreement between most repeated measurement of variables within the same batch (Table 6.2) and between batches (Table 6.3). For all repeated measurements the intra-class correlation coefficient was greater for the repeated intra-batch measurements than for the inter-batch measurements.

Relatively poor intra-batch agreement was found for hip and waist circumference and the dependent hip:waist circumference ratio.

Poor inter-batch agreement was apparent for α -carotene concentration; the repeat values were significantly higher than the original results (Addendum 2, Table A2.4b). For completeness, available results are presented in full in subsequent sections, but inferences concerning the affected α -carotene results are not made.

Table 6.2. Repeated intra-batch measures of anthropometric and biochemical parameters (mean \pm sd)

Variable	Assay 1 (n=10)	Assay 2 (n=10)	Intra-class correlation coefficient
Fasting serum triglyceride concentration (mmol/l)	1.63 \pm 0.81	1.62 \pm 0.80	0.96
Serum cholesterol concentration (mmol/l)	5.74 \pm 0.83	5.72 \pm 0.95	0.97
Serum HDL-cholesterol concentration (mmol/l)	0.91 \pm 0.14	0.90 \pm 0.13	0.89
γ gt concentration (U/l)	60.6 \pm 46.9	61.5 \pm 47.1	0.99
Serum ferritin concentration (μ g/l)	140.5 \pm 127.1	136.9 \pm 124.6	0.99
Red blood cell folate (μ g/l)	134.8 \pm 6.6	139.2 \pm 6.9	0.96
Plasma retinol (mg/100ml)	5.74 \pm 2.62	5.95 \pm 2.67	0.92
Plasma α -tocopherol (mg/l)	13.1 \pm 4.6	13.2 \pm 4.2	0.94
Plasma β -carotene (mg/100ml)	6.4 \pm 2.9	6.6 \pm 3.1	0.92
Plasma α -carotene (mg/100ml)	10.7 \pm 6.2	11.0 \pm 6.7	0.88
Weight (kg)	60.1 \pm 18.4	60.2 \pm 18.4	0.99
Height (cm)	161.6 \pm 9.3	161.4 \pm 8.5	0.99
Waist circumference (cm)	88.1 \pm 12.6	88.9 \pm 14.2	0.87
Hip circumference (cm)	86.5 \pm 10.9	89.8 \pm 11.2	0.85

Table 6.3. Repeated inter-batch measures of metabolic parameters (mean \pm sd)

Variable	Original (n=10)	Repeated (n=10)	Intra-class correlation coefficient
Fasting serum triglyceride concentration (mmol/l)	1.75 \pm 0.95	1.78 \pm 0.86	0.96
Serum cholesterol concentration (mmol/l)	6.32 \pm 1.54	6.28 \pm 1.39	0.94
Serum HDL-cholesterol concentration (mmol/l)	0.96 \pm 0.14	0.95 \pm 0.14	0.80
serum γ gt concentration (U/l)	60.3 \pm 45.3	61.4 \pm 46.8	0.98
Serum ferritin concentration (μ g/l)	137 \pm 123	134 \pm 123	0.97
Red blood cell folate concentration (μ g/l)	134.1 \pm 6.9	128.1 \pm 6.5	0.93
Plasma retinol concentration (mg/100ml)	5.53 \pm 2.53	5.21 \pm 2.44	0.90
Plasma α -tocopherol (mg/l)	13.8 \pm 4.7	13.1 \pm 4.0	0.93
Plasma β -carotene (mg/100ml)	6.5 \pm 3.0	6.1 \pm 2.4	0.88
Plasma α -carotene (mg/100ml)	1.7 \pm 2.2	11.5 \pm 7.5	0.58

6.2.2. Internal quality control

In addition to the application of the customary validation and reliability protocols within the laboratories analysing samples, the non-Aboriginal researcher and Aboriginal research assistant participated in all surveys as a (weak) form of internal quality control. Samples were collected, transported and analysed blindly under identical conditions to those of the Minjilang sample. For both 'controls', the initial results of α -carotene concentrations were very low and the final ascorbic acid concentration measured for the non-Aboriginal researcher was also low compared to the Caucasian reference range. For all other measurements at all other surveys the results of the non-Aboriginal researcher lay within the accepted Caucasian reference range. The red blood cell folate, plasma ascorbic acid and plasma β -carotene concentration of the Aboriginal research assistant were initially low but all subsequent measurements were within the Caucasian reference range. It was not possible to determine the effect that dietary change may have contributed to these results.

Analysis of variance indicated that most results were consistent over all surveys; the two exceptions were serum thiamine concentration ($F_{1,9}=12.5$, $p<0.05$) and red blood cell thiamine ($F_{1,9}=8.80$, $p<0.05$). Although these vitamins remained consistent for the non-Aboriginal researcher, the Aboriginal research assistant's results were relatively high at December 1989. Subsequent analysis suggested that dietary change may have contributed to these high results (section 7.2.8.3).

The fact that most 'control' results were consistent over all surveys and lay within the accepted Caucasian reference ranges supported the results of the repeated intra- and inter-batch measurement (section 6.2), suggesting that, with the exception of α -carotene, there had been no major systematic error due to collection, handling, transport or analysis of samples.

6.3. Statistical considerations

By adjusting for between subject differences it was possible to provide a more powerful test for differences between occasion (section 4.10). However it was necessary to ensure that it was statistically valid to compare the results of all subjects surveyed at any three month period over the intervention project, in particular (due to the non-orthogonal nature of the study) that the inter-individual variation was not confounded with the intra-individual variation.

Therefore the subset of subjects attending on all occasions (where these effects are orthogonal), was used to investigate the effect of intra-individual and inter-individual change in key variables over the intervention period (Table 6.4a). Under these circumstances subject identification code, when fitted as a 21 level factor, necessarily explained all of the inter-individual variation in the variable under consideration, while survey number, when fitted as a five-level factor, necessarily explained all of the intra-individual variation in the variable under consideration. To measure the proportion of intra-individual variation that reflected any linear change of the variable under consideration over the intervention period, survey number was fitted, not as a factor, but as an ordinal (linear) variable.

However restriction of analysis to those 21 individuals for which a complete data set was available severely reduced the information available. This was particularly so in respect of comparison of biological and dietary data. For this purpose the best estimate of the nutritional status of the community necessarily included as many individuals as possible each time the dietary data was obtained. Moreover, as previously demonstrated (section 6.1), there was evidence that the subset of subjects attending on all occasions was biased with respect to some variables, (including γ gt, β -carotene, ascorbic acid and serum folate concentrations), compared with the community as a whole.

Therefore analysis was also conducted for all available data for key variables over the five surveys; that is, for all subjects attending at any survey during the intervention period.

Although the linear estimate of intra-individual variance of some key variables (BMI, serum cholesterol concentration and red blood cell folate concentration) tended to be slightly greater for those subjects attending any survey than the intra-individual variance for the same variables measured for the subset of subjects attending survey on all occasions, there was no substantial difference between either observed total intra-individual, linear intra-individual or inter-individual variance of the two groups (Table 6.4a). Neither was there a substantial

difference in estimates of intra-individual¹ and inter-individual variance of potentially biased variables (section 6.1) in those subjects attending at every survey compared to the intra-individual and inter-individual variance of the subset of subjects attending on all occasions (Table 6.4b).

Therefore, survey number, when fitted as a five-level factor across all available (non-orthogonal) data, was shown to closely approximate the intra-individual variation in the variable under consideration. Likewise subject identification code, when fitted concurrently as a 94 level factor across all available (non-orthogonal) data, was shown to closely approximate the inter-individual variation in the variable under consideration. An estimation of the proportion of intra-individual variation that reflected any linear change over the period of the intervention, was obtained by fitting survey number, not as a factor, but as an ordinal (linear) variable across all available data.

Care must be taken in interpretation of the results of analysis of variance due to the dependent nature of many of the variables investigated (particularly the nutritional variables). In particular, it must be recognised that some of the explained variance is due to chance.

¹ There was a greater intra-individual decrease in γ_{gt} in the subset of subjects attending all surveys.

Table 6.4a. Intra- and inter-individual variance of key variables for the subset of subjects attending on all occasions and the estimate of intra- and inter- individual variance for the same variables measured for those attending any survey

	BMI [*]	Serum cholesterol concentration	Fasting serum Triglyceride [*]	Red Blood Cell Folate ^o concentration	Serum Haemoglobin ^o concentration	Systolic blood pressure
Subset (n) ^o	95	100	80	89	82	98
Inter-individual variance	$F_{20,75} = 164.4^{***}$	$F_{20,80} = 15.8^{***}$	$F_{17,63} = 4.00$	$F_{18,71} = 5.04^{***}$	$F_{18,64} = 16.6^{***}$	$F_{20,78} = 14.7^{***}$
Intra-individual variance (total)	$F_{4,75} = 8.50^{**}$	$F_{4,80} = 3.71^{**}$	$F_{4,63} = 0.63$	$F_{4,71} = 29.4^{***}$	$F_{4,64} = 5.73^{**}$	$F_{4,78} = 7.49^{**}$
Intra-individual variance (linear)	$F_{1,75} = 6.27^*$	$F_{1,80} = 5.72^*$	$F_{1,63} = 1.25$	$F_{18,71} = 92.4^{***}$	$F_{1,64} = 0.15$	$F_{1,78} = 10.3^{**}$
Total sample (n) ^o	230	230	201	219	209	229
Inter-individual variance	$F_{71,199} = 175.9^{***}$	$F_{71,199} = 11.0^{***}$	$F_{65,136} = 2.64$	$F_{68,151} = 2.16^{**}$	$F_{68,141} = 24.7^{***}$	$F_{69,160} = 10.8^{***}$
Intra-individual variance (total)	$F_{4,199} = 25.56^{***}$	$F_{4,199} = 6.56^{**}$	$F_{4,136} = 1.78$	$F_{4,151} = 56.7^{***}$	$F_{4,141} = 4.43^{**}$	$F_{4,160} = 7.05^{**}$
Intra-individual variance (linear)	$F_{1,199} = 17.53^{***}$	$F_{1,199} = 16.3^{***}$	$F_{1,136} = 2.68$	$F_{1,151} = 206.8^{***}$	$F_{68,141} = 0.12$	$F_{1,160} = 13.8^{**}$

^o Excluding missing values and relevant results where fasting status was not confirmed (section 4.7.10)

^{*} Excluding diabetics ^o Excluding women provided with iron and folate supplements * p = <0.05 ** p = <0.01 *** p = <0.005 ^o p = <0.001 ^{**} p = <0.0005 ^{***} p = <0.0001

Table 6.4b. Intra- and inter-individual variance of additional variables for the subset of subjects attending on all occasions and the estimate of intra- and inter- individual variance for the same variables measured for those attending any survey

	serum γ gt concentration	serum folate ^o concentration	plasma β -carotene concentration	plasma ascorbic acid concentration
Subset (n) ^o	100	72	72	49
Inter-individual variance	$F_{20,80} = 22.00^{***}$	$F_{18,54} = 7.68^{***}$	$F_{20,30} = 1.69$	$F_{20,30} = 1.56$
Intra-individual variance (total)	$F_{4,30} = 14.99^{***}$	$F_{3,34} = 9.26^{***}$	$F_{2,30} = 5.2^*$	$F_{2,30} = 4.63^*$
Intra-individual variance (linear)	$F_{1,30} = 9.14^*$	$F_{1,34} = 15.01^{***}$	$F_{1,30} = 1.25$	$F_{1,30} = 4.25^*$
Total sample (n) ^o	229	193	106	106
Inter-individual variance	$F_{69,160} = 58.3^{***}$	$F_{68,125} = 4.43^{***}$	$F_{36,51} = 2.35$	$F_{36,51} = 1.01$
Intra-individual variance (total)	$F_{4,160} = 4.32^*$	$F_{3,125} = 14.7^{***}$	$F_{2,51} = 7.9^{***}$	$F_{2,51} = 4.89^*$
Intra-individual variance (linear)	$F_{1,160} = 11.9^{**}$	$F_{1,125} = 28.9^{***}$	$F_{1,51} = 2.37$	$F_{1,51} = 7.8^{**}$

^o Excluding missing values and relevant results where fasting status was not confirmed (section 4.7.10)

^{*} Excluding women provided with iron and folate supplements * $p < 0.05$ ** $p < 0.01$ *** $p < 0.005$ ^o $p < 0.001$ ** $p < 0.0005$ *** $p < 0.0001$

6.4. Substance use and abuse over the intervention period

The small numbers arising from the sub-classification of the sample demanded careful interpretation of cross-sectional analysis. Of those residing at Minjilang in June 1989, 25 men (75.8%) and 26 women (74.3%) stated that they smoked cigarettes, 24 men (72.7%) and 8 women (22.9%) stated that they drank alcohol when it was available, and 12 men (36.4%) and 13 women (37.1%) stated that they drank kava. All results were confirmed using the method of consensus ranking and were supported by intimate knowledge of individual behaviour gained by residing in the community.

In June 1990, 18 men (81.8%) and 17 women (68.0%) were still smoking cigarettes, 19 men (86.4%) and 8 women (32.0%) stated that they drank alcohol when it was available and 9 men (40.9%) and 6 women (24.0%) drank kava. When cross tabulated against the initial findings, no significant change in the use of any of these three substances was revealed by the chi-square statistical test.

6.5. Change in anthropometric and metabolic parameters over the intervention period

The initial findings of the screening process in June 1989 were used both to identify specific target groups and to provide a focus for subsequent intervention strategies. Results are presented in detail by age and sex groups (Addendum 2, Table A2.5) and, for relevant variables, by age and sex groups excluding diabetics (Addendum 2, Table A2.6). From a seasonal perspective, the findings of the final monitoring process in June 1990 were directly comparable to the initial screening results. These are also presented in detail by age and sex groups (Addendum 2, Table A2.7), and, for those parameters affected by diabetic status, by age and sex groups excluding diabetics (Addendum 2, Table A2.8).

Anthropometric and metabolic results for the subset of subjects participating in surveys in both June 1989 and June 1990 are presented in Table 6.5 and Table 6.6. Anthropometric and metabolic results for the total sample surveyed at each three month period are presented in Table 6.7 to 6.8. The results for the subset of subjects participating in every survey are presented in Table 6.9 and Table 6.10.

Table 6.5. Anthropometric and metabolic data: pre-intervention (June 1989) and post-intervention (June 1990),
Minjilang n=44 repeated pairs^o (mean \pm se)

Variable	n	June 1989	June 1990	t-test (paired)
BMI (kg/m ²)	43	23.9 \pm 0.8	23.6 \pm 0.8	-1.84
Weight (kg)	43	66.1 \pm 2.4	65.4 \pm 2.3	-1.67
Waist:Hip	43	0.89 \pm 0.1	0.87 \pm 0.1	-3.09***
Systolic blood pressure (mm Hg)	43	119.9 \pm 2.3	115.9 \pm 2.0	-2.68**
Diastolic blood pressure (mm Hg)	43	71.1 \pm 1.4	67.7 \pm 1.3	-2.47*
Pulse (beats/minute)	44	80.0 \pm 1.3	78.3 \pm 1.9	-2.0*
Serum cholesterol (mmol/l)	44	5.79 \pm 0.18	5.24 \pm 0.16	-4.97***
Serum HDL-cholesterol (mmol/l)	44	1.12 \pm 0.04	1.01 \pm 0.04	-3.16***
Cholesterol:HDL-cholesterol	44	5.38 \pm 0.24	5.44 \pm 0.24	0.41
Serum triglyceride (mmol/l)	40	1.89 \pm 0.28	1.64 \pm 0.15	-1.00
Fasting insulin (μ u/l)	40	14.6 \pm 1.5	17.7 \pm 2.0	1.60
2hr insulin (μ u/l)	36	50.2 \pm 8.1	40.2 \pm 7.0	-1.49
Fasting glucose (mmol/l)	40	5.90 \pm 0.36	6.09 \pm 0.40	0.95
2hr glucose (mmol/l)	34	8.35 \pm 0.96	8.23 \pm 0.91	-0.28
Serum fructosamine (mmol/l)	40	234.3 \pm 8.6	245.3 \pm 9.2	2.40*
Serum protein (g/l)	44	81.3 \pm 0.6	80.0 \pm 0.9	-1.60
Serum albumin (g/l)	44	42.1 \pm 0.3	41.2 \pm 0.5	-1.70
γ gt (U/l)	44	88.4 \pm 25.1	72.2 \pm 19.4	-2.46*

^o Excluding missing values and relevant results where fasting status was not confirmed (section 4.7.10)

* p = <0.05 ** p = <0.01 *** p = <0.005 † p = <0.001 †† p = <0.0005 ††† p = <0.0001

Table 6.6. Anthropometric and metabolic data: pre-intervention - June 1989; post-intervention - June 1990, (excluding diabetics) n=38 repeated pairs^o (mean \pm se)

Variable	n	June 1989	June 1990	t-test (paired)
BMI (kg/m ²)	37	23.0 \pm 0.82	22.8 \pm 0.81	-1.35
Weight (kg)	38	63.9 \pm 2.4	63.3 \pm 2.4	-1.26
Waist:Hip	37	0.88 \pm 0.01	0.86 \pm 0.01	-2.7**
systolic blood pressure (mm Hg)	37	118.5 \pm 2.0	114.5 \pm 1.9	-2.63*
diastolic blood pressure (mm Hg)	37	70.9 \pm 1.6	67.6 \pm 1.5	-2.13*
Pulse (beats/minute)	38	80.1 \pm 1.5	75.8 \pm 2.1	-2.0*
Serum cholesterol (mmol/l)	38	5.7 \pm 0.19	5.1 \pm 0.16	-4.7***
Serum HDL-cholesterol (mmol/l)	38	1.12 \pm 0.04	1.01 \pm 0.04	-2.96***
Cholesterol:HDL-cholesterol	38	5.26 \pm 0.25	5.30 \pm 0.25	0.22
Serum triglyceride (mmol/l)	34	1.81 \pm 0.32	1.40 \pm 0.14	-1.44
Fasting insulin (mu/l)	34	14.2 \pm 1.7	16.4 \pm 2.2	1.05
2hr insulin (mu/l)	30	48.1 \pm 9.0	42.1 \pm 8.3	-0.82
Fasting glucose (mmol/l)	34	5.1 \pm 0.1	5.2 \pm 0.1	1.09
2hr glucose (mmol/l)	28	6.0 \pm 0.3	6.2 \pm 0.4	0.50

^o Excluding missing values and relevant results where fasting status was not confirmed (section 4.7.10)

* p = <0.05 ** p = <0.01 *** p = <0.005 * p = <0.001 ** p = <0.0005 *** p = <0.0001

Table 6.7. Anthropometric and metabolic data for those participating in any survey^o (mean ± se)

Variable	n	June 1989	n	Sept 1989	n	Dec 1989	n	March 1990	n	June 1990	F-value Btwn.Grp.	F-value Linearity
Sex ratio (F:M)	68	0.50±0.02	45	0.51±0.03	51	0.51±0.03	32	0.53±0.04	47	0.51±0.03	0.15	0.10
Age (years)	68	37.9±1.8	45	41.1±2.3	51	40.8±2.1	32	38.1±2.3	47	36.8±2.1	0.80	0.32
BMI (kg/m ²)	68	23.3±0.6	45	23.6±0.8	50	23.1±0.7	29	24.6±0.8	46	23.5±0.8	0.46*	0.24*
Waist:Hip ratio	68	0.89±0.01	45	0.88±0.01	50	0.90±0.01	28	0.88±0.01	46	0.87±0.01	1.42	3.15
Mid-arm circumference (cm)	68	26.7±0.5	45	27.8±0.6	51	28.2±0.6	29	28.7±0.6	47	27.9±0.6	1.91	4.30*
Systolic blood pressure (mm Hg)	65	123.0±2.1	45	121.8±2.4	51	115.4±2.3	30	111.4±2.6	46	115.2±1.9	4.39***	13.06**
Diastolic blood pressure (mm Hg)	65	72.2±1.2	45	70.4±1.3	51	66.9±1.2	30	63.8±1.6	46	67.4±1.2	6.13***	16.29***
Pulse (beats/minute)	65	81.6±1.3	43	75.3±1.6	51	77.2±1.8	30	76.0±1.9	47	76.8±1.9	2.54*	4.02*
Serum cholesterol (mmol/l)	68	5.93±0.15	45	5.70±0.20	51	5.80±0.20	32	5.74±0.19	47	5.20±0.15	2.89*	8.26***
Serum HDL-cholesterol (mmol/l)	68	1.13±0.03	45	1.05±0.03	51	1.07±0.04	32	1.12±0.04	47	1.01±0.03	2.34	4.20*
Cholesterol:HDL-cholesterol	68	5.46±0.19	45	5.57±0.20	51	5.60±0.20	32	5.29±0.25	47	5.40±0.23	0.34	0.23
Serum triglyceride (mmol/l)	67	1.81±0.17	45	1.56±0.10	50	1.66±0.11	31	1.85±0.14	43	1.62±0.14	0.68	0.28
Fasting insulin (mu/l)	50	16.5±2.0	-	-	-	-	-	-	42	17.1±2.0	-	1.02
2hr. insulin (mu/l)	65	46.5±5.5	-	-	-	-	-	-	38	39.6±6.7	-	0.67
Fasting glucose (mmol/l)	67	5.61±0.2	-	-	-	-	-	-	43	6.0±0.4	-	0.25
2hr glucose (mmol/l)	66	7.09±0.6	-	-	-	-	-	-	39	7.9±0.8	-	0.56
Serum fructosamine (mmol/l)	50	233±6.9	-	-	47	231±11	-	-	42	243.6±8.8	0.53	0.67
γgt (U/l)	68	91.9±18.1	45	59.4±9.8	51	60.4±7.7	32	54.9±5.6	47	69.2±18.2	1.14'***	1.64**

^o Excluding missing values and relevant results where fasting status was not confirmed (section 4.7.10)

* p = <0.05 ** p = <0.01 *** p = <0.005 † p = <0.001 †† p = <0.0005 ††† p = <0.0001

* Using appropriate test of greater sensitivity significant change was subsequently apparent (section 6.5.1; Table 6.4)

** Using appropriate test of greater sensitivity significant change was subsequently apparent (section 6.5.7; Table 6.4)

' Because of the skewed distribution and heterogeneity between variances the fact that there is not a difference between the means may be misleading

Table 6.8. Anthropometric and metabolic data for all those participating in any survey (excluding diabetics)^o (mean ± se)

Variable	n	June 1989	n	Sept 1989	n	Dec 1989	n	March 1990	n	June 1990	F-value Btwn.grp.	F-value Linearity
Age (years)	62	37.1±1.9	39	40.2±2.6	45	39.9±2.4	29	37.5±2.4	42	35.7±2.2	0.72	0.30
BMI (kg/m ²)	62	22.7±0.6	39	22.7±0.8	44	22.3±0.7	26	24.0±0.8	41	22.8±0.7	0.56	0.17
Waist:Hip	62	0.89±0.01	39	0.87±0.01	44	0.89±0.01	25	0.87±0.01	41	0.86±0.01	1.5	3.1
Serum cholesterol (mmol/l)	62	5.90±0.16	39	5.68±0.19	45	5.76±0.17	29	5.63±0.20	42	5.06±0.15	3.7 ^{***}	11.4 [†]
Serum HDL-cholesterol (mmol/l)	62	1.13±0.03	39	1.06±0.03	45	1.08±0.04	29	1.13±0.04	42	1.0±0.03	2.6 [*]	4.9 [*]
Cholesterol:HDL-cholesterol	62	5.4±0.2	39	5.5±0.2	45	5.5±0.2	29	5.1±0.3	42	5.3±0.2	0.44	0.63
Serum triglyceride (mmol/l)	61	1.79±0.19	39	1.43±0.10	44	1.55±0.10	28	1.70±0.13	38	1.45±0.14	2.6	4.9 [*]
Fasting insulin (mu/l)	44	16.5±2.2	-	-	-	-	-	-	37	15.7±2.0	-	1.21
2hr insulin (mu/l)	59	46.4±5.9	-	-	-	-	-	-	33	41.7±7.6	-	0.60
Fasting glucose (mmol/l)	61	5.1±0.1	-	-	-	-	-	-	38	5.2±0.1	-	0.96
2hr glucose (mmol/l)	60	5.8±0.2	-	-	-	-	-	-	34	6.2±0.3	-	0.44

^o Excluding missing values and relevant results where fasting status was not confirmed (section 4.7.10)

* p = <0.05 ** p = <0.01 *** p = <0.005 † p = <0.001 †† p = <0.0005 ††† p = <0.0001

Table 6.9. Anthropometric and metabolic data for those participating in every survey^o (mean \pm se)

Variable	n	June 1989	n	Sept 1989	n	Dec 1989	n	March 1990	n	June 1990	F-value Btwn.grp.	F-value Linearity
BMI (kg/m ²)	21	24.5 \pm 1.2	21	24.2 \pm 1.1	20	24.0 \pm 1.1	18	25.4 \pm 1.0	20	24.7 \pm 1.0	0.24	0.18
Weight (kg)	21	66.5 \pm 3.2	21	65.6 \pm 2.9	20	64.8 \pm 3.1	18	69.1 \pm 2.7	20	66.9 \pm 2.7	0.28	0.19
Waist:Hip	21	0.90 \pm 0.01	21	0.86 \pm 0.01	20	0.90 \pm 0.01	17	0.90 \pm 0.01	20	0.86 \pm 0.01	0.61	0.52
Mid-arm circumference (cm)	21	26.9 \pm 0.8	21	27.7 \pm 0.7	21	28.9 \pm 0.9	18	28.8 \pm 0.9	21	28.6 \pm 0.8	1.0	3.1
Systolic blood pressure (mm Hg)	21	121.2 \pm 3.8	21	119.8 \pm 2.5	21	114 \pm 3.0	19	110.2 \pm 3.2	21	117.1 \pm 2.9	2.0	3.1
Diastolic blood pressure (mm Hg)	21	72.0 \pm 1.9	21	69.0 \pm 1.6	21	66.3 \pm 1.7	19	62.4 \pm 2.1	21	68.3 \pm 1.8	3.8**	5.9*
Pulse (beats/minute)	21	79.9 \pm 1.9	21	71.1 \pm 2.0	21	74.0 \pm 2.1	19	74.7 \pm 2.0	21	73.7 \pm 1.8	2.7*	2.1
Serum cholesterol (mmol/l)	21	6.11 \pm 0.20	21	5.70 \pm 0.23	21	5.95 \pm 0.23	21	5.93 \pm 0.25	21	5.57 \pm 0.20	0.94	1.4
Serum HDL-cholesterol (mmol/l)	21	1.09 \pm 0.05	21	1.04 \pm 0.04	21	1.03 \pm 0.05	21	1.10 \pm 0.04	21	0.95 \pm 0.05	1.7	2.4
Cholesterol:HDL-cholesterol	21	5.88 \pm 0.36	21	5.62 \pm 0.29	21	5.98 \pm 0.30	21	5.53 \pm 0.31	21	6.05 \pm 0.31	0.53	0.06
Serum triglyceride (mmol/l)	21	1.49 \pm 0.12	21	1.41 \pm 0.12	21	1.61 \pm 0.16	21	1.93 \pm 0.17	19	1.76 \pm 0.23	1.7	4.3*
Fasting insulin (μ U/l)	21	16.5 \pm 2.0	-	-	-	-	-	-	19	19.4 \pm 2.9	-	0.48
2hr insulin (μ U/l)	21	52.8 \pm 10.6	-	-	-	-	-	-	19	39.7 \pm 7.8	-	1.85
Fasting glucose (mmol/l)	21	6.1 \pm 0.5	-	-	-	-	-	-	19	6.1 \pm 0.5	-	0.98
2hr glucose (mmol/l)	20	8.5 \pm 1.3	-	-	-	-	-	-	19	8.2 \pm 1.2	-	1.08
Serum fructosamine (mmol/l)	21	237 \pm 14	-	-	21	236 \pm 22	-	-	19	246 \pm 15	0.10	0.13
γ gt (U/l)	21	61.4 \pm 9.6	21	43.1 \pm 6.10	21	44.1 \pm 5.3	21	47.8 \pm 5.8	21	44.7 \pm 6.6	1.2	1.8

^o Excluding missing values and relevant results where fasting status was not confirmed (section 4.7.10)

* p = <0.05 ** p = <0.01 *** p = <0.005 † p = <0.001 †† p = <0.0005 ††† p = <0.0001

Table 6.10. Anthropometric and metabolic data for those participating in all surveys (excluding diabetics)^o (mean ± se)

Variable	n	June 1989	n	Sept 1989	n	Dec 1989	n	March 1990	n	June 1990	F-value Btwn.Grp.	F-value Linearity
BMI (kg/m ²)	18	23.4±1.0	18	23.2±1.0	17	22.9±1.0	15	24.4±0.8	17	23.6±0.8	0.34	0.26
Waist:Hip	18	0.87±0.02	18	0.85±0.01	17	0.87±0.01	14	0.87±0.01	17	0.85±0.01	0.70	0.47
Mid-arm circumference (cm)	18	26.4±0.8	18	27.1±0.7	18	27.9±0.8	15	27.8±0.7	17	0.85±0.01	0.69	1.9
Serum cholesterol (mmol/l)	18	6.0±0.2	18	5.6±0.2	18	5.7±0.2	18	5.8±0.3	18	5.4±0.2	0.88	1.8
Serum HDL-cholesterol (mmol/l)	18	1.09±0.06	18	1.05±0.05	18	1.03±0.05	18	1.11±0.03	18	0.94±0.04	1.8	2.6
Cholesterol:HDL-cholesterol	18	5.8±0.4	18	5.5±0.3	18	5.8±0.3	18	5.3±0.3	18	5.9±0.3	0.57	0.00
Serum triglyceride (mmol/l)	18	1.41±0.12	18	1.34±0.14	18	1.47±0.14	17	1.68±0.13	16	1.57±0.23	0.76	1.81
Fasting insulin (mu/l)	18	16.4±2.2	-	-	-	-	-	-	16	18.6±3.3	-	0.44
2hr insulin (mu/l)	18	53.1±12.2	-	-	-	-	-	-	16	41.6±9.1	-	1.80
Fasting glucose (mmol/l)	18	5.31±0.2	-	-	-	-	-	-	16	5.26±0.1	-	4.02
2hr glucose (mmol/l)	17	6.3±0.5	-	-	-	-	-	-	16	6.3±0.4	-	1.56

^o Excluding missing values and relevant results where fasting status was not confirmed (section 4.7.10)

* p = <0.05 ** p = <0.01 *** p = <0.005 † p = <0.001 †† p = <0.0005 ††† p = <0.0001

6.5.1. Body mass index

In June 1989 the mean BMI was $23.3 \pm 0.6 \text{ kg/m}^2$, and tended to be highest in younger men and older women, and lowest in younger women (Addendum 2, Table A2.5). There was a strong association between status of glucose tolerance and BMI (Table 6.12), and after excluding diabetics, BMI still tended to be highest in younger men (Addendum 2, Table A2.6). These observations were further investigated in section 6.6.1. Relative to the range of values considered to represent various BMI categories in Caucasian subjects², 14.7% of adults at Minjilang were 'very underweight' (BMI < 18 kg/m²), 16.2% were 'underweight' (BMI 18 - 19.9 kg/m²), 33.8% were 'acceptable' (BMI 20 - 24.9 kg/m²), 23.5% were 'overweight' (BMI 25 - 30 kg/m²), and 11.8% were 'obese' (BMI \geq 30 kg/m²). Of those very underweight by Caucasian standards five were young women, three were elderly women and two were elderly men. Of those obese by Caucasian standards, all were over the age of 35 years; five were women and three were men.

In June 1990, relative to the range of values considered to represent various BMI categories in Caucasian subjects, 10.9% of adults at Minjilang were 'very underweight', 17.4% were 'underweight', 43.4% were 'acceptable', 17.4% were 'overweight' and 10.8% were 'obese'. When these categories were cross tabulated with the original results, there was no significant change in BMI groupings for all individuals weighed and measured over the entire intervention period ($\chi^2_{16}=8.43$), nor for all individuals weighed and measured during the initial and final surveys ($\chi^2_4=1.64$).

In order to investigate changes in the relatively small numbers of specific individuals participating in both June 1989 and June 1990, BMI groupings were reduced to only three categories; 'underweight' (BMI < 20 kg/m²), 'acceptable BMI' (BMI 20 - 25 kg/m²) and 'overweight' (\geq 25 kg/m²). Using these categories there was a change in BMI which bordered on statistical significance ($\chi^2_4=3.3$, $p=0.05$); those individuals initially 'overweight' tended to lose weight, whereas those individuals initially 'underweight' tended to increase weight (Figure 6.1).

Initial direct analysis of variance suggested that mean BMI did not significantly change over the intervention period, for the whole population (Table 6.7), for non-diabetics (Table 6.8) or for those who were monitored at each survey (Table 6.9). Due to a slight

² Caucasian BMI standards may be inappropriate for Aboriginal groups (see section 8.2.2).

change in the standard deviation of the mean BMI over the intervention period, analysis of variance in the logarithm of BMI was also investigated. Results revealed no significant change in the magnitude of the F value either between surveys ($F_{4,233}=0.06$, ns) or linearly over time ($F_{1,234}=0.38$, ns). Mean BMI was not significantly different for any subjects, nor non-diabetic subjects, participating in both pre-intervention and post-intervention surveys (Table 6.5, Table 6.6). However when subjects were separated into age and sex categories (Addendum 2, Table A2.9), there was a small but highly significant decrease in the mean BMI of older women participating in both pre-intervention and post-intervention surveys ($n=13$, $t=-3.81$, $p<0.001$). Moreover, from a statistical perspective, direct comparison of the between survey results did not account for the different attendance of individuals at each three month period. Using GLIM, a more sensitive analysis of variance was conducted after controlling for inter-individual variance, which disclosed a small but statistically significant reduction in BMI over the intervention period ($F_{4,159}=25.6$, $p<0.0001$) including a significant linear reduction ($F_{1,159}=17.5$, $p<0.0005$) (Table 6.4). The mean adjusted BMI for each survey was consecutively 22.7, 22.4, 22.2, 22.5 and 22.4 kg/m^2 , suggesting that BMI reduced particularly during the first six months of the intervention period.

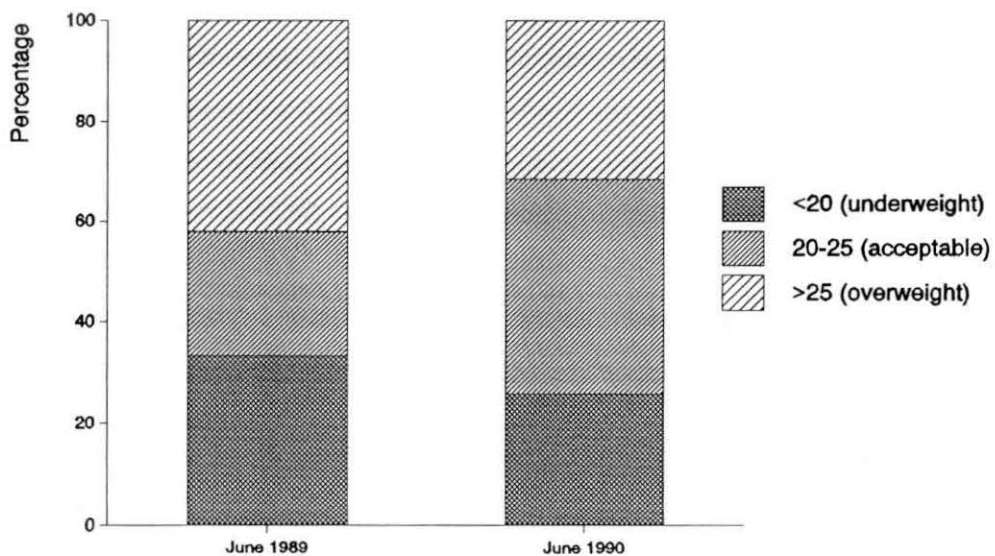


Figure 6.1. Distribution of body mass index, pre-intervention (June 1989) and post-intervention (June 1990) ($n=43$)

6.5.2. Waist:hip and mid-arm circumference

In June 1989, the mean waist:hip circumference of men (0.92 ± 0.01) was acceptable relative to the Caucasian reference standard of less than 1.0, however the mean waist:hip circumference of women (0.86 ± 0.01) was high relative to the Caucasian reference standard of less than 0.8³. Waist:hip circumference tended to be highest in older men, but was much higher in older women than younger women (Addendum 2, Table A2.5). 12.1% of men (4) and 77.1% of women (27) had a high waist hip circumference ratio relative to the respective Caucasian standards. Waist:hip circumference tended to be higher in those individuals with impaired glucose tolerance and diabetes (Table 6.13).

In June 1990, 70.8% (17) of women had a high waist:hip circumference relative to the respective Caucasian standards. There was a trend for the waist:hip circumference of men to reduce to an acceptable level over the intervention period ($\chi^2_1 = 2.86$, $p = 0.08$); none of the men participating in the final survey had a waist:hip ratio greater than 1.0.

Analysis of variance suggested that the mean ratio of waist to hip circumference did not significantly change over the intervention period, for the whole population (Table 6.7), for non-diabetics (Table 6.8) or for those who were monitored at each survey (Table 6.9). However waist:hip circumference did significantly decrease in all those individuals (Table 6.5) and non-diabetic subjects (Table 6.6) participating in both pre-intervention and post-intervention surveys. The ratio of waist to hip circumference was significantly correlated with BMI over all surveys ($r = 0.60$ $p < 0.001$ for men and $r = 0.39$ $p < 0.001$ for women). However, measurement of waist and hip circumference was relatively unreliable (Table 6.2) and was accordingly less emphasised than BMI.

There was a small linear increase in mid arm circumference (Table 6.7), but this effect was no longer significant when only those individuals participating in all surveys were included in analysis (Table 6.9).

6.5.3. Glucose tolerance

In the initial screening 55 (80.9%) of the 68 subjects screened had normal glucose tolerance, 6 (8.8%) had impaired glucose tolerance (two-hour plasma glucose ≥ 7.8

³ Again, caucasian standards may be inappropriate for Aboriginal groups, particularly women

mmol/l) and 7 (10.3%) had diabetes (two-hour plasma glucose \geq 11.1 mmol/l) (Table 6.11). Of those identified as diabetic, two subjects had not previously been diagnosed. None of those subjects exhibiting the characteristics of impaired glucose tolerance were aware of the condition at the time of screening. No individual less than 35 years of age had diabetes, and only one young woman had impaired glucose tolerance (Table 6.11). More older women had impaired glucose tolerance than older men (Table 6.11). There was a strong relationship between glucose tolerance and BMI, with diabetic subjects having the highest BMI (Table 6.12). Diabetics also had higher concentrations of both fasting serum triglyceride (Table 6.14) and fructosamine (Table 6.15). These observations were further investigated in section 6.6.⁴ In June 1989, those with impaired glucose tolerance tended to have the highest fasting insulin levels, however the effect was not statistically significant (Table 6.16). Two-hour serum insulin levels were significantly higher in those with impaired glucose tolerance (Table 6.17).

Table 6.11. Prevalence of impaired glucose tolerance by age and sex group, June 1989

	Men		Women	
	<35 yrs	\geq 35 yrs	<35 yrs	\geq 35 yrs
	n (%)	n (%)	n (%)	n (%)
Normal glucose tolerance	16 (100)	13 (76.5)	13 (92.9)	13 (61.9)
Impaired glucose tolerance	-	1 (5.9)	1 (7.1)	4 (19.0)
Diabetes	-	3 (17.6)	-	4 (19.0)

Analysis of variance suggested that glucose tolerance did not significantly change over the intervention period in the whole population (Table 6.7), when diabetics were excluded from analysis (Table 6.8), or in those subjects or non-diabetics who participated in all surveys (Table 6.9, Table 6.10).

There was no significant change in either fasting plasma glucose, two hour plasma glucose, fasting serum insulin or two hour plasma insulin concentrations in those individuals (Table 6.5), or non-diabetic subjects (Table 6.6) participating in both pre-

⁴ Heterogenous variances in glucose tolerance data suggests that there was some change between pre- and post-intervention data; this issue will be explored in greater detail at a later date.

intervention and post-intervention surveys. When subjects participating in both pre-intervention and post-intervention surveys were separated into age and sex categories (Addendum 2, Table A2.9), over the intervention period there was a significant increase in fasting plasma glucose concentration in young women ($n=8$, $t=2.37$, $p<0.05$) and a significant increase in two hour plasma glucose concentration in young men ($n=6$, $t=3.01$, $p<0.05$). However there was a small but significant decrease in two hour plasma glucose concentration in older women ($n=11$, $t=-2.40$, $p<0.05$). This may be due to type one error; however the BMI of older women also appeared to decrease (section 6.5.1) which suggested that glucose tolerance may have improved in this group over the period of intervention.

Analysis of variance suggested that fasting serum fructosamine concentration did not change significantly over the intervention period in the whole population (Table 6.7), or in those individuals participating in all surveys (Table 6.9). Because of the skewed distribution of fasting serum fructosamine concentration, analysis of variance in the logarithm of this variable was also investigated. Results revealed a slight increase in the magnitude of the F value, but not to the level of significance, either between surveys ($F_{2,136}=1.18$), or linearly over time ($F_{1,137}=0.89$). There was a small significant difference between fasting serum fructosamine concentration in non-diabetic subjects over the intervention period, with results tending to be lowest in December 1989 and highest in June 1990 (Table 6.8, Table 6.10). The increase in fasting serum fructosamine concentration was significant in those individuals participating in both pre-intervention and post-intervention surveys (Table 6.5).

Table 6.12. Body mass index by glucose tolerance group

	n	June 1989	n	June 1990
Normal glucose tolerance	55	22.2±0.63	35	21.9±0.74
Impaired glucose tolerance	6	26.7±2.3	6	27.4±1.2
Diabetes	7	29.1±1.7	5	29.9±1.9
F-value (Btwn.Grp.)	8.3 ($p<0.001$)		10.5 ($p<0.0005$)	
F-value (linear)	16.3 ($p<0.0005$)		20.4 ($p<0.0001$)	

Table 6.13. Ratio of waist to hip circumference by glucose tolerance group

	n	June 1989	n	June 1990
Normal glucose tolerance	55	0.88 ± 0.01	35	0.85 ± 0.01
Impaired glucose tolerance	6	0.91 ± 0.04	6	0.87 ± 0.02
Diabetes	7	0.96 ± 0.02	5	0.95 ± 0.02
F-value (Btwn.Grp.)	3.43 (p < 0.05)		5.32 (p < 0.01)	
F-value (linear)	6.73 (p < 0.05)		9.28 (p < 0.005)	

Table 6.14. Fasting serum triglyceride concentration by glucose tolerance group

	n	June 1989	n	June 1990
Normal glucose tolerance	53	1.59 ± 0.09	32	1.34 ± 0.14
Impaired glucose tolerance	6	1.67 ± 0.22	6	2.03 ± 0.42
Diabetes	7	2.21 ± 0.23	5	2.88 ± 0.21
F-value (Btwn.Grp.)	3.03 (ns)		9.00 (p < 0.001)	
F-value (linear)	5.39 (p < 0.05)		18.0 (p < 0.001)	

Table 6.15. Fasting serum fructosamine concentration by glucose tolerance group

	n	June 1989	n	June 1990
Normal glucose tolerance	38	217 ± 2.3	31	226 ± 3.6
Impaired glucose tolerance	5	210 ± 4.4	6	227 ± 4.0
Diabetes	7	330 ± 26.9	5	372 ± 37.9
F-value (Btwn.Grp.)	47.9 (p < 0.001)		43.7 (p < 0.001)	
F-value (linear)	75.0 (p < 0.001)		66.3 (p < 0.001)	

Table 6.16. Fasting serum insulin concentration by glucose tolerance group

	n	June 1989	n	June 1990
Normal glucose tolerance	38	15.3±2.5	31	14.6±2.1
Impaired glucose tolerance	5	24.6±4.8	6	21.5±6.2
Diabetes	7	17.1±1.1	5	27.6±5.0
F-value (Btwn.Grp.)	0.98 (ns)		2.91 (ns)	
F-value (linear)	0.48 (ns)		5.81 (p < 0.05)	

Table 6.17. Two hour serum insulin concentration by glucose tolerance group

	n	June 1989	n	June 1990
Normal glucose tolerance	52	37.9±5.3	27	33.7±5.8
Impaired glucose tolerance	6	101.5±19.7	6	77.5±30.6
Diabetes	7	62.9±17.2	5	26.2±4.6
F-value (Btwn.Grp.)	7.4 (p < 0.01)		3.5 (p < 0.05)	
F-value (linear)	6.9 (p < 0.05)		0.3 (ns)	

6.5.4. Fasting serum triglyceride concentration

In June 1989 the mean fasting serum triglyceride concentration (1.81 ± 0.17 mmol/l) was high relative to the Caucasian reference range (< 1.8 mmol/l); 38.9% of those screened had an elevated fasting serum triglyceride concentration relative to this reference standard. Fasting serum triglyceride levels tended to be highest in men and older subjects (Addendum 2, Table A2.5), and this trend persisted when diabetic subjects were excluded from analysis (Addendum 2, Table A2.6). Serum triglyceride levels were significantly related to status of glucose tolerance and highest in diabetic subjects (Table 6.14). These observations were further investigated in section 6.6.6.

Analysis of variance suggested that fasting serum triglyceride concentration did not significantly change over the intervention period in the whole population (Table 6.7). Due to fluctuations in the standard deviation of the mean concentrations of fasting serum triglyceride, analysis of variance in the logarithm of this variable was also investigated. Results revealed a slight increase in the magnitude of the F value, but not to the level of statistical significance, either between surveys ($F_{4,231}=1.19$) or linearly over time ($F_{1,234}=1.99$). There was a significant linear decrease in serum triglyceride over the intervention period in non-diabetics (Table 6.8). Conversely, there was a significant linear increase in fasting serum triglyceride concentration in those who were monitored at each survey (Table 6.9), although the increase was less systematic when diabetics were excluded from analysis (Table 6.10). These results suggest that there were differences in change in fasting serum triglyceride status between those regularly attending and other subjects, but more particularly between diabetic and non-diabetic subjects. Serum triglyceride concentration tended to increase over the intervention period in those with diabetes and in some individuals with impaired glucose tolerance, but tended to decrease in those with normal glucose tolerance (Table 6.14). It should also be noted that the initial fasting serum triglyceride concentration tended to be lower in those non-diabetics who were ultimately to participate in all surveys. However, as previously demonstrated (section 6.1), this difference was not statistically significant.

Although there was a trend for fasting serum triglyceride concentration to decrease in those individuals participating in both pre-intervention and post-intervention surveys, the results were not statistically significant (Table 6.5, Table 6.6). Neither was there a significant post-intervention reduction in fasting serum triglyceride concentration for any age and sex category (Addendum 2, Table A2.9).

From a statistical perspective, direct comparison of the between survey results did not account for the different attendance of individuals at each three month period. Using GLIM, a more sensitive analysis of variance was conducted after controlling for inter-individual variance. Even so the reduction in fasting serum triglyceride concentration over the intervention period remained insignificant (Table 6.2). After controlling for inter-individual variance the mean adjusted fasting serum triglyceride concentration for each survey was 1.81, 1.48, 1.53, 1.66 and 1.49 mmol/l.

6.5.5. Serum cholesterol concentration

In June 1989 the mean serum cholesterol concentration (5.93 ± 0.15 mmol/l) was high relative to the Caucasian reference range (< 5.5 mmol/l); 32.4% of those screened had acceptable serum cholesterol levels, 29.4% had high levels (5.5-6.4 mmol/l) and 38.2% had very high levels (> 6.5 mmol/l). Serum cholesterol levels tended to be lowest in young women, but similar in other age and sex groups (Addendum 2, Table A2.5). Mean HDL-cholesterol concentration of men and women was 1.14 ± 0.04 mmol/l and 1.12 ± 0.04 mmol/l respectively; 6.1% of men and 37.1% of women had low levels relative to the accepted Caucasian standards (0.9-1.5 mmol/l and 1.1-1.9 mmol/l respectively). As HDL-cholesterol levels tend to be related to the concentration of total serum cholesterol, the ratio of total cholesterol to HDL-cholesterol (the cholesterol ratio) was also investigated. The mean cholesterol ratio (5.47 ± 0.19) in both sexes (5.56 ± 0.28 for men and 5.38 ± 0.27 for women) was high relative to the recommended ratio of < 4.5 , and 72.1% of subjects had an elevated ratio relative to the recommended level. Neither total serum cholesterol concentration, HDL-cholesterol, nor cholesterol ratio were significantly different when diabetics were excluded from analysis (Addendum 2, Table A2.6). The relationship between cholesterol concentrations and other variables was further investigated in section 6.6.4.

Analysis of variance suggested a marked linear decrease in serum cholesterol concentration, measured at three month intervals over the twelve month intervention period (Table 6.7). The effect was slightly stronger when diabetic subjects were excluded from analysis (Table 6.8), although the effect was no longer statistically significant when the relatively smaller number of individuals participating in all five surveys was considered (Table 6.9, Table 6.10). In June 1990, the mean total serum cholesterol concentration of the adult population at Minjilang had reduced by 12.3%, and by 14.2% in non-diabetics. The reduction in serum cholesterol concentration was highly significant in those individuals participating in both pre-intervention and post-intervention surveys (Table 6.5) and similar in non-diabetic subjects (Table 6.6). When subjects participating in both pre-intervention and post-intervention surveys were separated into age and sex categories (Addendum 2, Table A2.9), there was a highly significant decrease in total serum cholesterol concentration in both older men ($n=11$, $t=-3.56$, $p<0.005$) and older women ($n=13$, $t=-3.61$, $p<0.001$). In June 1990, relative to the respective Caucasian reference range, 29 subjects (55.3%) had acceptable cholesterol levels, 14 subjects (29.8%) had persistently very high serum cholesterol concentration, and seven subjects

(14.9%) had persistently very high serum cholesterol concentration (Figure 6.2)⁵. Compared with the initial cholesterol groupings, there was a significant increase in the proportion of individuals with acceptable concentration of serum cholesterol in June 1990 ($\chi^2=10.1$, $p<0.01$).

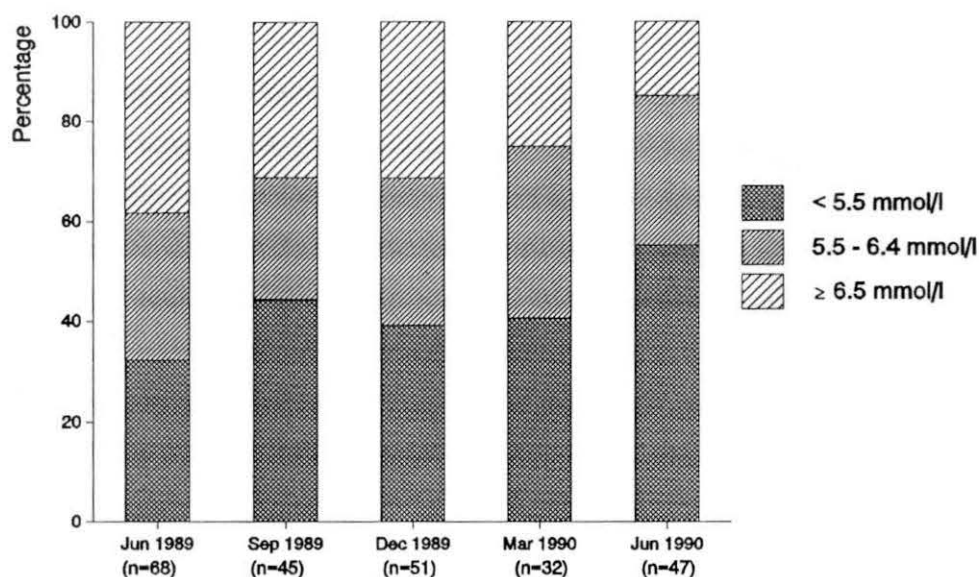


Figure 6.2. Distribution of serum cholesterol concentration

Although analysis of variance indicated that there was a significant reduction in serum HDL-cholesterol concentration over the intervention period, there was no significant increase in the ratio of total serum cholesterol concentration to HDL-cholesterol concentration (Table 6.7, Table 6.8). Neither HDL-cholesterol concentration, nor the ratio of total cholesterol to HDL-cholesterol concentration, changed significantly throughout the intervention period in those individuals participating in all five surveys (Table 6.9). The ratio of total cholesterol to HDL-cholesterol concentration also did not change significantly in those individuals participating in both pre-intervention and post-intervention surveys (Table 6.5).

⁵ Three subjects in each of the latter categories had serum cholesterol levels which had reduced to the lowest value in each category.

6.5.6. Blood pressure and pulse

All subjects had diastolic blood pressure below 95 mm Hg. Young women tended to have lower blood pressure (Addendum 2, Table A2.5). There was no significant difference in either diastolic ($F_{1,67}=0.10$) or systolic ($F_{1,67}=0.24$) blood pressure between diabetic and non-diabetic subjects. The relationship between blood pressure and other variables was further investigated in section 6.6.2 and section 6.6.3.

A highly significant linear decrease in both diastolic and systolic blood pressure was observed over the intervention period (Table 6.7)⁶. This significant effect persisted for diastolic blood pressure in those individuals participating in all surveys (Table 6.9). Systolic and diastolic blood pressure both decreased significantly in all those individuals participating in both pre-intervention and post-intervention surveys (Table 6.5). At no time over the intervention period did any individual have a diastolic blood pressure > 95 mm Hg. One of the original three subjects on anti-hypertensive therapy discontinued medication in December 1989 following weight loss; all subsequent blood pressure readings were acceptable.

There was also a small but statistically significant decrease in pulse over the intervention period (Table 6.7, Table 6.9, Table 6.5).

6.5.7. Gamma-glutamyl transferase

In June 1989 the mean serum gamma-glutamyl transferase (γ gt) concentration (91.9 ± 18.1 U/l) was high relative to the Caucasian reference range (<60 U/l). Of the original 68 subjects, 60.3% had acceptable levels and 39.7% had high levels relative to this reference range. Serum γ gt levels tended to be lowest in young women, and highest in young men (Addendum 2, Table A2.5). These observations were further investigated in section 6.6.9.

There was a tendency for serum concentration of γ gt to decrease over the twelve month intervention period, although analysis of variance suggested that the effect was not significant (Table 6.7). Because of the skewed distribution of the serum γ gt concentration, analysis of variance in the logarithm of this variable was also investigated. Results revealed a slight increase in the magnitude of the F value, but not to the level of statistical

⁶ The potential effect of 'familiarisation' is discussed in section 8.2.3.3.

significance, either between surveys ($F_{4,238}=1.18$) or linearly over time ($F_{1,235}=2.19$). However, in June 1990, the mean serum γ gt concentration of the adult population living at Minjilang had fallen by 24.7%, and there was a significant reduction in serum γ gt concentration in those individuals participating in both pre-intervention and post-intervention surveys (Table 6.5). When subjects participating in both pre-intervention and post-intervention surveys were separated into age and sex categories (Addendum 2, Table A2.9), there was a highly significant decrease in serum γ gt concentration in older women ($n=13$, $t=-3.82$, $p<0.001$). Moreover, from a statistical perspective, direct comparison of the between survey results did not account for the different attendance of individuals at each three month period. Using GLIM, a more sensitive analysis of variance was conducted after controlling for inter-individual variance, which disclosed a small but statistically significant reduction in serum γ gt concentration over the intervention period ($F_{4,160}=4.3$, $p<0.05$) including a significant linear reduction ($F_{1,160}=11.9$, $p<0.0005$) (Table 6.4). In June 1990, relative to the Caucasian reference range, 34 subjects (72.3%) had acceptable γ gt levels and 13 subjects (27.7%) had a persistently high serum γ gt concentration (Figure 6.3). Compared with the initial γ gt groupings, there was no significant change in the proportion of individuals with acceptable concentration of serum γ gt in June 1990 ($\chi^2_1=1.8$).

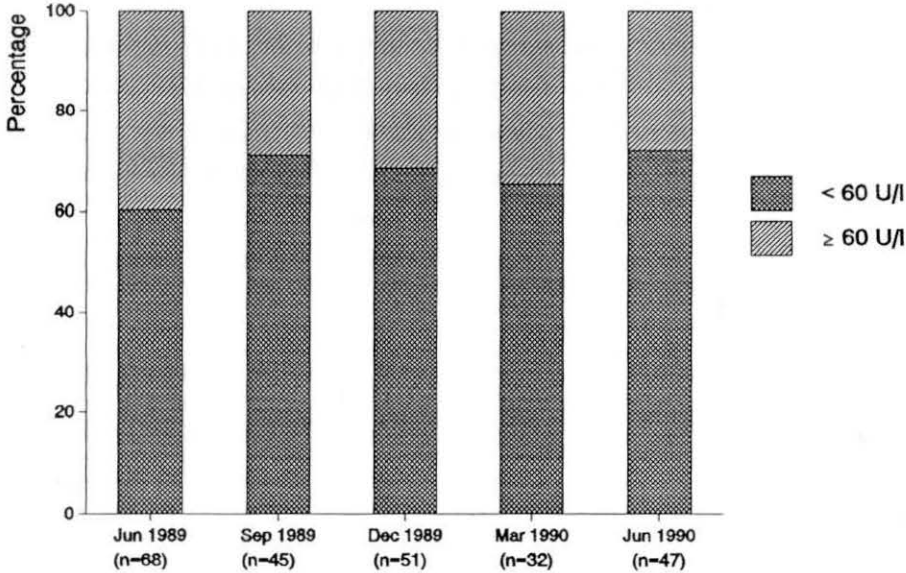


Figure 6.3. Distribution of serum γ gt concentration

6.6. The relationship between anthropometric and metabolic variables at each survey

Analysis of inter-individual variance of variables at each survey was conducted in order to reveal associated conditions. Potentially confounding factors relevant to the comparison of change in dietary intake and nutritional status addressed in analysis of intra-individual variance of biological variables (Chapter 7), were also identified.

6.6.1. Analysis of inter-individual variance of body mass index

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in BMI in non-diabetic subjects, was investigated by analysis of variance over the intervention period (Addendum 2, Table A2.10). 98.8% of the observed variance in BMI was explained, 97.8% by inter-individual and 1.0% by intra-individual variation. Survey number as an ordinal (linear) variable ($F_{1,125}=1.00$, ns) explained only 1.0% of the variance in BMI explained by survey number as a factor ($F_{3,125}=33.5$, $p<0.001$); thus there was no continuing and systematic change in BMI over the period of the intervention.

The relationship between BMI and gender, age, status of cigarette smoking, alcohol and kava consumption at each three month period, was investigated by analysis of inter-individual variance (Addendum 2, Table A2.10). After controlling for survey number, (approximating intra-individual change over time), BMI was higher in men than women ($F_{1,125}=30.80$, $p<0.001$), and was positively correlated with age ($F_{1,125}=4.80$, $p<0.05$), fasting serum triglyceride concentration ($F_{1,125}=188.0$, $p<0.001$) and alcohol consumption ($F_{1,125}=250.0$, $p<0.001$). BMI was negatively associated with cigarette smoking ($F_{1,125}=1287$, $p<0.001$) and kava consumption ($F_{1,125}=817.9$, $p<0.001$). There was a significant interactive effect between gender and both alcohol ($F_{1,125}=250.0$, $p<0.001$) and cigarette smoking ($F_{1,125}=180.4$, $p<0.001$), and age and both triglyceride concentration ($F_{1,125}=75.7$, $p<0.001$) and alcohol consumption ($F_{1,125}=255.1$, $p<0.001$), with BMI being relatively higher in men who drank alcohol and smoked cigarettes, and increasing relatively more with age in those who had higher fasting serum triglyceride concentrations and in those who drank alcohol. 30.5% of the total inter-individual variance in BMI was explained by the factors measured.

6.6.2. Analysis of inter-individual variance of diastolic blood pressure

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in diastolic blood pressure was investigated by analysis of variance (Addendum 2, Table A2.11). 66.5% of the observed variance in diastolic blood pressure was explained, 57.9% by inter-individual and 8.6% by intra-individual variation. There was a strong linear effect during the intervention period, with survey number as an ordinal (linear) variable ($F_{1,159}=27.7$, $p<0.001$) explaining 67.8% of the variance in diastolic blood pressure explained by survey number as a factor ($F_{3,159}=4.40$, $p<0.01$); thus there was a continuing and systematic decrease in diastolic blood pressure over the period of the intervention.

The relationship between diastolic blood pressure and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period was investigated by analysis of inter-individual variance (Addendum 2, Table A2.11). After controlling for survey number (approximating intra-individual variance), diastolic blood pressure was positively correlated with age ($F_{1,159}=45.1$, $p<0.001$) and BMI ($F_{1,159}=7.10$, $p<0.001$), and was higher in men than women ($F_{1,159}=25.5$, $p<0.001$). There was a significant interactive effect between gender and age ($F_{1,159}=28.2$, $p<0.001$), with diastolic blood pressure increasing relatively more with age in men than in women. Diastolic blood pressure was not significantly affected by alcohol or kava consumption, cigarette smoking, the interaction between BMI and gender or between BMI and age. 38.6% of the total inter-individual variance in diastolic blood pressure was explained by the factors measured.

6.6.3. Analysis of inter-individual variance of systolic blood pressure

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in systolic blood pressure was investigated by analysis of variance (Addendum 2, Table A2.12). 82.9% of the observed variance in systolic blood pressure was explained, 76.4% by inter-individual and 6.4% by intra-individual variation. There was a strong linear effect during the intervention period, with survey number as an ordinal (linear) variable ($F_{1,158}=45.3$, $p<0.001$) explaining 76.4% of the variance in systolic blood pressure explained by survey number as a factor ($F_{3,158}=4.68$, $p<0.005$); thus there was a continuing and systematic decrease in systolic blood pressure over the period of the intervention.

The relationship between systolic blood pressure and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period was investigated by analysis of inter-individual variance (Addendum 2, Table A2.12). After controlling for survey number (approximating intra-individual variance), systolic blood pressure was positively correlated with age ($F_{1,158}=141.7$, $p<0.001$), BMI ($F_{1,158}=44.6$, $p<0.001$) and alcohol intake ($F_{1,158}=13.93$, $p<0.005$), and was higher in men than women ($F_{1,158}=28.0$, $p<0.001$). There was a significant interactive effect between gender and age ($F_{1,158}=26.7$, $p<0.001$), with systolic blood pressure increasing relatively more with age in women than in men. Systolic blood pressure was not significantly affected by kava consumption, cigarette smoking, or the interaction between BMI and gender or between BMI and age. 36.1% of the total inter-individual variance in systolic blood pressure was explained by the factors measured.

6.6.4. Analysis of inter-individual variance of serum cholesterol concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in total serum cholesterol concentration was investigated by analysis of variance (Addendum 2, Table A2.13). 85.9% of the observed variance in total serum cholesterol concentration was explained, 80.9% by inter-individual and 5.0% by intra-individual variation. Survey number as an ordinal (linear) variable ($F_{1,157}=37.1$, $p<0.001$) explained 66.0% of the variance in total serum cholesterol concentration explained by survey number as a factor ($F_{3,157}=6.41$, $p<0.001$), indicating a very strong linear decrease in serum cholesterol concentration over the intervention period.

The relationship between serum cholesterol concentration and gender, age, BMI, status of cigarette smoking,

alcohol and kava consumption at each three month period was investigated by analysis of inter-individual variance (Addendum 2, Table A2.13). After controlling for survey number (approximating intra-individual variance), total serum cholesterol concentration was positively correlated with age ($F_{1,157}=32.0$, $p<0.001$), BMI ($F_{1,157}=48.44$, $p<0.001$), high density lipoprotein (HDL) cholesterol ($F_{1,157}=35.72$, $p<0.001$), and was higher in men than women ($F_{1,157}=27.02$, $p<0.001$). There was a significant interactive effect between gender and age ($F_{1,157}=56.19$, $p<0.001$) and HDL-cholesterol concentration and age ($F_{1,157}=14.53$, $p<0.001$), with total serum cholesterol concentration increasing relatively more in women with increasing age, and increasing relatively more with increasing HDL in older people. Smoking status, alcohol consumption and kava consumption did not significantly affect serum cholesterol concentration. 24.8% of the total inter-individual variance in the concentration of total serum cholesterol was explained by the factors measured.

In order to investigate the relationship between serum lipid concentrations, additional analysis of variance in total serum cholesterol was conducted (Addendum 2, Table A2.14). Serum cholesterol concentration was found to be positively associated with fasting serum triglyceride concentration ($F_{1,151}=153.8$, $p<0.001$). A significant positive interaction was also apparent between BMI and fasting serum triglyceride concentration ($F_{1,151}=17.92$, $p<0.001$). 86.2% of the observed variance in total serum cholesterol concentration was explained, 81.7% by inter-individual and 4.5% by intra-individual variation.

6.6.5. Analysis of inter-individual variance of HDL-cholesterol concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in HDL-cholesterol concentration was investigated by analysis of variance (Addendum 2, Table A2.15). 70.9% of the observed variance in HDL-cholesterol concentration was explained, 67.1% by inter-individual and 3.7% by intra-individual variation. Survey number as an ordinal (linear) variable ($F_{1,158}=9.13$, $p<0.005$) explained 45.6% of the variance in HDL-cholesterol concentration explained by survey number as a factor ($F_{3,158}=3.55$, $p<0.05$); thus there was a significant linear decrease in HDL-cholesterol over the course of the study.

The relationship between HDL-cholesterol concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period, was investigated by analysis of inter-individual variance (Addendum 2, Table A2.15). After controlling for survey number (approximating intra-individual variance), HDL-cholesterol concentration was positively correlated with total serum cholesterol ($F_{1,158}=19.3$, $p<0.001$). There was a significant interactive effect between gender and age ($F_{1,158}=7.72$, $p<0.01$), and between gender and BMI ($F_{1,158}=9.38$, $p<0.005$), with HDL-cholesterol increasing relatively more in men than women with both age and increasing BMI. Alcohol consumption, kava consumption and cigarette smoking did not significantly affect HDL-cholesterol concentration. 11.5% of the total inter-individual variance in the concentration of HDL-cholesterol was explained by the factors measured.

In additional analysis of variance in serum HDL-cholesterol concentration, which included fasting serum triglyceride concentration in order to investigate the relationship between serum lipid concentrations (Addendum 2, Table A2.16), serum HDL-cholesterol concentration was also found to be negatively associated with fasting serum triglyceride concentration ($F_{1,152}=32.06$, $p<0.0001$). 71.4% of the observed variance in HDL-cholesterol concentration was explained, 67.1% by inter-individual and 4.3% by intra-individual variation. Survey number as

an ordinal (linear) variable ($F_{1,132}=11.27$, $p<0.001$) explained 49.4% of the variance in HDL-cholesterol concentration explained by survey number as a factor ($F_{3,132}=3.87$, $p<0.05$). 18.0% of the total inter-individual variance in the concentration of HDL-cholesterol was explained by the factors measured.

6.6.6. Analysis of inter-individual variance of fasting serum triglyceride concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in fasting serum triglyceride concentration was investigated by analysis of variance (Addendum 2, Table A2.17). Because of the strong positive association between diabetes and fasting triglyceride concentration, subjects with diabetes were excluded from analysis. 58.6% of the observed variance in serum triglyceride concentration was explained, 56.3% by inter-individual and only 2.3% by intra-individual variation. Survey number as an ordinal (linear) variable ($F_{1,132}=2.05$, ns) explained 28.2% of the variance in serum triglyceride concentration explained by survey number as a factor ($F_{3,132}=1.73$, ns).

The relationship between fasting serum triglyceride concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period, was investigated by analysis of inter-individual variance (Addendum 2, Table A2.17). After controlling for survey number (approximating intra-individual variance), serum triglyceride concentration was positively correlated with age ($F_{1,132}=3.40$, $p<0.05$), BMI ($F_{1,132}=7.13$, $p<0.01$), and cigarette smoking ($F_{1,132}=17.0$, $p<0.001$). There was a significant interactive effect between gender and age ($F_{1,132}=6.36$, $p<0.05$) and between gender and alcohol consumption ($F_{1,132}=5.12$, $p<0.05$), with serum triglyceride concentration increasing relatively more in women with increasing age, and increasing relatively more in men who drank alcohol than women. There was no significant interaction between cigarette smoking and either gender or age. Kava consumption did not significantly affect fasting serum triglyceride concentration. 22.3% of the total inter-individual variance in the concentration of serum triglyceride was explained by the factors measured.

In additional analysis of variance in fasting serum triglyceride concentration which included cholesterol concentrations in order to investigate the relationship between serum lipid concentrations (Addendum 2, Table A2.18), fasting serum triglyceride concentration was found to be positively associated with total serum cholesterol concentration ($F_{1,130}=37.0$, $p<0.001$), but negatively associated with HDL-cholesterol concentration ($F_{1,130}=22.95$, $p<0.001$). Once the cholesterol concentrations were fitted, neither age nor the interaction between age and gender, remained significantly associated with fasting serum triglyceride concentration. 60.1% of the observed variance in fasting serum triglyceride concentration was explained, 57.9% by inter-individual and 2.3% by intra-individual variation. 47.2% of the total inter-individual variance in the concentration of fasting serum triglyceride was explained by the factors measured.

6.6.7. Analysis of inter-individual variance of two-hour plasma glucose concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in two-hour plasma glucose concentration in non-diabetics was investigated by analysis of variance (Addendum 2, Table A2.19). 84.9% of the observed variance in two-hour plasma glucose concentration was explained, 83.6% by inter-individual and only 1.3% by intra-

individual variation. As a glucose tolerance test was conducted on only two occasions (June 1989 and June 1990), it was not possible to investigate linear change in two-hour plasma glucose concentration over the intervention period.

The relationship between two-hour plasma glucose concentration in non-diabetic subjects, and gender, age, BMI, fasting serum triglyceride concentration, fasting plasma glucose concentration, fasting and two-hour serum insulin concentration, status of cigarette smoking, alcohol and kava consumption at the first and last survey was investigated by analysis of inter-individual variance (Addendum 2, Table A2.19). After controlling for survey number (approximating intra-individual variance), two-hour plasma glucose concentration was positively correlated with age ($F_{1,24}=6.14$, $p<0.05$), BMI ($F_{1,24}=9.57$, $p<0.005$), fasting plasma glucose concentration ($F_{1,24}=12.11$, $p<0.001$) and two-hour serum insulin concentration ($F_{1,24}=29.35$, $p<0.001$), and was higher in women than in men ($F_{1,24}=21.92$, $p<0.001$). Alcohol consumption, kava consumption, cigarette smoking, fasting serum triglyceride concentration and fasting serum insulin concentration did not significantly affect two-hour plasma glucose concentration. 59.4% of the total inter-individual variance in the concentration of two-hour plasma glucose was explained by the factors measured.

6.6.8. Analysis of inter-individual variance of gamma-glutamyl transferase

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in serum gamma-glutamyl transferase (γ gt) concentration was investigated by analysis of variance (Addendum 2, Table A2.20). 96.8% of the observed variance in serum γ gt concentration was explained, 95.2% by inter-individual and 1.6% by intra-individual variation. Survey number as an ordinal (linear) variable ($F_{1,132}=20.7$, $p<0.001$) explained 28.6% of the variance in serum γ gt concentration explained by survey number as a factor ($F_{3,132}=17.27$, $p<0.001$); thus there was a linear decrease in γ gt concentration over the period of the intervention.

The relationship between serum γ gt concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period, was investigated by analysis of inter-individual variance (Addendum 2, Table A2.20). After controlling for survey number (approximating intra-individual variance), serum γ gt concentration was positively associated with BMI ($F_{1,132}=101.7$, $p<0.001$), kava ($F_{1,132}=519.2$, $p<0.001$) and alcohol consumption ($F_{1,132}=42.38$, $p<0.001$), cigarette smoking ($F_{1,132}=28.0$, $p<0.001$) and fasting serum triglyceride concentration ($F_{1,132}=320.0$, $p<0.001$). It was negatively correlated with age ($F_{1,132}=30.60$, $p<0.001$) and was higher in men than women ($F_{1,132}=81.0$, $p<0.001$). There was a significant interactive effect between gender and kava consumption ($F_{1,132}=71.8$, $p<0.001$), with serum γ gt concentration increasing relatively more in men with consumption of kava, and also between gender and BMI ($F_{1,132}=87.8$, $p<0.001$), with γ gt concentration being relatively lower with increasing BMI in women than in men. There was also a very strong significant interaction between BMI and kava consumption ($F_{1,132}=223.4$, $p<0.001$), with γ gt concentration being relatively higher in kava drinkers of low BMI, and also between fasting serum triglyceride concentration and alcohol consumption ($F_{1,132}=13.6$, $p<0.001$) with γ gt being relatively higher in those alcohol drinkers with highest fasting serum triglyceride levels. There was no significant interaction between gender and cigarette smoking, between gender and age, or between BMI and either alcohol consumption, age or serum triglyceride concentration. 35.0% of the total inter-individual variance in the concentration of serum γ gt was explained by the factors measured.

6.7. Change in folate and haematological status over the intervention period

Initial findings of the screening process in June 1989 are presented in detail (Addendum 2, Table A2.5). In June 1989 no woman was receiving iron/folate supplementation for obstetric reasons. In all ensuing presentation of folate and haematological data, the three women who did receive such supplementation during the one year intervention period have been excluded. In March the survey was conducted completely by Minjilang Aboriginal Health Workers and residents; apart from the two researchers from MSHR no external health professionals were involved in that survey (section 8.3.1.6). Unfortunately, due to insufficient supervision, several blood samples collected during this survey were not adequately mixed with reagents in the collecting tubes resulting in clotting in several samples collected for haematological measurement. Consequently these specimens could not be analysed resulting in the smaller sample size for affected parameters at that survey. From a seasonal perspective, the findings of the final monitoring process in June 1990 were directly comparable with the initial screening results, and are also presented in detail by age and sex groups (Addendum 2, Table A2.7).

Results for the total sample screened and monitored at each three month period are presented in Table 6.17 and Table 6.18. Results for the subset of subjects participating in the surveys at both June 1989 and June 1990 are presented in Table 6.19.

Table 6.18. Folic acid status and haematological data for those participating in any survey^o (excluding women supplied with folate/iron supplementation) (mean \pm se)

Variable	n	June 1989	n	Sept 1989	n	Dec 1989	n	March 1990	n	June 1990	F-value Btwn.Grp.	F-value Linearity
RBC folate ($\mu\text{g/l}$)	65	81.6 \pm 3.0	42	105.5 \pm 6.1	49	135.3 \pm 6.3	30	117.4 \pm 6.7	43	191.4 \pm 9.6	47.2 ^{***}	161.9 ^{***}
Serum folate ($\mu\text{g/l}$)	65	2.1 \pm 0.2	42	1.8 \pm 0.1	48	2.8 \pm 0.2	-		43	2.8 \pm 0.2	6.8 ^{**}	13.3 ^{**}
Haemoglobin (g/l)	65	145.7 \pm 1.6	41	145.7 \pm 2.2	49	144.9 \pm 1.9	17	140.4 \pm 3.6	45	147.0 \pm 1.9	0.81 ^{†*}	0.00
Haematocrit (%)	65	0.45 \pm 0.01	41	0.44 \pm 0.01	49	0.43 \pm 0.01	17	0.42 \pm 0.01	45	0.43 \pm 0.01	2.7 [*]	6.7 ^{**}
MCV (fl)	65	89.5 \pm 0.5	41	88.4 \pm 0.6	49	89.4 \pm 0.5	17	87.5 \pm 0.8	45	87.2 \pm 0.6	3.3 [*]	8.9 ^{***}
RDW (%)	65	14.3 \pm 0.2	41	13.5 \pm 0.2	49	13.7 \pm 0.1	16	13.1 \pm 0.1	45	13.8 \pm 0.2	6.4 ^{***}	7.0 ^{**}
Ferritin ($\mu\text{g/l}$)	65	121.7 \pm 13.5	42	183.8 \pm 22.3	49	171.7 \pm 16.7	30	150.2 \pm 18.2	45	161.6 \pm 17.4	2.2	1.8

^o Excluding missing values

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.005$ † $p < 0.001$ †† $p < 0.0005$ ††† $p < 0.0001$

* Using appropriate test of greater sensitivity significant change was subsequently apparent (section 6.7.2; Table 6.4)

† Because of the skewed distribution and heterogeneity between variances the fact that there is not a difference between the means may be misleading

Table 6.19. Folic acid status and haematological data for those participating in all surveys^o (excluding women supplied with folate/iron supplementation) (mean \pm se)

Variable	n	June 1989	n	Sept 1989	n	Dec 1989	n	March 1990	n	June 1990	F-value Btwn.Grp.	F-value Linearity
RBC folate ($\mu\text{g/l}$)	19	82.6 \pm 6.9	19	100.6 \pm 8.4	19	138.9 \pm 10.7	19	118.1 \pm 9.7	19	197.9 \pm 16.5	16.1 ^{***}	50.8 ^{***}
Serum folate ($\mu\text{g/l}$)	19	2.2 \pm 0.3	19	2.1 \pm 0.2	19	3.3 \pm 0.3	-		19	3.1 \pm 0.3	3.5 [*]	5.6 [*]
Haemoglobin (g/l)	19	142.4 \pm 2.7	19	143.3 \pm 2.7	19	142.4 \pm 2.5	11	136.5 \pm 4.2	19	145.2 \pm 2.6	0.93	0.02
Haematocrit (%)	19	0.44 \pm 0.01	19	0.43 \pm 0.01	19	0.42 \pm 0.01	11	0.41 \pm 0.01	19	0.43 \pm 0.01	1.0	1.04
MCV (fl)	19	89.8 \pm 1.1	19	88.2 \pm 1.0	19	89.5 \pm 0.9	11	86.6 \pm 1.0	19	87.2 \pm 1.0	1.6	3.8 [*]
RDW (%)	19	13.8 \pm 0.2	19	13.2 \pm 0.2	19	13.3 \pm 0.1	11	13.0 \pm 0.1	19	13.5 \pm 0.2	2.3	1.6

^o Excluding missing values

* p = <0.05 ** p = <0.01 *** p = <0.005 * p = <0.001 ** p = <0.0005 *** p = <0.0001

Table 6.20. Folate status and haematological data: pre-intervention (June 1989) and post-intervention (June 1990) (excluding women supplied with folate/iron supplementation) n=42 repeated pairs^o (mean \pm se)

Variable	n	June 1989	June 1990	T-test (paired)
RBC folate ($\mu\text{g/l}$)	40	82.0 \pm 3.7	188.9 \pm 8.6	12.7 ^{***}
Serum folate ($\mu\text{g/l}$)	41	2.0 \pm 0.2	2.9 \pm 0.2	3.90 ^{***}
Haemoglobin (g/l)	42	145.2 \pm 2.0	147.2 \pm 2.0	1.87
Haematocrit (%)	42	0.45 \pm 0.01	0.43 \pm 0.01	-3.26 ^{***}
MCV (fl)	42	89.3 \pm 0.6	87.4 \pm 0.6	-5.63 ^{***}
RDW (%)	42	14.2 \pm 0.2	13.8 \pm 0.2	-2.74 ^{**}
Ferritin ($\mu\text{g/l}$)	42	125.7 \pm 15.1	170.0 \pm 17.9	4.03 ^{***}

^o Excluding missing values

* p = <0.05 ** p = <0.01 *** p = <0.005 # p = <0.001 ## p = <0.0005 ### p = <0.0001

6.7.1. Serum and red blood cell folate concentrations

In June 1989, both the mean folate red blood cell (82.8 \pm 3.0 $\mu\text{g/l}$) and mean serum folate concentrations (2.2 \pm 0.2 $\mu\text{g/l}$) were very low relative to the respective Caucasian reference ranges of 115-600 $\mu\text{g/l}$ and 2.7-18.5 $\mu\text{g/l}$. Relative to the respective Caucasian reference range, 89.7% of those screened in June 1989 had low red blood cell folate concentration and 80.9% had low serum folate concentration. Young women tended to have the lowest concentration of red blood cell folate, and young men the highest. However young men also tended to have the lowest serum folate concentrations (Addendum 2, Table A2.5). These observations were further investigated in section 6.8.

In June 1990, the mean red blood cell folate concentration of the adult population living at Minjilang had increased by 235% and mean serum folate had increased by 132%. Relative to the respective Caucasian reference range, only two subjects (4.5%) had persistently low red blood cell folate concentration in June 1990 (figure 6.4). However, 23 subjects (51.1%) had low serum folate concentration at June 1990 (Figure 6.5).

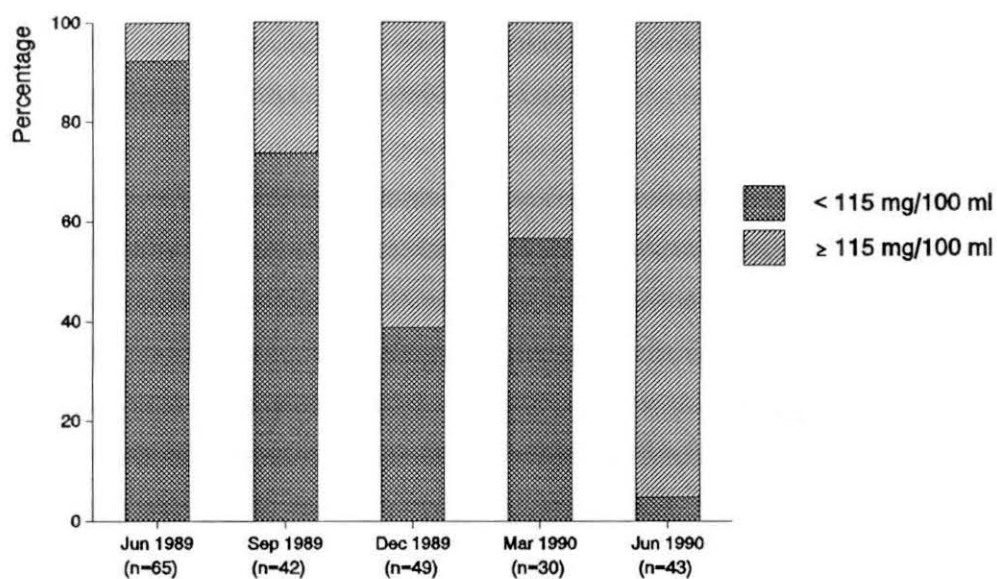


Figure 6.4. Distribution of red blood cell folate concentration

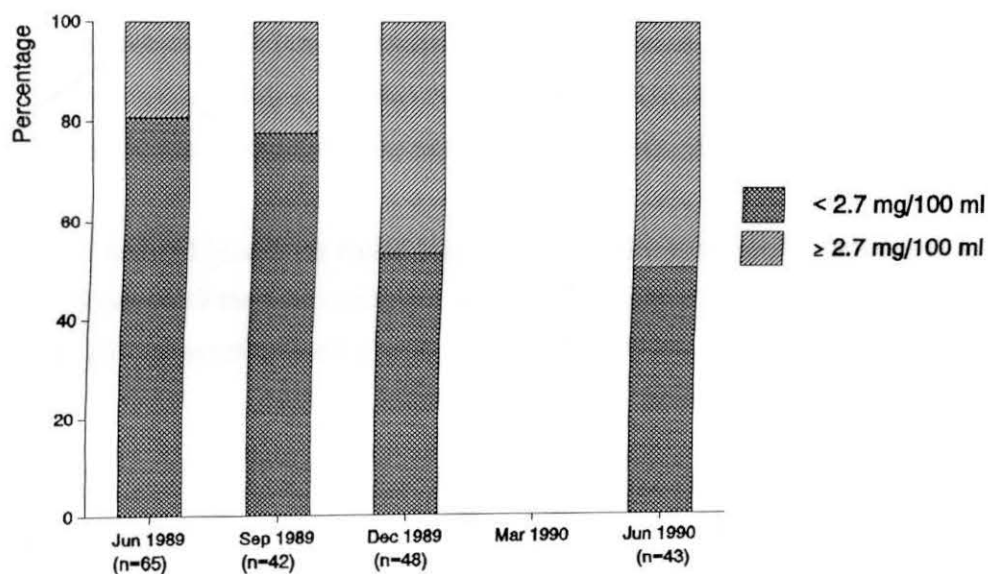


Figure 6.5. Distribution of serum folate concentration

Direct analysis of variance suggested a marked linear increase in red blood cell folate concentration, and serum folate concentration measured at three month intervals over the intervention period (Table 6.18). These improvements were still statistically significant when only those individuals participating in all five surveys were considered (Table 6.19).

Due to the increase in variance of red blood cell folate concentration over time, analysis of variance in the logarithm of red blood cell folate was also investigated, and revealed an even more significant linear increase in red blood cell folate concentration over the intervention period ($F_{4,227} = 188.6$, $p < 0.0001$).

From a statistical perspective direct comparison of the between survey results did not account for the different attendance of individuals at each three month period. Using GLIM a more sensitive analysis of variance was conducted after controlling for inter-individual variance, which consistently supported the dramatic linear increase in red blood cell folate concentration measured over the intervention period (Table 6.4). After controlling for inter-individual variance the mean adjusted red blood cell folate concentration for each survey was consecutively 81.6, 105.8, 141.0, 121.4 and 188.3 $\mu\text{g/l}$.

Comparison, by paired t-test, of the folate status of the 42 non-supplemented subjects participating in the initial screening in June 1989 and re-monitored in June 1990, also suggested a marked and significant increase in both folate concentration of the red blood cells and serum (Table 6.20). Although the increase in red blood cell folate concentration occurred over all age and sex groups in these subjects, (Addendum 2, Table A2.9), the effect was particularly marked in older women ($n = 13$, $t = 8.49$, $p < 0.0001$).

Although serum and red blood cell folate concentrations were positively correlated in June 1989 and December 1989 the correlation was not significant at other surveys (Table 6.21). However, over all surveys the positive correlation was highly significant.

Table 6.21. The relationship between serum and red blood cell folate concentration

	June 1989	September 1989	December 1989	March 1990	June 1990	All surveys
n	65	42	48	-	42	207
r	0.47	0.17	0.46	-	-0.04	0.31
p	<0.001	ns	<0.001	-	ns	<0.0001

6.7.2. Change in haematological status

Results of pre-intervention screening in June 1989, revealed that, of the 68 adults screened in June 1989, only one person (male) had frank macrocytic anaemia (haemoglobin less than 130 g/l, MCV > 100 fl, HCT > 0.54). One woman had an elevated haematocrit value, and two women had low haematocrit values relative to the Caucasian reference range of 0.40-0.54 (men) and 0.37-0.47 (women) respectively. However 38.2% of all people had a high ratio of red cell distribution width (RDW > 14.5%). As expected, younger women tended to have the lowest serum ferritin and haemoglobin concentrations and MCV (Addendum 2, Table A2.5).

Available haematological data for all non-supplemented adults monitored every three months are also presented in Table 6.18. Analysis of variance suggested significant linear decreases in the mean haematocrit, mean cell volume, and red cell distribution width of Minjilang adults measured at three month intervals throughout the 12 month intervention period. Although the mean haemoglobin concentration increased, the linear trend was not significant. When the comparatively small number of individuals screened every time was considered, improvements remained statistically significant only for MCV (Table 6.19).

Comparison, by paired t-test, of the haematological status of the 42 non-supplemented individuals participating in the initial screening in June 1989 and re-monitored in June 1990, also demonstrated a small, but highly significant, decrease in mean cell volume, haematocrit and red cell distribution width (Table 6.20). Although haemoglobin concentration tended to increase over the year, the change was not significant. However, from a statistical perspective direct comparison of the between survey results did not account for the different attendance of individuals at each three month period. Using GLIM, a more sensitive analysis of variance was conducted after controlling for inter-individual variance, which suggested a small but statistically significant change in haemoglobin concentration over the intervention period ($F_{4,141}=4.4$, $p<0.01$), although the linear change remained insignificant (Table 6.4b). After controlling for inter-individual variance the mean adjusted haemoglobin concentration for each survey was consecutively 145.7, 146.3, 145.4, 143.6 and 147.4 mg/l. Serum ferritin also tended to increase over the intervention period (Table 6.18, Table 6.19); however the increase was only statistically significant when pre- and post-intervention measures were compared by t-test (Table 6.20).

In June 1990, in comparison to Caucasian reference standards, all individuals screened had acceptable haemoglobin concentration and haematocrit. One young woman with a low mean cell volume and marginal haemoglobin concentration had menorrhagia in May 1990. Ten subjects (21.3%) had persistently elevated red cell distribution width (that is, anisocytosis).

6.7.3. The relationship between intra-individual change in haematological variables and folate status over the intervention period

In order to investigate whether change in individual haematological status over the intervention period was related to change in folate status, analysis of intra-individual variance was conducted for haematological variables.

The relationship between individual change in haemoglobin concentration and individual change in BMI, folate status, status of cigarettes smoking, kava and alcohol consumption over time, was investigated by analysis of intra-individual variance (Addendum 2, Table A2.36). After controlling for subject identification code (fitted as a factor to approximate inter-individual variance), there was a significant interaction between individual red blood cell folate concentration and survey number fitted as an ordinal (linear) variable ($F_{1,136}=4.69$, $p<0.05$), but not between the two when survey number was fitted as a factor ($F_{3,136}=0.77$, ns). This interaction demonstrated that intra-individual variation in haemoglobin was affected to a greater degree by the increase in red blood cell folate concentration during the last three months of the intervention period. Changes in individual status serum folate concentration, cigarette smoking, alcohol and kava intake, and BMI did not significantly affect individual change in haemoglobin concentration over the intervention period.

The relationship between individual change in mean cell volume and individual change in BMI, folate status, status of cigarettes smoking, kava and alcohol consumption over time, was investigated by analysis of intra-individual variance (Addendum 2, Table A2.37). After controlling for subject identification code (fitted as a factor to approximate inter-individual variance), the rise in red blood cell folate concentration ($F_{1,131}=25.4$, $p<0.001$) significantly explained the decrease in mean cell volume over the intervention period. Those individuals who stopped drinking kava during the intervention period also experienced a significant decrease in mean cell volume ($F_{1,131}=17.18$, $p<0.001$). There was no significant interaction between change in red blood cell folate concentration and kava consumption. There was a significant interaction between red blood cell folate concentration and survey number fitted as a factor ($F_{3,131}=2.76$, $p<0.05$), but not between the two when survey number was fitted as an ordinal (linear) variable ($F_{1,131}=0.10$, ns). This interaction demonstrated that intra-individual variation in mean cell volume was affected to a greater degree by the increase in red blood cell folate concentration during the first six months of the intervention period. Changes in individual serum folate concentration, status of cigarette smoking, alcohol intake and BMI did not significantly affect mean cell volume over the intervention period.

The relationship between individual change in haematocrit and individual change in BMI, folate status, status of

cigarettes smoking, kava and alcohol consumption over time, was investigated by analysis of intra-individual variance (Addendum 2, Table A2.38). After controlling for subject identification code (fitted as a factor to approximate inter-individual variance), the rise in red blood cell folate concentration ($F_{1,132}=10.30$, $p<0.001$) significantly explained the decrease in haematocrit over the intervention period. There was a significant interaction between individual red blood cell folate concentration and survey number fitted as an ordinal (linear) variable ($F_{1,132}=11.78$, $p<0.001$), but not between the two when survey number was fitted as a factor ($F_{3,132}=0.69$, ns). This interaction suggested that intra-individual variation in haematocrit was affected to a greater degree by the increase in red blood cell folate concentration from the third to the ninth month of the intervention period. Change in individual status of cigarette smoking, kava and alcohol consumption and BMI did not affect haematocrit over the intervention period.

The relationship between individual change in red cell distribution width and individual change in BMI, folate status, status of cigarettes smoking, kava and alcohol consumption over time, was investigated by analysis of intra-individual variance (Addendum 2, Table A2.39). After controlling for subject identification code (fitted as a factor to approximate inter-individual variance), the rise in red blood cell folate concentration ($F_{1,135}=5.87$, $p<0.001$) significantly contributed to the decrease in red cell distribution width over the intervention period. Those individuals who stopped drinking kava during the intervention period also experienced a significant decrease in red cell distribution width ($F_{1,135}=5.11$, $p<0.001$). There was no significant interaction between change in red blood cell folate concentration and kava consumption. There was no significant interaction between red blood cell folate concentration and survey number fitted as an ordinal (linear) variable ($F_{1,135}=0.13$, ns); however the interaction between the two variables was significant when survey number was fitted as a factor ($F_{3,135}=4.23$, $p<0.001$). This interaction demonstrated that intra-individual variation in red cell distribution width was affected to a greater degree by the increase in red blood cell folate concentration during the first six months of the intervention period. Changes in individual serum folate concentration, status of cigarette smoking, alcohol consumption and BMI did not significantly affect red cell distribution width over the intervention period.

Therefore analysis of intra-individual variance of haematological variables suggested that improvements in individual haematological status were related to improvements in individual folate status over the intervention period.

6.8. The relationship between folic acid, haematological status and other variables at each survey

Analysis of inter-individual variance of variables at each survey was conducted in order to reveal associated conditions. Potentially confounding factors relevant to the comparison of change in dietary intake and nutritional status addressed in analysis of intra-individual variance of biological variables (Chapter 7), were also identified.

6.8.1

Analysis of inter-individual variance of red blood cell folate concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in red blood cell folate concentration over the intervention period, were investigated by analysis of variance (Addendum 2, Table A2.21). 71.8% of the observed variance in red blood cell folate was explained, 26.6% by inter-individual and 45.2% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,148}=206.6$, $p<0.001$) explained 87% of the variance in red blood cell folate concentration explained by survey number fitted as a factor ($F_{3,148}=10.2$, $p<0.001$), indicating a very strong linear change in individual red blood cell folate over the intervention period.

The relationship between red blood cell folate concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period was investigated by analysis of inter-individual variance (Addendum 2, Table A2.21). After controlling for survey number, (approximating intra-individual change over time), red blood cell folate concentration tended to be lower in women ($F_{1,148}=3.28$, ns), and was significantly lower in those who smoked cigarettes ($F_{1,148}=17.2$, $p<0.005$). There was a strong interactive effect between cigarette smoking and gender ($F_{1,148}=15.8$, $p<0.005$); red blood cell folate concentration was relatively lower in men who smoked than in women who smoked. BMI, age, alcohol consumption and kava consumption did not significantly affect red blood cell folate concentration. 23% of the inter-individual variance in red blood cell folate concentration was explained by the factors measured.

6.8.2.

Analysis of inter-individual variance of serum folate concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in serum folate concentration over the intervention period, were investigated by analysis of variance (Addendum 2, Table A2.22). 74.1% of the observed variance in serum folate was explained, 64.8% by inter-individual and only 9.3% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,121}=27.4$, $p<0.001$) explained 62.9% of the variance in serum folate concentration explained by survey number fitted as a factor ($F_{2,121}=8.1$, $p<0.001$), indicating a very strong linear change in individual serum folate concentration over the intervention period.

The relationship between serum folate concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period, was investigated by analysis of inter-individual variance (Addendum 2, Table A2.22). After controlling for survey number, (approximating intra-individual change over time), serum folate concentration was significantly higher in older people ($F_{1,121}=17.5$, $p<0.001$), in men ($F_{1,121}=8.1$, $p<0.005$), and in those who did not drink alcohol ($F_{1,121}=24.1$, $p<0.001$) or smoke cigarettes ($F_{1,121}=50.5$, $p<0.001$). There was a strong interactive effect between gender and smoking status ($F_{1,121}=20.0$, $p<0.001$), with cigarette smoking in women having less relative effect on serum folate concentration than in men, and also between age and smoking status ($F_{1,121}=15.8$, $p<0.001$), with serum folate reduced to a greater degree in older people who smoked than in younger smokers. Neither BMI nor kava consumption significantly affected serum folate concentration. 32% of the inter-individual variance in serum folate concentration was explained by the factors measured.

The relationship between cigarette smoking and folate status was examined in more detail due to the possible

clinical significance of the results. In the first survey, June 1989, red blood cell folate concentration was negatively correlated with the number of cigarettes smoked ($r=-0.25$, $p<0.05$); however the correlation decreased as red blood cell folate concentration increased over time, and was no longer significant in June 1990. No significant interaction was found between smoking status and survey number fitted as an ordinal (linear) variable ($F_{1,147}=0.92$, ns).

In all surveys, individual serum folate concentration was negatively correlated with the number of cigarettes smoked (Table 6.22). Over all surveys the correlation was highly significant ($n=198$, $r= -0.29$, $p<0.0001$).

Table 6.22. The relationship between number of cigarettes smoked per day and serum folate concentration

	June 1989	September 1989	December 1989	March 1990	June 1990
n	65	42	43	-	42
r	-0.32	-0.39	-0.34	-	-0.32
p	<0.01	<0.01	<0.05	-	<0.05

6.8.3. Analysis of inter-individual variance of haemoglobin

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) of haemoglobin concentration over the intervention period, were investigated by analysis of variance (Addendum 2, Table A2.23). 90.3% of the observed variance in haemoglobin concentration was explained, 89.4% by inter-individual and only 0.9% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,128}=0.06$, ns) explained less than one percent of the variance in haemoglobin concentration explained by survey number fitted as a factor ($F_{3,128}=4.1$, $p<0.01$).

The relationship between haemoglobin concentration and gender, age, BMI, folate status, status of cigarette smoking, alcohol and kava consumption at each three month period, was investigated by analysis of inter-individual variance (Addendum 2, Table A2.23). After controlling for survey number, (approximating intra-individual change over time), haemoglobin concentration was higher in men ($F_{1,128}=419.5$, $p<0.001$), in cigarette smokers ($F_{1,128}=44.4$, $p<0.001$), and in those who drank alcohol ($F_{1,128}=13.5$, $p<0.005$). However, highly significant interactive effects were found for gender and cigarette smoking ($F_{1,128}=19.9$, $p<0.001$), and for gender and alcohol consumption ($F_{1,128}=31.2$, $p<0.001$), with both cigarette smoking and alcohol consumption having a relatively greater positive effect on haemoglobin concentration in men than in women. Haemoglobin concentration was positively correlated with red blood cell folate concentration ($F_{1,128}=26.4$, $p<0.001$), but there was a significant interaction between survey number and red blood cell folate concentration ($F_{3,128}=16.2$, $p<0.001$). This interaction suggested that those individuals with better folate status had relatively higher haemoglobin levels during the last three months of the intervention project. Age, kava consumption and serum folate concentration did not significantly affect haemoglobin concentration. 45.3 % of the inter-individual variance in haemoglobin concentration was explained by the factors measured.

6.8.4. Analysis of inter-individual variance of mean cell volume

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) of mean cell volume over the intervention period, were investigated by analysis of variance (Addendum 2, Table A2.24). 94.0% of the observed variance in mean cell volume was observed, 88.7% by inter-individual and only 5.3% by intra-individual variance. Survey number fitted as an ordinal (linear) variable ($F_{1,125}=72.4$, $p<0.001$), explained 85.1% of the variance in mean cell volume explained by survey number fitted as a factor ($F_{3,125}=12.67$, $p<0.001$), indicating a very strong linear change in individual mean cell volume over the intervention period.

The relationship between mean cell volume and gender, age, BMI, folate status, status of cigarette smoking, alcohol and kava consumption at each three month period over the intervention period, was investigated by analysis of inter-individual variance (Addendum 2, Table A2.24). After controlling for survey number, (approximating intra-individual change over time), mean cell volume was positively associated with being male ($F_{1,125}=12.84$, $p<0.001$), kava consumption ($F_{1,125}=197.7$, $p<0.001$) and cigarette smoking ($F_{1,125}=5.57$, $p<0.05$). Mean cell volume was negatively associated with red blood cell folate concentration ($F_{1,125}=5.04$, $p<0.05$). A strong interactive effect was found between gender and cigarette smoking ($F_{1,125}=50.7$, $p<0.001$), and between alcohol consumption and gender ($F_{1,125}=72.7$, $p<0.001$), with both cigarette smoking and alcohol consumption having a greater relative effect on mean cell volume in men than in women. A significant interaction was also apparent between gender and kava consumption ($F_{1,125}=41.2$, $p<0.001$), with kava consumption having a greater relative effect on mean cell volume in women than in men. The effect of smoking on mean cell volume was stronger than the effect of alcohol intake, but mean cell volume was higher in those who both smoked and drank alcohol, than in those who only smoked cigarettes. There was a significant interaction between survey number and red blood cell folate concentration ($F_{3,125}=46.8$, $p<0.001$). Age, BMI and serum folate concentration did not significantly affect mean cell volume. 26.7 % of the inter-individual variance in mean cell volume was explained by the factors measured.

6.8.5. Analysis of inter-individual variance of haematocrit

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in haematocrit over the intervention period, were investigated by analysis of variance (Addendum 2, Table A2.25). 90.3% of the observed variance in haematocrit was explained, 85.9% by inter-individual and only 4.5% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,127}=38.25$, $p<0.001$) explained 64.8% of the variance in haematocrit concentration explained by survey number fitted as a factor ($F_{3,127}=6.96$, $p<0.001$), indicating a very strong linear change in individual haematocrit over the intervention period.

The relationship between haematocrit and gender, age, BMI, folate status, status of cigarette smoking, alcohol and kava consumption at each three month period, was investigated by analysis of inter-individual variance (Addendum 2, Table A2.25). After controlling for survey number, (approximating intra-individual change over time), haematocrit was higher in men ($F_{1,127}=410.6$, $p<0.001$), in cigarette smokers ($F_{1,127}=30.33$, $p<0.001$) and in those who drank alcohol ($F_{1,127}=15.33$, $p<0.001$). However, highly significant interactive effects were found for gender and cigarette smoking ($F_{1,127}=18.47$, $p<0.001$) and for gender and alcohol consumption

($F_{1,127}=17.50$, $p<0.001$), with haematocrit being relatively more affected by alcohol consumption and cigarette smoking in men than in women. The effect of smoking on haematocrit was stronger than the effect of alcohol intake, but haematocrit was higher in those who smoked and drank alcohol, than in those who only smoked cigarettes. Haematocrit was also found to increase more with increasing age in women than in men ($F_{1,127}=85.13$, $p<0.001$). Haematocrit was negatively correlated with red blood cell folate concentration ($F_{1,127}=57.9$, $p<0.001$), but there was a significant interaction between survey number and red blood cell folate concentration ($F_{1,127}=25.4$, $p<0.001$). BMI, kava consumption and serum folate concentration did not significantly affect haematocrit. 55.9 % of the inter-individual variance in haematocrit was explained by the factors measured.

6.8.6. Analysis of inter-individual variance of red cell distribution width

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification code to the model) in red cell distribution width over the intervention period, were investigated by analysis of variance (Addendum 2, Table A2.26). 82.3% of the observed variance in red cell distribution width was explained, 71.2% by inter-individual and 11.1% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,125}=17.4$, $p<0.001$) explained only 2.5% of the variance in red cell distribution width explained by survey number fitted as a factor ($F_{3,125}=20.3$, $p<0.001$).

The relationship between red cell distribution width and gender, age, BMI, folate status, status of cigarette smoking, alcohol and kava consumption at each three month period, was investigated by analysis of inter-individual variance (Addendum 2, Table A2.26). After controlling for survey number, (approximating intra-individual change over time), red cell distribution width was higher in cigarette smokers ($F_{1,125}=13.5$, $p<0.001$), in older people ($F_{1,125}=29.5$, $p<0.001$) and in those with lower red blood cell concentration of folate ($F_{1,125}=13.9$, $p<0.001$). However, highly significant interactive effects were found for gender and cigarette smoking ($F_{1,125}=27.0$, $p<0.001$) and for gender and alcohol consumption ($F_{1,125}=7.47$, $p<0.01$), with red cell distribution width being relatively more affected by alcohol and cigarette smoking in men than in women. Red cell distribution width was found to increase more with increasing age in men than in women ($F_{1,125}=23.2$, $p<0.001$). There was a significant interaction between survey number and red blood cell folate concentration ($F_{1,125}=3.50$, $p<0.05$). Neither BMI nor serum folate concentration significantly affected red cell distribution width. 26.4% of the inter-individual variance in red cell distribution width was explained by the factors measured.

6.8.7. Analysis of inter-individual variance of serum ferritin concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification number to the model) in serum ferritin concentration over the intervention period, were investigated by analysis of variance (Addendum 2, Table A2.27). 90.6% of the observed variance in serum ferritin concentration was explained, 86.0% by inter-individual and 4.6% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,134}=21.4$, $p<0.001$) explained 32.5% of the variance in serum ferritin concentration explained by survey number fitted as a factor ($F_{3,134}=14.8$, $p<0.001$).

The relationship between serum ferritin concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period, was investigated by analysis of variance (Addendum 2, Table A2.27). After controlling for survey number (approximating intra-individual change over time), serum ferritin concentration was significantly higher in men ($F_{1,134}=57.9$, $p<0.001$), in those who did not drink kava ($F_{1,134}=4.25$, $p<0.05$) and in those who smoked cigarettes ($F_{1,134}=22.5$, $p<0.001$) and was strongly and positively correlated with BMI ($F_{1,134}=177.0$, $p<0.001$) and age ($F_{1,134}=190.1$, $p<0.001$). There was a significant interactive effect between gender and BMI ($F_{1,134}=21.75$, $p<0.001$), between cigarette smoking and gender ($F_{1,134}=7.25$, $p<0.01$), and between gender and kava consumption ($F_{1,134}=8.95$, $p<0.005$); ferritin concentration increased more with increasing BMI in men than in women, was higher in men who smoked than in women who smoked, and was lower in men who drank kava than in women who drank kava. Consumption of alcohol was not found to significantly affect serum concentration of ferritin. 39.8% of the total inter-individual variance in concentration of serum ferritin was explained by the factors measured.

6.9. Change in vitamin status over the intervention period

The vitamin status of Minjilang adults in June 1989 is presented in detail by age and sex groups (Addendum 2, Table A2.5). From a seasonal perspective, the findings of the final monitoring process in June 1990 were directly comparable with the initial screening results, and are also presented in detail (Addendum 2, Table A2.7).

Results for the total sample screened at monitored at each three month period are presented in Table 6.23 and Table 6.24. Results for the subset of subjects participating in surveys at both June 1989 and June 1990 are presented in Table 6.25.

Table 6.23. Vitamin data for those participating in any survey^o (mean \pm se)

Variable	n	June 1989	n	Sept 1989	n	Dec 1989	n	March 1990	n	June 1990	F-value Btwn.Grp.	F-value Linearity
Serum thiamine (μ g/l)	68	7.5 \pm 0.3	45	5.8 \pm 0.2	51	11.9 \pm 0.4	-		45	8.9 \pm 0.6	39.5 ^{***}	23.7 ^{***}
RBC thiamine (μ g/l)	68	61.7 \pm 1.8	44	67.7 \pm 1.9	51	106.8 \pm 4.2	-		45	56.0 \pm 1.7	77.2 ^{***†}	0.32 [†]
Serum vitamin B ₆ (nmol/l)	68	41.4 \pm 2.0	45	53.8 \pm 2.8	51	42.8 \pm 3.3	-		45	65.0 \pm 4.8	11.8 ^{***}	22.3 ^{***}
Serum vitamin B ₁₂ (ng/l)	68	755 \pm 22	45	836 \pm 45	51	650 \pm 35	-		45	743 \pm 24	5.40 ^{***}	1.36
Plasma vitamin C (mg/l)	67	4.10 \pm 0.34	-		49	9.51 \pm 2.24	-		46	10.34 \pm 2.49	4.20 ^{**}	7.44 ^{***†}
Plasma retinol (mg/100 ml)	59	62.0 \pm 2.4	42	59.7 \pm 2.1	42	68.0 \pm 3.1	-		45	68.2 \pm 3.4	1.52	2.39
Plasma α -tocopherol (mg/l)	59	12.3 \pm 0.5	42	11.2 \pm 0.6	42	10.6 \pm 0.4	-		45	11.6 \pm 0.7	1.85	0.89
Plasma α -carotene (mg/100 ml)	58	(2.0 \pm 0.5) ^o	-		42	9.6 \pm 1.4	-		45	12.6 \pm 1.8	n/a ^o	0.60 ^o
Plasma β -carotene (mg/100 ml)	58	5.7 \pm 0.5	-		42	10.4 \pm 1.9	-		45	7.6 \pm 1.1	3.93 [*]	1.72

^o Excluding missing values

* p = <0.05 ** p = <0.01 *** p = <0.005 † p = <0.001 ** p = <0.0005 *** p = <0.0001

[†] Heterogenous variances may be important but are unlikely to have invalidated the major positive findings; this issue will be explored in detail at a later date

^o Inferences not possible due to suspected measurement error: Linear F-value was related only to results from December 1989 and June 1990.

Table 6.24. Vitamin data for those participating in every survey^o (mean ± se)

Variable	n	June 1989	n	Sept 1989	n	Dec 1989	n	March 1990	n	June 1990	F-value Btwn.Grp.	F-value Linearity
Serum thiamine (µg/l)	21	6.7±0.3	21	5.7±0.3	21	11.6±0.4	-		21	9.6±0.8	21.5 ^{***}	25.0 ^{***}
RBC thiamine (µg/l)	21	61.5±3.7	21	68.1±2.2	21	110.1±5.6	-		21	54.0±2.6	45.0 ^{***}	0.78 [†]
Serum vitamin B ₆ (nmol/l)	21	44.4±4.3	21	53.3±4.0	21	48.3±5.7	-		21	67.1±7.0	3.4 [*]	8.2 ^{**}
Serum vitamin B ₁₂ (ng/l)	21	743±35	21	743±43	21	661±64	-		21	719±38	0.69	0.30
Plasma vitamin C (mg/l)	21	3.92±0.59	-		20	16.8±4.79	-		21	12.61±4.58	3.0 [†]	2.6 [†]
Plasma retinol (mg/100 ml)	21	64.0±3.6	20	58.3±3.2	20	69.1±4.7	-		21	66.4±4.8	1.19	0.78
Plasma α-tocopherol (mg/l)	21	12.6±0.7	20	10.6±0.6	20	11.2±0.6	-		21	11.1±0.6	1.81	1.53
Plasma α-carotene (mg/100 ml)	21	(1.1±0.6) ^o	-		20	9.8±1.8	-		21	8.5±1.3	n/a ^o	1.92 ^o
Plasma β-carotene (mg/100 ml)	21	6.3±1.1	-		20	15.5±3.4	-		21	8.5±1.7	4.5 [*]	0.47

^o Excluding missing values

* p = <0.05 ** p = <0.01 *** p = <0.005 † p = <0.001 †† p = <0.0005 ††† p = <0.0001

[†] Heterogenous variances may be important but are unlikely to have invalidated the major positive findings; this issue will be explored in detail at a later date

^o Inferences not possible due to suspected measurement error: Linear F-value related only to results from December 1989 and June 1990.

Table 6.25. Vitamin data: pre-intervention - June 1989, post-intervention - June 1990^o (mean \pm se)

Variable	n	June 1989	June 1990	t-test (paired)
Serum thiamine ($\mu\text{g/l}$)	43	7.2 \pm 0.39	9.0 \pm 0.64	2.70**
RBC thiamine ($\mu\text{g/l}$)	43	62.4 \pm 2.3	56.1 \pm 1.8	-2.42*
Serum vitamin B ₆ (nmol/l)	43	42.7 \pm 2.5	66.0 \pm 4.9	5.40***
Serum vitamin B ₁₂ (ng/l)	43	729.2 \pm 27.3	741.2 \pm 24.8	0.42
Plasma vitamin C (mg/l)	43	3.98 \pm 0.40	10.87 \pm 2.65	2.64* ¹
Plasma retinol (mg/100 ml)	44	62.7 \pm 23.3	69.4 \pm 34.6	2.34*
Plasma α -tocopherol (mg/l)	44	12.3 \pm 0.6	11.7 \pm 0.7	-0.98
Plasma α -carotene ^o (mg/100 ml)	43	(2.1 \pm 0.6) ^o	14.8 \pm 2.8	n/a ^o
Plasma β -carotene (mg/100 ml)	43	6.4 \pm 0.9	8.4 \pm 1.2	2.44*

^o Excluding missing values

* p = <0.05 ** p = <0.01 *** p = <0.005 ¹ p = <0.001 ** p = <0.0005 *** p = <0.0001

¹ Heterogenous variances may be important but are unlikely to have invalidated the findings; this issue will be explored in detail at a later date

^o Inferences not possible due to suspected measurement error

6.9.1. Change in status of water soluble vitamins over the intervention period

Results of vitamin assay of samples, revealed that in June 1989, 20.6% of the adult population at Minjilang had high levels of serum thiamine concentration compared with the Caucasian reference range of 3.0 to 9.3 $\mu\text{g/l}$. This proportion changed markedly during the intervention period, and increased to 37.8% one year later (Figure 6.6). At no time during the intervention period did any subject have a low serum concentration of thiamine relative to the Caucasian reference range. However, in June 1989, 20.6% of adults had a low red blood cell thiamine concentration relative to the Caucasian reference range of 50-106 $\mu\text{g/l}$. Although this proportion remained similar one year later, there was a dramatic decrease in the proportion of adults with relatively low red blood cell thiamine concentration in December 1989, when 52.9% had a relatively high concentration of red blood cell thiamine (Figure 6.7).

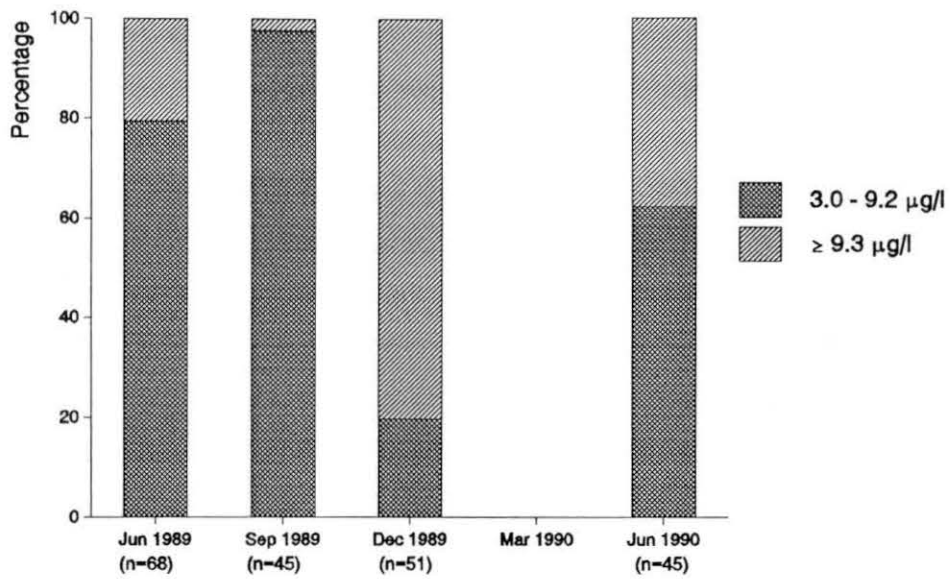


Figure 6.6. Distribution of serum thiamine concentration

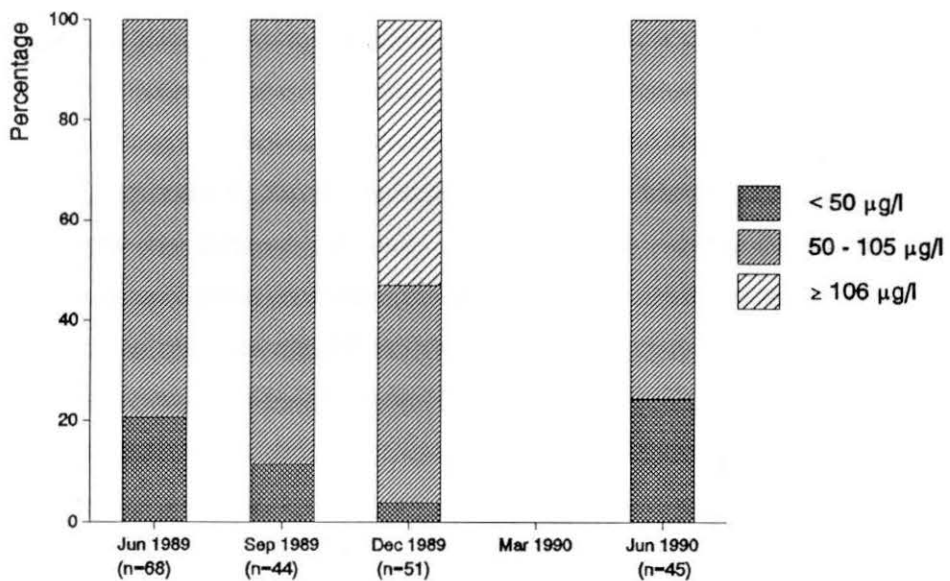


Figure 6.7. Distribution of red blood cell thiamine concentration

Compared with the Caucasian reference range of 60-850 µg/l, no Minjilang adult was found to have a low serum concentration of vitamin B₁₂. In June 1989, 27.9% of adults had a high serum concentration of vitamin B₁₂, and this proportion remained similar 12 months later, although slightly increasing in September 1989 (Figure 6.8).

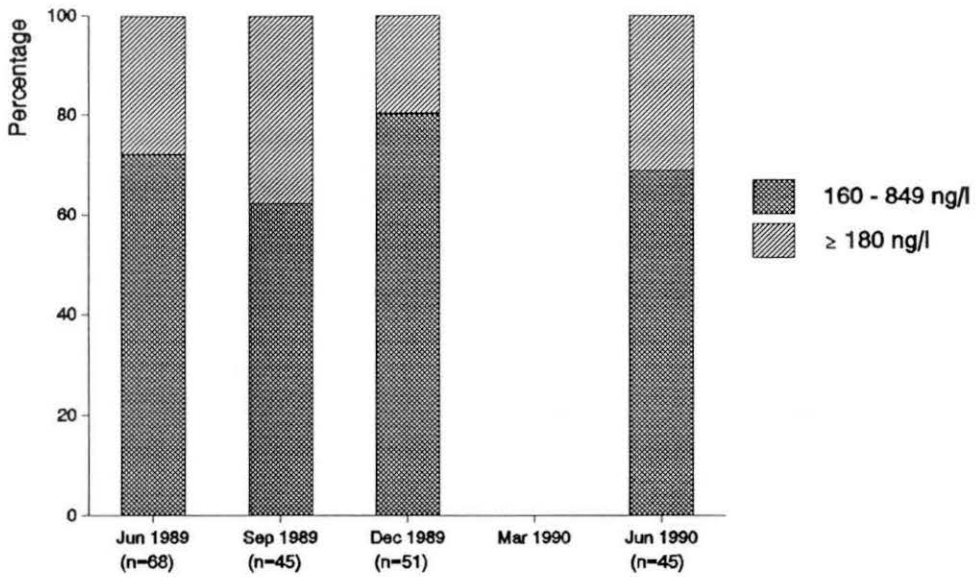


Figure 6.8. Distribution of serum vitamin B₁₂ concentration

The normal Caucasian reference range of serum pyridoxal concentration varies according to age and gender (section 1.6.13.5). However, in analysis of inter-individual variance, no significant association was found between age and serum concentration of pyridoxal at Minjilang (Addendum 2, Table A2.28). Therefore, to enable further investigation of the change in pyridoxal status of Minjilang adults, results were compared with the Caucasian reference range for the mean age of those adults screened at Minjilang on the basis of gender only (21.6-54.0 nmol/l for women and 25.2-66.0 nmol/l for men). In the initial screening, 55.9% of men and 51.5% of women were found to have comparatively low levels of serum pyridoxal concentration; no subject had a high serum pyridoxal concentration in June 1990. However, during the year there was an increase in the proportion of both sexes with relatively high serum pyridoxal concentration (men: $\chi^2_9=36.6$, $p<0.001$ and women: $\chi^2_9=30.2$, $p<0.001$) (Figure 6.9).

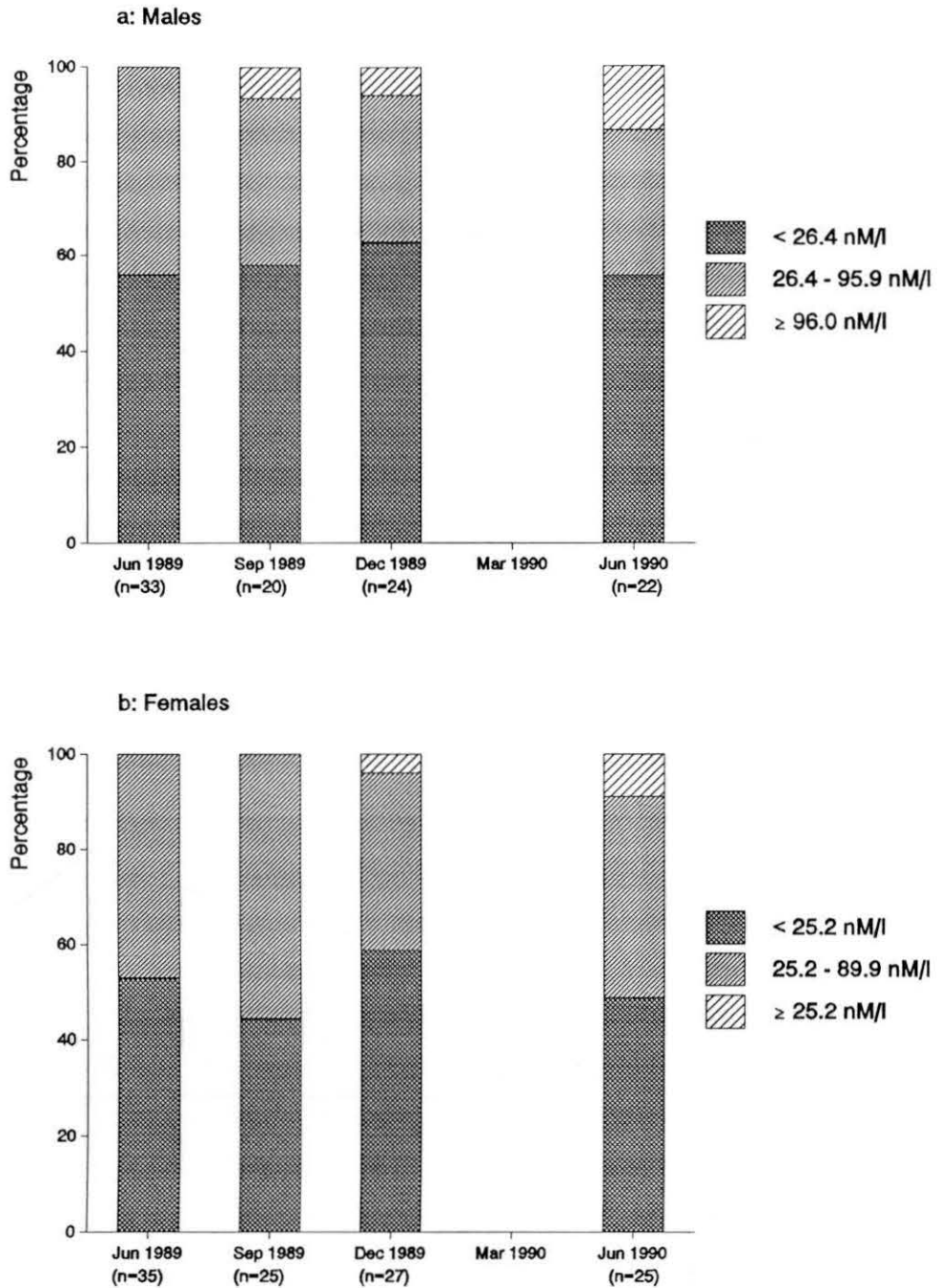


Figure 6.9. Distribution of serum pyridoxal concentration

Analysis of variance suggested a marked linear increase in serum concentration of vitamin B₆ and thiamine measured at three month intervals over the twelve month intervention period (Table 6.23). During this period there was neither a significant linear trend in serum vitamin B₁₂ nor mean red blood cell thiamine concentrations. However a marked seasonal variation was observed in both serum and red blood cell thiamine concentration, and a smaller, but highly significant, change in serum B₁₂ concentration (Table 6.23).

When only those individuals monitored at each three month period were considered, the seasonal change in serum B₁₂ concentration was no longer significant (Table 6.24). Because of the skewed distribution of the concentrations of red blood cell thiamine, serum vitamin B₁₂ and serum vitamin B₆ over the intervention period, analysis of variance in the logarithm of these variables was also investigated. Results revealed no change in the magnitude of the F values, either between surveys (red blood cell thiamine (F_{3,204}=63.1, p<0.0001), serum vitamin B₁₂ (F_{3,205}=5.35, p<0.005), serum vitamin B₆ (F_{3,205}=12.7, p<0.001)), or linearly over time (red blood cell thiamine (F_{1,206}=0.07, ns), serum vitamin B₁₂ (F_{1,207}=0.89, ns), serum vitamin B₆ (F_{1,207}=16.8, p<0.0001)).

Although serum and red blood cell thiamine concentrations were not significantly correlated in previous surveys throughout the year, a negative correlation was found in June 1990 (Table 6.26)⁷.

Table 6.26. The relationship between serum and red blood cell thiamine concentration

	June 1989	September 1989	December 1989	March 1990	June 1990
n	68	44	51	-	45
r	0.06	0.15	-0.06	-	-0.43
p	ns	ns	ns	-	<0.005

In June 1989, no Minjilang adult was found to have a concentration of plasma ascorbic acid within the 'normal' Caucasian reference range of 4.0-10.0 mg/l; 97.1% had values below the accepted low range (1.0 to 4.0 mg/l). In June 1990 there was a significant increase in the proportion of the population with acceptable plasma concentration of ascorbic acid ($\chi^2_4 = 15.11$, p<0.005) (Figure 6.10).

⁷ These findings will be explored in more detail, at a later stage, using statistical methods applicable to bivariate measurements, which can allow for correlations between variables as well as for heterogeneous sources of variation over time etc.

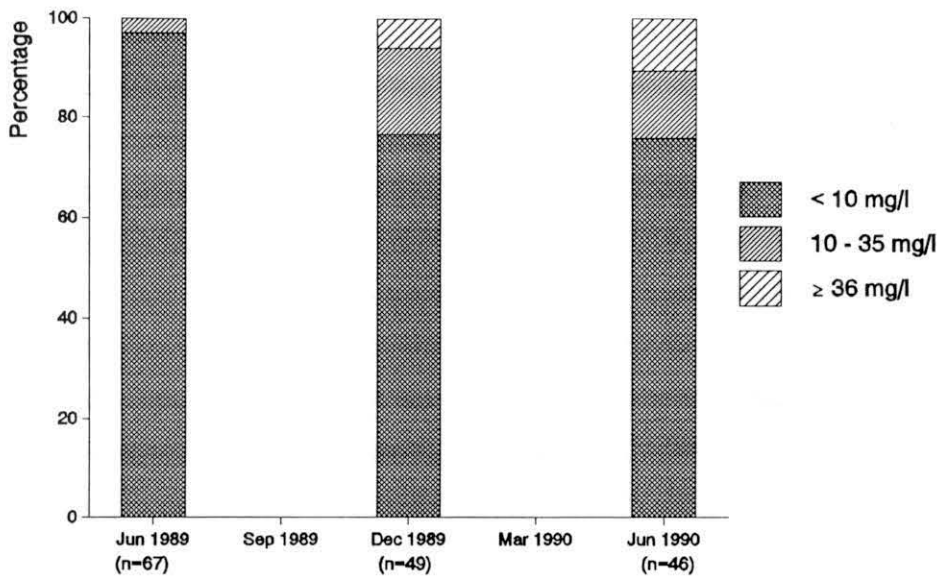


Figure 6.10. Distribution of plasma ascorbic acid concentration

Analysis of variance suggested a significant increase in concentration of ascorbic acid measured at three month intervals over the twelve month intervention period (Table 6.23). When only those individuals monitored at each three month period were considered this change was no longer significant (Table 6.24). Because of the skewed distribution of the concentration of plasma ascorbic acid over the intervention period, analysis of variance in the logarithm of this variable was also investigated. Results revealed no significant change in the magnitude of the F-value, either between surveys ($F_{2,131}=3.4$, $p < 0.05$) or linearly over time ($F_{1,132}=6.7$, $p < 0.01$). When change in the logarithm of the ascorbic acid concentration was investigated for those individuals participating in all five surveys, the F-value was also significant ($F_{2,50}=3.6$, $p < 0.05$).

Comparison, by paired t-test, of the water-soluble vitamin status of the 44 individuals participating in the initial screening in June 1989 and re-monitored in June 1990, suggested a markedly significant increase in serum vitamin B₆, a significant increase in both serum thiamine concentration and plasma ascorbic acid concentration, a decrease in red blood cell thiamine concentration, and no significant change in the serum concentration of vitamin B₁₂ (Table 6.25).

6.9.2. Change in fat soluble vitamin status over the intervention period

In June 1989, 52.5% of the adult population at Minjilang had high levels of plasma retinol concentration compared with the Caucasian reference range of 20-60 mg/100 ml, and a year later this proportion remained similar. Two young men initially had relatively low plasma retinol concentrations which increased to acceptable levels during the intervention period. Initially, two elderly men were found to have relatively low plasma α -tocopherol concentrations compared to Caucasian reference standards of greater than 0.5 mg/100ml. However, both had low serum cholesterol concentrations, and at no time during the year did the ratio of plasma α -tocopherol concentration to total lipid concentration of any subject decrease below 0.8 mg/g. In June 1989, 12 subjects (20.7% of the sample) had a plasma β -carotene concentration less than 10 mg/100 ml. In June 1990 five subjects (11.1% of the sample) had a low level of β -carotene. When the available valid results of plasma concentration of α -carotene and β -carotene were considered together, three subjects had low plasma carotene levels (< 20 mg/100 ml) in December 1989, but only one subject had low levels in June 1990.

Analysis of variance indicated no significant linear trends in either mean plasma retinol or mean plasma α -tocopherol concentrations (Table 6.23). During this period seasonal variation was observed only in mean plasma β -carotene concentration. Because of the skewed distribution of the concentrations of plasma retinol and β -carotene over the intervention period, analysis of variance in the logarithm of these variables was also investigated. Results revealed a slight reduction in the magnitude of the F-value for retinol concentration over the intervention period, both between surveys ($F_{3,184}=0.56$, ns) and linearly ($F_{1,186}=0.06$, ns). There was little difference in the magnitude of the F-value for plasma β -carotene concentration either between surveys ($F_{2,131}=3.78$, $p < 0.05$), or linearly over time ($F_{1,132}=2.10$, ns). Similar changes were observed when those individuals monitored at each three month period were considered (Table 6.24).

Comparison by paired t-test of the vitamin status of the 44 individuals participating in the initial screening in June 1989 and re-surveyed in June 1990, suggested a significant increase in plasma β -carotene and plasma retinol concentration, but not in α -tocopherol concentration (Table 6.25).

6.10. The relationship between vitamins and other variables at each survey

The correlation coefficients between the serum, plasma and red blood cell concentrations of the major vitamins found in fruit and vegetables are presented in Table 6.27. Results suggested that there was a significant positive association between the biological measure of the status of all these vitamins observed over the intervention period.

Table 6.27. Correlation coefficients (r) between selected biological indicators of vitamin status

	Plasma β -carotene concentration (mg/100 ml)	Serum folate concentration (μ g/l)	Red blood cell folate concentration (μ g/l)
Plasma ascorbic acid concentration (mg/l)	0.34 ^{***} (n=145)	0.14 [*] (n=160)	0.17 [*] (n=160)
Plasma β -carotene concentration (mg/100 ml)	-	0.27 ^{***} (n=186)	0.19 ^{**} (n=186)
Serum folate concentration (μ g/l)	-	-	0.31 ^{***} (n=207)

* p < 0.05 ** p < 0.01 *** p < 0.005 ' p < 0.001 ** p < 0.0005 *** p < 0.0001

Analysis of inter-individual variance of variables at each survey was conducted in order to identify potentially confounding factors relevant to the comparison of change in dietary intake and vitamin status (Chapter 7).

6.10.1. Analysis of inter-individual variance of serum vitamin B₆ concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification number to the model) in serum vitamin B₆ concentration over the intervention period were investigated by analysis of variance (Addendum 2, Table A2.28). 66.3% of the observed variance in serum vitamin B₆ concentration was explained, 15.5% by inter-individual and 50.7% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,129}=38.7$, $p < 0.001$) explained 65.2% of the variance in serum vitamin B₆ concentration explained by survey number fitted as a factor ($F_{2,129}=10.3$, $p < 0.001$), indicating a very strong linear increase in individual serum vitamin B₆ concentration over the intervention period.

The relationship between serum vitamin B₆ concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period was investigated by analysis of variance (Addendum 2, Table A2.28). After controlling for survey number (approximating intra-individual change over time), serum

vitamin B₆ concentration was significantly higher in men ($F_{1,129}=6.7$, $p<0.01$), in those with higher BMI ($F_{1,129}=15.1$, $p<0.001$) and in those who did not smoke cigarettes ($F_{1,129}=15.4$, $p<0.001$). There was a significant interactive effect between cigarette smoking and gender ($F_{1,129}=15.9$, $p<0.001$), with cigarette smoking in women having less relative effect on serum vitamin B₆ concentration than in men. Age, alcohol consumption and kava consumption did not significantly affect serum vitamin B₆ concentration. 33.6% of the total inter-individual variance in serum concentration of vitamin B₆ was explained by the factors measured.

6.10.2. Analysis of inter-individual variance of serum vitamin B₁₂ concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification number to the model) in serum vitamin B₁₂ concentration over the intervention period were investigated by analysis of variance (Addendum 2, Table A2.29). 67.0% of the observed variance in serum vitamin B₁₂ concentration was explained, 60.0% by inter-individual and 7.0% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,130}=1.77$, ns) explained only 6.4% of the variance in serum concentration of vitamin B₁₂ which was explained by survey number fitted as a factor ($F_{2,130}=12.8$, $p<0.001$).

The relationship between serum vitamin B₁₂ concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period was investigated by analysis of variance (Addendum 2, Table A2.29). After controlling for survey number (approximating intra-individual change over time), serum vitamin B₁₂ concentration was significantly higher in older people ($F_{1,130}=21.6$, $p<0.001$), in those who did not drink alcohol ($F_{1,130}=17.4$, $p<0.001$) and also tended to be higher in men ($F_{1,130}=2.46$, ns). There was a strong interactive effect between age and gender ($F_{1,130}=15.7$, $p<0.001$), and between gender and alcohol consumption ($F_{1,130}=8.46$, $p<0.005$); serum vitamin B₁₂ concentration increasing more with age in women than in men and being comparatively reduced in men who drank alcohol than in women who drank. BMI, cigarette smoking and kava consumption did not significantly affect vitamin B₁₂ concentration. 27.8% of the total inter-individual variance in serum vitamin B₁₂ concentration was explained by the factors measured.

6.10.3. Analysis of inter-individual variance of serum thiamine concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification number to the model) in serum thiamine concentration over the intervention period were investigated by analysis of variance (Addendum 2, Table A2.30). 66.3% of the observed variance in serum thiamine concentration was explained, only 15.5% by inter-individual and 50.7% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,129}=34.5$, $p<0.001$) explained 65.2% of the variance in serum thiamine concentration explained by survey number fitted as a factor ($F_{2,129}=73.9$, $p<0.001$), indicating a strong linear increase in individual serum thiamine concentration over the intervention period.

The relationship between serum thiamine concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period was investigated by analysis of variance (Addendum 2, Table A2.30). After controlling for survey number (approximating intra-individual change over time), serum thiamine concentration significantly increased with BMI ($F_{1,129}=30.2$, $p<0.001$). There was a significant

interactive effect between BMI and alcohol consumption ($F_{1,129}=8.49$, $p<0.005$), with serum thiamine concentration being relatively reduced in underweight drinkers. Age, gender, cigarette smoking and kava consumption did not significantly affect serum thiamine concentration. 22.9% of the total inter-individual variance in serum concentration of thiamine was explained by the factors measured.

6.10.4. Analysis of inter-individual variance of red blood cell thiamine concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification number to the model) in red blood cell thiamine concentration over the intervention period were investigated by analysis of variance (Addendum 2, Table A2.31). 77.7% of the observed variance in red blood cell thiamine concentration was explained, only 25.2% by inter-individual and 52.5% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,129}=0.81$, ns) explained less than 1% of the variance in red blood cell thiamine concentration explained by survey number fitted as a factor ($F_{2,129}=151.8$, ns).

The relationship between serum thiamine concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period was investigated by analysis of variance (Addendum 2, Table A2.31). After controlling for survey number (approximating intra-individual change over time), there was a small but statistically significant interactive effect between gender and smoking status ($F_{1,129}=4.98$, $p<0.05$), with red blood cell thiamine being relatively more reduced in men who smoked cigarettes than women who smoked. Age, BMI, alcohol and kava consumption did not significantly affect the concentration of red blood cell thiamine. Only 2.3% of the total inter-individual variance in red blood cell concentration of thiamine was explained by the factors measured.

6.10.5. Analysis of inter-individual variance of plasma ascorbic acid concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification number to the model) in plasma ascorbic acid concentration over the intervention period were investigated by analysis of variance (Addendum 2, Table A2.32). 56.2% of the observed variance in plasma ascorbic acid concentration was explained; 51.0% by inter-individual and 5.2% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,84}=8.70$, $p<0.005$) explained 46.4% of the variance in plasma ascorbic acid concentration explained by survey number fitted as a factor ($F_{2,84}=5.00$, $p<0.005$), indicating a very strong linear increase in individual plasma ascorbic acid concentration over the intervention period.

The relationship between plasma ascorbic acid concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period was investigated by analysis of variance (Addendum 2, Table A2.32). After controlling for survey number (approximating intra-individual change over time), plasma ascorbic acid concentration was significantly increased in those with higher BMI ($F_{1,84}=4.83$, $p<0.05$) and in those who did not smoke cigarettes ($F_{1,84}=12.60$, $p<0.001$). There was a significant interactive effect between cigarette smoking and BMI ($F_{1,84}=10.29$, $p<0.005$), with cigarette smoking having greater relative effect on those individuals of lowest BMI. Gender, age, alcohol and kava consumption did not significantly affect plasma ascorbic acid concentration. 28.5% of the total inter-individual variance in plasma ascorbic acid was explained by

the factors measured.

6.10.6. Analysis of inter-individual variance of plasma retinol concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification number to the model) in plasma retinol concentration over the intervention period were investigated by analysis of variance (Addendum 2, Table A2.33). 82.5% of the observed variance in plasma retinol concentration was explained, 81.2% by inter-individual and only 1.2% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,64}=1.32$, ns) explained only 29.0% of the variance in plasma retinol concentration explained by survey number fitted as a factor ($F_{1,64}=3.23$, ns).

The relationship between plasma retinol concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period was investigated by analysis of variance (Addendum 2, Table A2.33). After controlling for survey number (approximating intra-individual change over time), plasma retinol concentration was significantly lower in women ($F_{1,64}=8.05$, $p<0.01$) but tended to be positively correlated with BMI ($F_{1,64}=3.47$, ns) and significantly higher in those who drank kava ($F_{1,64}=14.63$, $p<0.001$). There was a significant interactive effect between cigarette smoking and gender ($F_{1,64}=15.50$, $p<0.001$), age ($F_{1,64}=9.99$, $p<0.005$) and BMI ($F_{1,64}=10.92$, $p<0.001$); plasma retinol concentration being relatively lower in male smokers, relatively lower in older smokers and relatively higher with increasing BMI in non-smokers. No significant interactive effect was found between kava status and other variables, nor did consumption of alcohol have a significant effect. 22.8% of the total inter-individual variance in plasma concentration of retinol was explained by the factors measured.

6.10.7. Analysis of inter-individual variance of plasma α -tocopherol concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification number to the model) in plasma α -tocopherol concentration over the intervention period were investigated by analysis of variance (Addendum 2, Table A2.34). 78.6% of the observed variance in plasma α -tocopherol concentration was explained, 75.1% by inter-individual and 3.5% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,74}=1.32$, ns) explained only 22.5% of the variance in plasma α -tocopherol concentration explained by survey number fitted as a factor ($F_{1,74}=3.23$, ns).

The relationship between plasma α -tocopherol concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period was investigated by analysis of variance (Addendum 2, Table A2.34). After controlling for survey number (approximating intra-individual change over time), plasma α -tocopherol concentration was strongly and positively associated with serum cholesterol concentration ($F_{1,74}=109.9$, $p<0.001$) and BMI ($F_{1,74}=21.61$, $p<0.001$), and was higher in those who drank kava ($F_{1,74}=7.09$, $p<0.05$). Gender, age, cigarette smoking and alcohol consumption did not significantly affect plasma α -tocopherol concentration. 40.1% of the total inter-individual variance in concentration of plasma α -tocopherol was explained by the factors measured.

6.10.8. Analysis of inter-individual variance of plasma β -carotene concentration

Total intra-individual variance (estimated by fitting survey number to the model) and inter-individual variance (estimated by fitting subject identification number to the model) in plasma β -carotene concentration over the intervention period were investigated by analysis of variance (Addendum 2, Table A2.35). 71.2% of the observed variance in plasma β -carotene concentration was explained, 63.2% by inter-individual and 8.0% by intra-individual variation. Survey number fitted as an ordinal (linear) variable ($F_{1,67}=5.80$, $p<0.05$) explained 31.1% of the variance in plasma β -carotene concentration explained by survey number fitted as a factor ($F_{1,67}=12.84$, $p<0.001$).

The relationship between plasma β -carotene concentration and gender, age, BMI, status of cigarette smoking, alcohol and kava consumption at each three month period was investigated by analysis of variance (Addendum 2, Table A2.35). After controlling for survey number (approximating intra-individual change over time), plasma β -carotene concentration was significantly lower in those who smoked cigarettes ($F_{1,67}=31.3$, $p<0.001$). There was a significant interactive effect between cigarette smoking and alcohol consumption ($F_{1,67}=4.15$, $p<0.05$), with plasma β -carotene concentration being lower in those who both drank alcohol and smoked cigarettes. Gender, age, BMI and kava consumption did not significantly affect plasma β -carotene concentration. 25.9% of the total inter-individual variance in concentration of plasma β -carotene was explained by the factors measured.

6.11. Major points

94% of all adults residing in the community participated in the initial screening. Amongst initial findings, 10.2% had NIDDM, a further 7.4% had impaired glucose tolerance, 63.3% were hypercholesterolaemic (mean \pm se, 5.93 ± 0.15 mmol/l), 38.9% of non-diabetics were hypertriglyceridaemic (mean \pm se, 1.79 ± 0.19 mmol/l), and 92.3% had low red blood cell folate status (mean \pm se, 81.6 ± 3.0 mmol/l). Initial results were used to classify health and nutritional problems of the community, to identify specific target groups and to provide a focus for subsequent intervention strategies.

Both analysis of variance and paired t-tests suggested improvements in several biological markers of health and nutritional status over the intervention period. These included a 12.3% decrease in mean serum cholesterol concentration, a marked improvement in red blood cell folate, serum vitamin B₆ and ascorbic acid concentrations and a significant decrease in both diastolic and systolic blood pressure. Significant linear improvements were also measured in a number of haematological variables and these were shown to reflect the improvement in folate status over the intervention period. The status of the fat soluble vitamins retinol and α -tocopherol were initially acceptable and did not change over the intervention year. After adjusting for inter-individual variance, a small but statistically

significant reduction in mean BMI was observed over the intervention period. There was also a significant trend for those individuals initially 'overweight' to lose weight, and those initially 'underweight' to gain weight. However, there was no significant improvement in fasting serum triglyceride concentration over the 12 month intervention period which would have been expected if biologically significant weight loss had occurred. Glucose tolerance appeared to improve in women over the age of 35 years, but did not improve in any other age/sex group.

CHAPTER 7: VALIDATION OF THE STORE-TURNOVER METHOD AS A MEASURE OF DIETARY INTAKE IN A REMOTE, CENTRALISED ABORIGINAL COMMUNITY.

This chapter presents the results of validation of the store-turnover method for application in remote, centralised Aboriginal communities. The method was congruently validated against biological measurement of nutritional status over the intervention period (that is, by analysis of intra-individual variance investigating the relationship between change in nutrient intake and change in biological parameters). Aspects of the face validity of the store-turnover method are also considered here.

7.1. Congruent validation of the store-turnover method

To validate the store-turnover method, the relationship between change in nutrient intake over the intervention period and change in biochemical, anthropometric and haematological indicators of nutritional status across all available data was investigated by analysis of intra-individual variance (section 4.10).

Dietary variables were chosen on a rational basis following consideration of the relevant literature (section 1.7.4).

Care must be taken in the interpretation of the results of analysis of intra-individual variance due to the dependent nature of many of the dietary variables. In particular, it must be recognised that some of the explained variance is due to chance and that any biological variable changing over time could be fortuitously correlated with changes in nutrient intake. It must also be stressed that, as the store-turnover method purports to measure the nutrient intake of the community as a whole, store-turnover results at any survey represent the best estimate of the diet of an 'average' community member at that time; intra-individual dietary variance is unknown. Nevertheless, if the changes over time in biological markers for individuals (ie. intra-individual changes) are correlated with prior changes in relevant nutrient intake as measured by store-turnover, this provides prima facie evidence for the congruent validity of the store-turnover method.

7.1.1. Change in BMI and style of diet over the intervention period

The relationship between the small individual reduction in BMI over the intervention period (section 6.5.1) and change in dietary intake as measured by the store-turnover method, together with change in individual status of cigarette smoking, kava and alcohol consumption, was investigated initially by analysis of intra-individual variance in non-diabetic subjects (Table 7.1). The change in the proportion of dietary fat contributing to total energy intake was chosen to represent the change in dietary intake because it was highly correlated with total energy intake ($r=0.92$, $p<0.0001$), yet independent of the changing total Minjilang population over the intervention period. The proportion of sugar intake contributing to total energy intake over the intervention period was also tested as a negative control variable.

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variation), reduction in the proportion of dietary fat contributing to total energy intake (as measured by the store-turnover method), significantly explained the change in BMI experienced in non-diabetic subjects over the intervention period. The model was not significantly affected by individual change over time in smoking status, alcohol intake, kava consumption or the proportion of dietary sugars contributing to total energy intake.

After controlling for inter-individual variance, survey number fitted as a factor explained 6.4% of the remaining variation in BMI, of which change in the proportion of energy derived from fat explained 76.6%¹. After controlling for inter-individual variance, survey number fitted as an ordinal (linear) variable explained 5.2% of the remaining variation in BMI, of which change in the proportion of energy intake derived from fat explained 93.6%.

Therefore, when diabetic subjects were excluded from analysis, change in the proportion of energy intake derived from dietary fat as measured by the store-turnover method, effectively explained most of the small linear and non-linear intra-individual change in BMI over the intervention period.

¹ Although awkward in style, the findings are presented in terms of a percentage of a percentage, as the second proportion is of principle interest; it represents the percentage of intra-individual variance over the intervention which is 'explained' by the dietary variable under consideration. 'Explained' is used in the statistical sense; ancillary biological evidence must be invoked to justify any causal explanations based on these data (section 1.7.4).

Table 7.1. Analysis of intra-individual variance of BMI (excluding diabetics)

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	4126.7	199					
Subject ID	55.92	137	4071	62	65.66	167.2	< 0.001
% energy from fat	53.19	136	2.73	1	2.73	6.94	< 0.01
Survey no. (ordinal)	53.01	135	0.1849	1	0.1849	0.47	ns
Survey no. (factor)	52.36	133	0.6473	2	0.3237	0.82	ns
residual	-	-	52.36	133	0.3937		

When diabetic subjects were included in analysis, the effect of change in the proportion of total intake of energy derived from dietary fat on intra-individual variance in BMI was even more marked (Table 7.2). The change in BMI could not be significantly explained by individual change over time in smoking status, alcohol intake, kava consumption or the proportion of dietary sugars contributing to total energy intake.

After controlling for inter-individual variance, survey number fitted as a factor explained 7.4% of the small remaining variation in BMI, of which change in the proportion of energy intake derived from fat explained 88.1%. After controlling for inter-individual variance, survey number fitted as an ordinal (linear) variable explained 6.7% of the remaining variation in BMI, of which change in the dietary intake of total fat explained 96.8%.

Therefore change in the proportion of energy intake derived from dietary fat as measured by the store-turnover method effectively explained most of the small linear and non-linear intra-individual change in BMI over the intervention period.

Table 7.2. Analysis of intra-individual variance of BMI (including diabetics)

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	5649.8	226					
Subject ID	64.81	158	5585	68	82.1	210.6	<0.001
% energy from fat	60.58	157	4.23	1	4.23	10.9	<0.001
Survey no. (ordinal)	60.44	156	0.134	1	0.134	0.34	ns
Survey no. (factor)	60.01	154	0.431	2	0.215	0.55	ns
residual	-	-	60.11	154	0.390		

7.1.2. Change in blood pressure and style of diet over the intervention period

7.1.2.1. Diastolic blood pressure

The relationship between individual change in diastolic blood pressure over the intervention period and change in dietary intake as measured by the store-turnover method, together with change in individual BMI and status of cigarette smoking, kava and alcohol consumption, was investigated by analysis of intra-individual variance (Table 7.3). Again the change in the proportion of dietary fat contributing to total energy intake was chosen to represent the change in dietary intake (section 7.1.1). The proportion of sugar intake contributing to total energy intake over the intervention period was also tested as a negative control variable.

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variation), the change in the proportion of total energy intake derived from dietary fat, significantly explained the individual change in diastolic blood pressure experienced over the intervention period. Diastolic blood pressure was not significantly affected by individual change over time in smoking status, alcohol intake, kava consumption or the proportion of dietary sugars contributing to total energy intake.

Although change in the density of intake of dietary sodium tended to be positively associated with change in diastolic blood pressure, it was no longer significantly related when fitted to the final model ($F_{1,158}=0.05$). Individual decrease in BMI tended to be positively associated with decrease in diastolic blood pressure but the effect was no longer significant when fitted to the final model ($F_{1,158}=0.79$).

After controlling for inter-individual variance, survey number fitted as a factor explained 13.6% of the remaining variation in diastolic blood pressure, of which change in the proportion of total energy intake derived from fat explained 82.4%. After controlling for inter-individual variance, survey number fitted as an ordinal (linear) variable explained 11.2% of the remaining variation in diastolic blood pressure, of which change in the dietary intake of total fat explained 99.6%.

Therefore change in the proportion of total energy intake derived from dietary fat as measured by the store-turnover method effectively explained most of the linear and non-linear intra-individual variance in diastolic blood pressure over the intervention period.

Table 7.3. Analysis of intra-individual variance of diastolic blood pressure over the intervention period

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	19107	233					
Subject ID	7452	164	11655	69	168.9	4.20	<0.001
% energy from fat	6619	163	832.7	1	832.7	20.7	<0.001
Survey no. (ordinal)	6615.9	162	3.55	1	3.55	0.09	ns
Survey no. (factor)	64441.0	160	174.9	2	87.5	2.17	ns
residual	-	-	6441.0	160	40.26		

7.1.2.2. Systolic blood pressure

The relationship between individual change in systolic blood pressure over the intervention period and change in dietary intake as measured by the store-turnover method, together with change in individual BMI and status of cigarette smoking, kava and alcohol consumption, was investigated by analysis of intra-individual variance (Table 7.4). Again the change in the proportion of dietary fat contributing to total energy intake was chosen to represent the change in dietary intake (section 7.1.1). The proportion of sugar intake contributing to total energy intake over the intervention period was also tested as a negative control variable.

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variation), the change in the proportion of total energy intake derived from dietary fat significantly explained the individual change in systolic blood pressure experienced over the intervention period. Systolic blood pressure was not significantly affected by individual change over time in smoking status, alcohol intake, kava consumption or the proportion of dietary sugars contributing to total energy intake. Although change in the density of intake of dietary sodium tended to be positively associated with change in systolic blood pressure, it was no longer significantly related when fitted to the final model ($F_{1,159}=2.64$). Individual decrease in BMI also tended to be positively associated with decrease in systolic blood pressure but the effect was no longer significant when fitted to the final model ($F_{1,159}=0.69$).

After controlling for inter-individual variance, survey number fitted as a factor explained 15.0% of the remaining variation in systolic blood pressure, of which change in the proportion intake of energy derived from fat explained 76.4%. After controlling for inter-individual variance, survey number fitted as an ordinal (linear) variable explained 11.6% of the remaining variation in systolic blood pressure, of which change in the dietary intake of total fat explained 98.8%.

Therefore change in the proportion of energy intake derived from dietary fat as measured by the store-turnover method effectively explained most of the linear and non-linear intra-individual change in systolic blood pressure over the intervention period.

Table 7.4. Analysis of intra-individual variance of systolic blood pressure over the intervention period

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	59481	233					
Subject ID	11982	164	47499	69	688.4	10.8	<0.001
% energy from fat	10610	163	1372	1	1372	21.6	<0.001
Survey no. (ordinal)	10594	162	15.67	1	15.67	0.25	ns
Survey no. (factor)	10186	160	407.6	2	203.8	3.20	ns
residual	-	-	10186	160	407.6		

7.1.3. Change in lipid status and style of diet over the intervention period

7.1.3.1. Serum cholesterol concentration

The relationship between change in total serum cholesterol concentration over the intervention period and change in dietary intake as measured by the store-turnover method, together with change in individual BMI and status of cigarette smoking, kava and alcohol consumption, was investigated by analysis of intra-individual variance. The specific dietary variables investigated were chosen following appraisal of the relevant literature (section 1.7.4). Two models were produced in order to investigate separately the effects of the change in both the quantity and quality of dietary lipid intake (Table 7.5) and the increase in density of dietary fibre intake (Table 7.6). It was necessary to develop two models due to the strong inverse correlation between the density of intake of dietary fibre with both the percentage of energy derived from saturated fat ($r=0.74$, $p<0.0001$) and the P:S ratio ($r=0.70$, $p<0.0001$)².

² Each model attempts to 'explain' the change in serum cholesterol concentration from a statistical perspective; if dietary fibre and lipids are fitted concurrently to the one model they compete for the same variance; this epitomises the difficulty in making causal inferences based on these data.

For the first model dietary variables investigated included the proportion of total energy intake derived from saturated fat, the ratio of polyunsaturated to saturated fatty acid intake (P:S ratio), the ratio of monounsaturated to saturated fatty acid intake and dietary cholesterol intake. For the second model density of intake of dietary fibre was investigated.

In the first model, after controlling for subject identification code (fitted as a factor to adjust for inter-individual variation), both the reduction in the proportion of total energy intake derived from saturated fat and the increase in the ratio of polyunsaturated to saturated fatty acid intake (P/S ratio) significantly explained the reduction in the concentration of total serum cholesterol over the intervention period (Table 7.5). No significant interaction was found between these two dietary variables ($r=0.05$). Serum cholesterol concentration was not significantly affected by individual change over time in BMI, smoking status, alcohol intake, kava consumption or the density of dietary cholesterol intake ($F_{1,158}=1.87$). Although the change in the ratio of monounsaturated to saturated fatty acids tended to be negatively associated with change in total serum cholesterol, the effect was no longer significant when fitted to the final model ($F_{1,158}=3.13$).

After adjustment for inter-individual variance, survey number fitted as a factor explained 13.1% of the remaining variation in total serum cholesterol concentration, of which change in the dietary intake of fatty acids explained 77.7%. After adjustment for inter-individual variance, survey number fitted as an ordinal (linear) variable explained 10.7% of the remaining variation in total serum cholesterol concentration, of which change in dietary intake of fatty acids explained 95.3%.

Therefore change in the dietary intake of fatty acids as measured by the store-turnover method, effectively explained most of the linear and much of the non-linear intra-individual change in total serum cholesterol level occurring over the intervention period.

Both the proportion of energy intake derived from saturated fat (figure 7.1) and the ratio of polyunsaturated fatty acids to saturated fatty acids (figure 7.2) were graphically depicted against the mean serum cholesterol concentration over the intervention period.

Table 7.5. Analysis of intra-individual variance of serum cholesterol concentration over the intervention period (including dietary intake of fatty acids)

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	340.51	237					
Subject ID	65.56	165	275.0	72	3.819	10.79	<0.001
% energy from saturated fat	62.788	164	2.772	1	2.772	7.84	<0.001
P/S ratio	58.876	163	3.912	1	3.912	11.06	<0.001
Survey no. (ordinal)	58.544	162	0.332	1	0.332	0.94	ns
Survey no. (factor)	56.958	161	1.586	1	1.586	4.48	<0.05
residual	-	-	56.958	161	0.354		

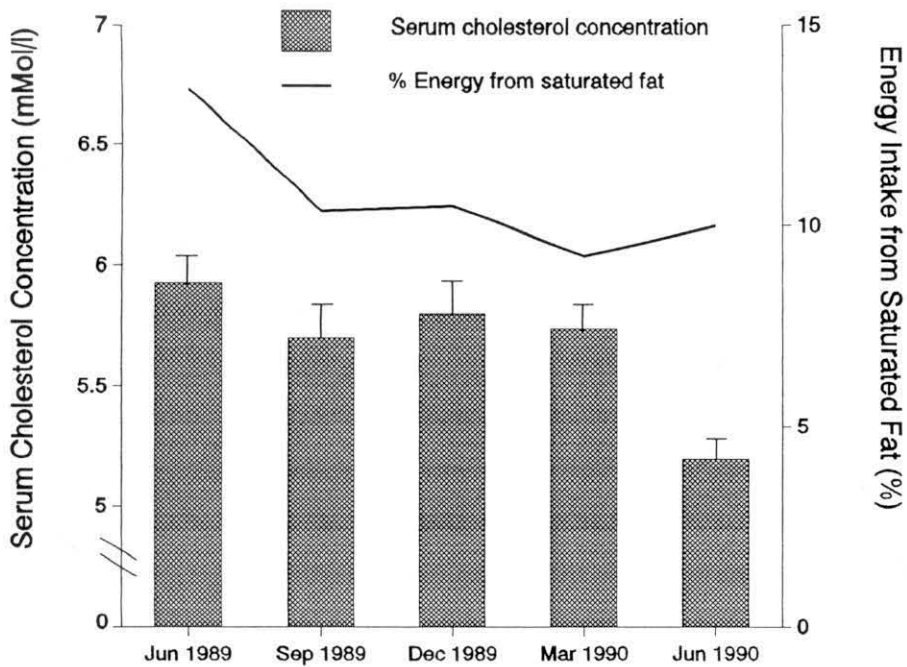


Figure 7.1. Percentage of energy intake derived from saturated fat and the mean serum cholesterol concentration over the intervention period

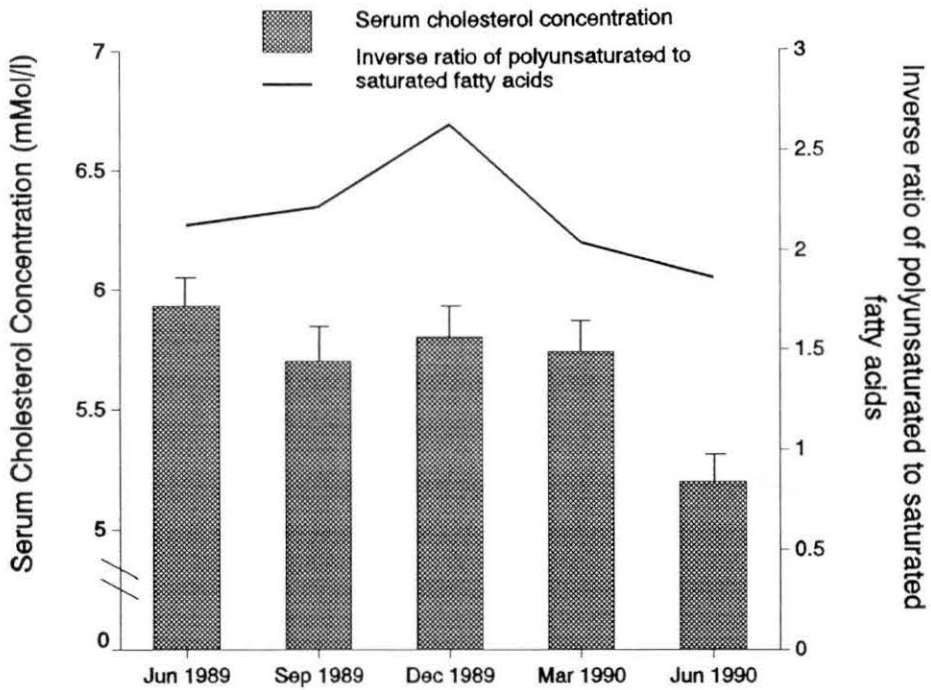


Figure 7.2. The inverse ratio of polyunsaturated to saturated fatty acids and the mean serum cholesterol concentration over the intervention period

In the second model, after controlling for inter-individual variance, increase in the density of dietary fibre intake significantly explained the reduction in the concentration of total serum cholesterol over the intervention period (Table 7.6). Total serum cholesterol concentration was not significantly affected by individual change over time in BMI, smoking status, alcohol intake, kava consumption or density of dietary cholesterol intake ($F_{1,160}=2.24$, ns).

After adjustment for inter-individual variance, survey number fitted as a factor explained 13.1% of the remaining variation in total serum cholesterol concentration, of which density of dietary fibre intake alone explained 81.1%. After adjustment for inter-individual variance, survey number fitted as an ordinal (linear) variable explained 10.8% of the remaining variation in total serum cholesterol concentration, of which density of dietary fibre intake alone explained 99.1%. Therefore change in the density of intake of dietary fibre as measured by the store-turnover method effectively explained most of the linear and non-linear intra-individual change in total serum cholesterol concentration occurring over the intervention period.

The inverse density of intake of dietary fibre and the mean serum cholesterol concentration over the intervention period are depicted graphically (Figure 7.3).

Table 7.6. Analysis of intra-individual variance of serum cholesterol concentration over the intervention period (including intake of dietary fibre)

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	340.51	237					
Subject ID	65.56	165	275.0	72	3.819	10.79	<0.001
Density of dietary fibre intake	58.56	164	7.004	1	7.004	19.8	<0.001
Survey no. (ordinal)	58.49	163	0.067	1	0.067	0.19	ns
Survey no. (factor)	56.96	161	1.531	2	0.766	2.16	ns
residual	-	-	56.96	161	0.354		

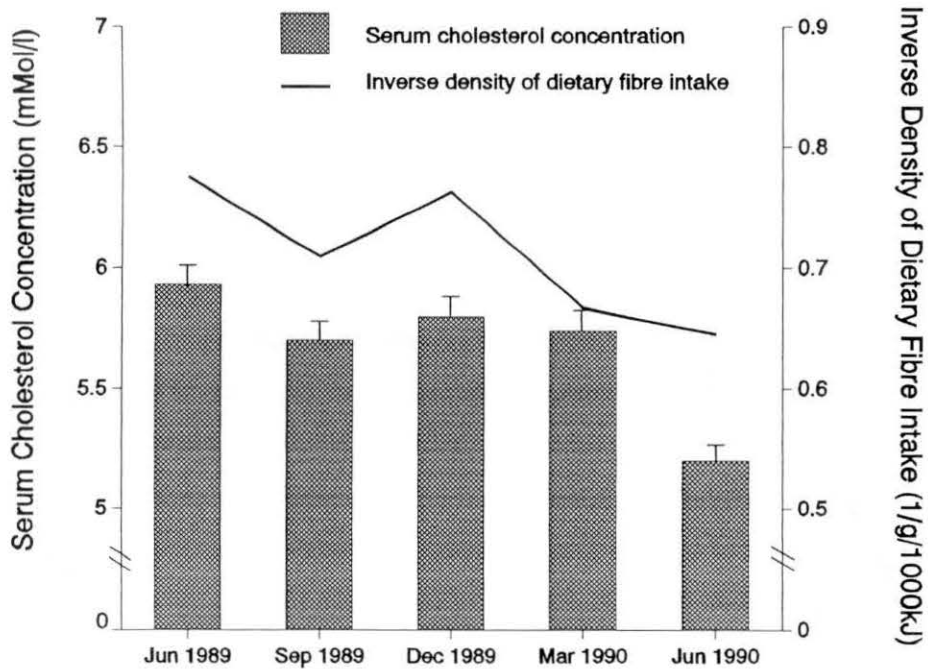


Figure 7.3. The inverse density of dietary fibre intake and the mean serum cholesterol concentration over the intervention period

7.1.3.2. HDL-cholesterol concentration

The relationship between individual change in serum HDL-cholesterol concentration over the intervention period and dietary change as measured by the store-turnover method, together with change in individual status of cigarette smoking, kava and alcohol consumption over the intervention period, was investigated by analysis of intra-individual variance. Dietary variables investigated included total energy intake, the proportion of total energy intake derived from fat, saturated fatty acids, total carbohydrate, refined carbohydrate and sugars, and the density of intake of dietary fibre. After controlling for inter-individual variance and the effect of change in total serum cholesterol concentration³ over the intervention period, little variance in serum HDL-cholesterol concentration remained, and no dietary variable significantly explained the small residual variance.

7.1.3.3. Fasting serum triglyceride concentration

The relationship between individual change in fasting serum triglyceride concentration over the intervention period and dietary change as measured by the store-turnover method, together with change in individual BMI and status of cigarette smoking, kava and alcohol consumption, was investigated by analysis of intra-individual variance. Dietary variables investigated included total energy intake, the proportion of total energy intake derived from fat, saturated fatty acids, total carbohydrate, refined carbohydrate and sugars, and the density of intake of dietary fibre. After controlling for inter-individual variance, little variance in fasting serum triglyceride concentration remained, and no dietary variable significantly explained the small residual variance.

7.1.4. Change in status of glucose tolerance and style of diet over the intervention period

7.1.4.1. Two-hour plasma glucose concentration

The relationship between the individual change in two-hour plasma glucose concentration over the intervention period and dietary change as measured by the store-turnover method, together with change in individual BMI and status of cigarette smoking, kava and alcohol

³ The ratio of HDL-cholesterol to total cholesterol may be a better indicator of risk of ischaemic heart disease than total cholesterol concentration *per se* (section 1.6.5); hence serum HDL-cholesterol level was controlled for total serum cholesterol concentration.

consumption, was investigated by analysis of intra-individual variance. Dietary variables investigated included total energy intake, the proportion of total energy intake derived from fat, saturated fatty acids, total carbohydrate, refined carbohydrate and sugars, and the density of intake of dietary fibre. After controlling for inter-individual variance no dietary variable was found to significantly explain the very small intra-individual change in two-hour glucose concentration over the intervention period.

7.1.4.2. Fasting serum fructosamine concentration

The relationship between change in individual fasting serum fructosamine concentration over the intervention period and dietary change as measured by the store-turnover method, together with change in individual BMI and status of cigarette smoking, kava and alcohol consumption, was investigated by analysis of intra-individual variance. Dietary variables investigated included total energy intake, the proportion of total energy intake derived from fat, saturated fatty acids, total carbohydrate, refined carbohydrate and sugars, and the density of intake of dietary fibre. After controlling for inter-individual variance, little variance in fasting serum fructosamine concentration remained, and no dietary variable significantly explained the small residual variance in intra-individual fasting serum fructosamine concentration over the intervention period.

7.1.5. Change in serum γ gt concentration and dietary intake over the intervention period

The relationship between change in individual serum γ gt concentration over the intervention period and dietary change as measured by the store-turnover method, together with change in the individual BMI and status of cigarette smoking, kava and alcohol consumption, was investigated by analysis of intra-individual variance. Dietary variables investigated included total energy intake, the proportion of total energy intake derived from fat, saturated fatty acids, total carbohydrate, refined carbohydrate and sugars, and the density of intake of dietary fibre. After controlling for inter-individual variance, the change in intra-individual serum γ gt concentration over the intervention period was not significantly explained by change in individual BMI, status of kava consumption ($F_{1,155}=0.88$), alcohol consumption or change in intake of any dietary variable.

7.1.6. Change in serum ferritin concentration and dietary intake over the intervention period

The relationship between serum ferritin concentration and dietary density of iron intake over the intervention period, together with individual change in BMI, total serum cholesterol concentration, smoking status, kava and alcohol intake, was investigated by analysis of intra-individual variance. After controlling for subject identification code (fitted as a factor to adjust for inter-individual variance), individual serum ferritin concentration tended to be positively associated with the increased density of iron intake over the intervention period, but the relationship was not statistically significant ($F_{1,137}=2.05$, ns). Individual change in status of cigarette smoking, alcohol and kava consumption and change in BMI were not significantly associated with the intra-individual change in serum ferritin concentration.

7.1.7. Change in vitamin status and dietary vitamin intake over the intervention period

In investigation of the relationships between change in dietary intake and vitamin status over the intervention period, the density of the intake of respective vitamins was usually considered in an attempt to control for confounding due to change in total energy intake. Actual vitamin intake was employed in the relevant statistical models only where it had been shown to be independent of total energy intake (section 4.10).

7.1.7.1. Red blood cell folate concentration

The relationship between individual change in red blood cell folate concentration over the intervention period and change in the dietary intake of folate as measured by the store-turnover method, together with change in individual BMI and status of cigarette smoking, kava and alcohol consumption, was investigated by analysis of intra-individual variance (Table 7.7).

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variance), the change in individual red blood cell folate was significantly explained by the change in density of folate intake as measured by the store-turnover method. Individual change over time in BMI, smoking status, alcohol intake and kava consumption did not significantly explain the individual change in red blood cell folate

concentration over the intervention period.

After adjustment for inter-individual variance, survey number fitted as a factor explained 60.0% of the remaining variance in red blood cell folate concentration, of which change in density of folate dietary intake alone explained 66.7%. After adjustment for inter-individual variance, survey number fitted as an ordinal (linear) variable explained 52.9% of the remaining variance in red blood cell folate concentration, of which density of folate intake alone explained 76.8%.

Therefore change in the dietary intake of folate as measured by the store-turnover method effectively explained most of the linear and non-linear intra-individual change in red blood cell folate concentration over the intervention period.

Table 7.7. Analysis of intra-individual variance of red blood cell folate concentration over the intervention period

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	638565	223					
Subject ID	452139	155	186427	68	2742	2.29	<0.001
Density of Folate intake	271086	154	181053	1	181053	151.3	<0.001
Survey no. (linear)	212948	153	58137	1	58137	48.6	<0.001
Survey no. (factor)	180802	151	32146	2	16073	13.4	<0.005
residual	-	-	180802	151	1197		

Density of folate intake and the mean red blood cell folate concentration were depicted graphically (figure 7.4).

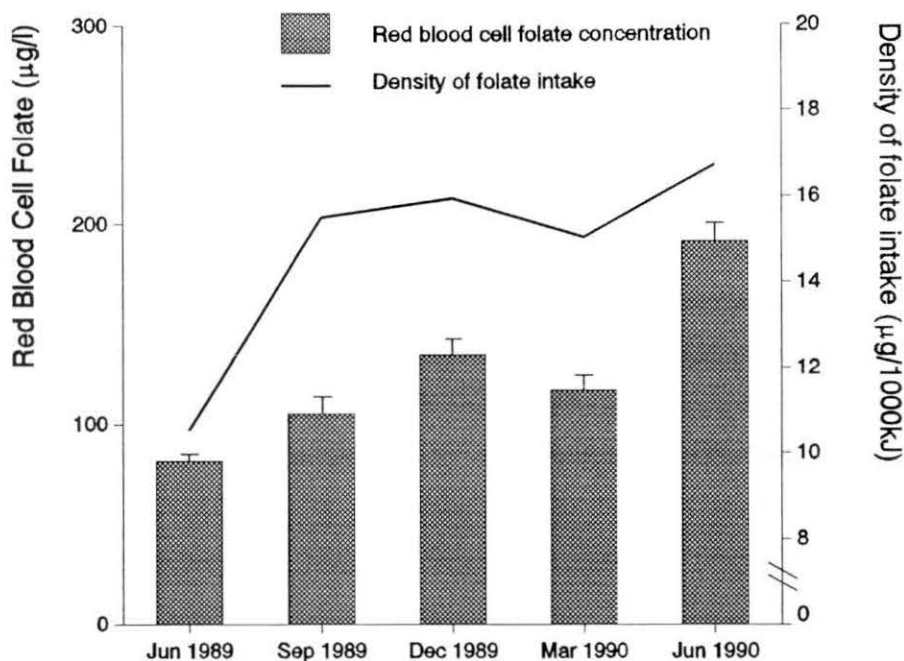


Figure 7.4. Density of folate intake and mean red blood cell folate concentration over the intervention period

7.1.7.2. Serum folate concentration

The relationship between individual change in serum folate concentration over the intervention period and change in the dietary intake of folate as measured by the store-turnover method, together with individual change in BMI and status of cigarette smoking, kava and alcohol consumption, was investigated by analysis of intra-individual variance (Table 7.8).

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variance), the change in individual serum folate was significantly explained by the change in density of folate intake as measured by the store-turnover method. Individual change over time in BMI, smoking status, alcohol and kava consumption did not significantly affect the change in the individual serum concentration of folate over the intervention period.

After adjustment for inter-individual variance, survey number fitted as a factor explained 26.8% of the total intra-individual variance in serum folate concentration, of which density of folate intake explained 28.1%. After adjustment for inter-individual variance,

survey number fitted as an ordinal (linear) variable explained 18.3% of the remaining variance in serum folate concentration; density of folate intake explained 46.3% of this total linear change in serum folate concentration over the intervention period.

Dietary intake of folate over the previous three month period explained a greater percentage of the intra-individual variance in red blood cell folate concentration than serum folate concentration. This would be expected due to the fluctuations in serum folate concentration in response to change in dietary folate intake in the short term.

Table 7.8. Analysis of intra-individual variance of serum folate concentration over the intervention period

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	376.07						
Subject ID.	135.96	128	240.1	68	3.5	4.44	<0.001
Density of Folate intake	125.7	127	10.3	1	10.3	12.9	<0.001
Survey no. (linear)	113.8	126	11.9	1	11.9	14.9	<0.001
Survey no. (factor)	99.5	125	14.3	1	4.3	17.9	<0.001
residual	-	-	99.5	125	0.80		

7.1.7.3. Serum vitamin B₆ concentration

The relationship between individual change in serum vitamin B₆ concentration and dietary intake of vitamin B₆ over the intervention period, together with individual change in BMI, smoking status, kava and alcohol intake, was investigated by analysis of intra-individual variance (Table 7.9).

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variance), the change in individual serum vitamin B₆ concentration was significantly explained by the change in density of vitamin B₆ intake as measured by the

store-turnover method. Individual change in BMI, smoking status, alcohol intake and kava consumption did not significantly affect the intra-individual change in serum vitamin B₆ concentration over the intervention period.

After adjustment for inter-individual variance, survey number fitted as a factor explained 27.5% of the remaining variance in serum vitamin B₆ concentration, of which dietary vitamin B₆ intake alone explained 98.9%. After controlling for inter-individual variance, survey number fitted as an ordinal (linear) variable, also explained 27.5% of the remaining variation in serum vitamin B₆ concentration, of which dietary vitamin B₆ intake alone explained 99.0%.

Therefore dietary intake of vitamin B₆ as determined by the store-turnover method effectively explained most of the linear and non-linear change in intra-individual serum vitamin B₆ concentration over the intervention period.

Table 7.9. Analysis of intra-individual variance of serum vitamin B₆ concentration over the intervention period

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	123619	206					
Subject ID	57833	135	65786	71	926.6	2.9	<0.001
Vitamin B ₆ intake	42092	134	15741	1	15741	49.6	<0.001
Survey no. (ordinal)	41930	133	162.1	1	162.1	0.51	ns
Survey no. (factor)	41914	132	15.4	1	15.4	0.05	ns
residual	-	-	41914	132	317.5		

The relationship between serum vitamin B₆ concentration and dietary vitamin B₆ intake is depicted graphically (Figure 7.5).

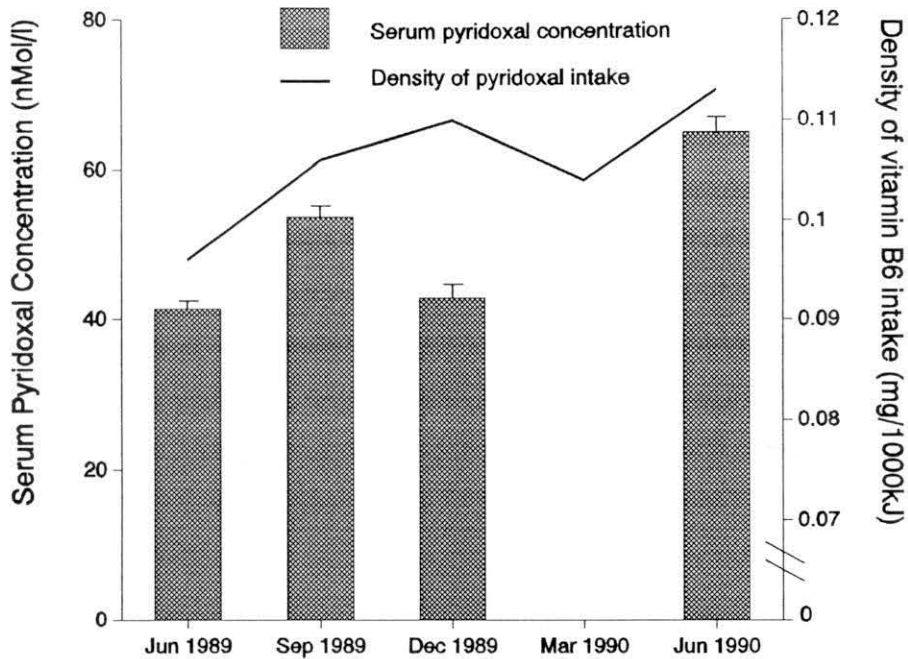


Figure 7.5. Vitamin B₆ intake and mean serum vitamin B₆ concentration over the intervention period

7.1.7.4. Serum vitamin B₁₂ concentration

The relationship between serum vitamin B₁₂ concentration and dietary intake of vitamin B₁₂ over the intervention period, together with individual change in BMI, smoking status, kava and alcohol intake, was investigated by analysis of intra-individual variance (Table 7.10).

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variance), the change in individual serum vitamin B₁₂ concentration was significantly explained by the change in density of vitamin B₁₂ intake as measured by the store-turnover method. Individual change over time in BMI, smoking status, alcohol intake and kava consumption did not significantly affect the intra-individual change in serum vitamin B₁₂ concentration over the intervention period.

After removing inter-individual variance, survey number fitted as a factor explained 14.3% of the remaining variance in serum vitamin B₁₂ concentration, of which dietary density of vitamin B₁₂ intake explained 14.3%. After removing inter-individual variance, survey number fitted as an ordinal (linear) variable explained only 4.3% of the remaining variance in serum vitamin B₁₂ concentration, of which density of vitamin B₁₂ intake alone

explained 99.1%.

Therefore dietary intake of vitamin B₁₂ as determined by the store-turnover method effectively explained most of the linear change in intra-individual serum vitamin B₁₂ concentration over the intervention period, but did not significantly explain the majority of the non-linear change in intra-individual serum vitamin B₁₂ concentration.

Table 7.10. Analysis of intra-individual variance of serum vitamin B₁₂ concentration over the intervention period

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	11140270	206					
Subject ID	4538915	135	6601355	71	92977	3.2	<0.001
Density of vitamin B ₁₂ intake	4344844	134	194072	1	194072	6.6	<0.01
Survey no. (ordinal)	4343178	133	1666	1	1666	0.06	ns
Survey no. (factor)	3889462	132	453716	1	453716	15.4	<0.001
residual	-	-	3889462	132	29466		

7.1.7.5. Red blood cell thiamine concentration

The results of the red blood cell thiamine assays were much higher at survey three (December 1989), than at other times throughout the study. Consideration of the relationship between individual results of red blood cell thiamine concentration over all surveys, suggested that an erratic episode occurred at survey three (December 1989). Potential explanations for the inconsistent results included the possibility of laboratory artefact and/or an episodic change in diet. However, the fact that the apparent increase in red blood cell thiamine at survey three was unevenly distributed throughout the population, suggested that the increase was due to individual change in diet rather than due to laboratory artefact. This possibility was also supported by the increase in mean serum thiamine concentration also measured in December 1989.

To control for this episode in analysis of intra-individual variance in red blood cell thiamine concentration, the overall linear trend was fitted first and then the third survey was fitted specifically. The relationship between red blood cell thiamine and dietary intake of thiamine over the intervention period, together with individual change in BMI, smoking status, kava and alcohol intake, was then investigated by analysis of intra-individual variance (Table 7.11). Results indicated that individual change in red blood cell thiamine concentration over the intervention period was explained significantly by the dietary density of thiamine intake as measured by the store-turnover method. Individual change over time in BMI, smoking status, alcohol intake and kava consumption did not significantly affect the change in the concentration of red blood cell thiamine over the intervention period.

After removing inter-individual variance, survey number fitted as a factor ($F_{1,130}=119.5$, $p<0.001$), explained 67.6% of the remaining variance in red blood cell thiamine concentration, of which dietary density of thiamine intake explained only 7.2%. After removing inter-individual variance, survey number fitted as an ordinal (linear) variable explained 37.7% of the remaining variance in red blood cell thiamine concentration, of which density of thiamine intake explained only 12.9%. Survey three (December 1990) accounted for almost the entire residual non-linear effect of survey number on red blood cell thiamine concentration.

As survey three was also subsequently found to account for effectively all of the residual non-linear effect of survey number on serum thiamine concentration (section 7.1.7.5), it could be speculated that this had arisen due to dietary change from non-store purchased sources in December 1989 (see section 8.2.6.2).

Table 7.11. Analysis of intra-individual variance of red blood cell thiamine concentration over the intervention period

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	143567	205					
Subject ID	99595	134	43972	71	619.3	2.5	<0.001
Density of thiamine intake	94738	133	4857	1	4857	19.6	<0.001
Survey no. (ordinal)	62101	132	32637	1	32637	132.0	<0.001
Survey no.3	32399	131	29702	1	29702	120.1	<0.001
residual	-	-	32399	131	247.3		

7.1.7.6. Serum thiamine concentration

The relationship between serum thiamine concentration and dietary intake of vitamin B₁₂ over the intervention period, together with individual change in BMI, smoking status, kava and alcohol intake, was investigated by analysis of intra-individual variance (Table 7.12).

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variance), individual change in serum thiamine concentration over the intervention period was significantly explained by the dietary density of thiamine intake as measured by the store-turnover method. Individual change over time in BMI, smoking status, alcohol intake and kava consumption did not significantly affect the change in serum thiamine concentration over the intervention period.

After removing inter-individual variance, survey number fitted as a factor explained 55.5% of the remaining variance in serum thiamine concentration, of which dietary density of thiamine intake explained 26.6%. After removing inter-individual variance, survey number fitted as an ordinal (linear) variable explained 15.1% of the remaining variance in serum thiamine concentration, of which density of thiamine intake explained 97.5%.

Therefore the dietary intake of thiamine density as measured by the store-turnover method effectively explained most of the linear intra-individual change in serum concentration of thiamine over the intervention period, but only explained a relatively small proportion of the seasonal change in serum thiamine concentration. Survey three at December 1989 ($F_{1,132}=119$, $p<0.001$) was subsequently found to account for effectively all of the residual non-linear effect of survey number on serum thiamine concentration.

Table 7.12. Analysis of intra-individual variance of serum thiamine concentration over the intervention period

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	2639.5	206					
Subject ID	1556.3	135	1083	71	15.25	2.91	<0.001
Density of thiamine intake	1326.8	134	229.5	1	229.5	43.76	<0.001
Survey no. (ordinal)	1320.9	133	5.969	1	5.969	1.14	ns
Survey no. (factor)	692.19	132	628.7	1	628.7	119.9	<0.001
residual	-	-	692.19	132	5.244		

7.1.7.7. Plasma ascorbic acid concentration

The relationship between individual change in plasma ascorbic acid concentration and density of dietary intake of ascorbic acid over the intervention period, together with individual change in BMI, smoking status, kava and alcohol intake, was investigated by analysis of intra-individual variance (Table 7.13).

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variance), the change in individual plasma ascorbic acid concentration was significantly explained by the change in density of ascorbic acid intake as measured by the

store-turnover method. Individual change over time in BMI, smoking status, alcohol intake and kava consumption did not significantly affect the intra-individual change in plasma ascorbic acid concentration over the intervention period.

After adjusting for inter-individual variance, survey number fitted as an ordinal (linear) variable explained 10.0% of the remaining variance in plasma ascorbic acid concentration, of which density of dietary ascorbic acid intake alone explained 99.2%.

Therefore density of dietary intake of ascorbic acid as determined by the store-turnover method effectively explained most of the change in intra-individual plasma ascorbic acid concentration over the intervention period.

Table 7.13. Analysis of intra-individual variance of plasma ascorbic acid concentration over the intervention period

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	2632168	158					
Subject ID	1336120	89	1296048	69	18783	1.36	ns
Density of Ascorbic Acid Intake	1203834	88	132286	1	132286	9.57	<0.005
Survey no. (ordinal)	1202763	87	1072	1	1072	0.08	ns
residual	-	-	1202763	87	13825		

The relationship between plasma ascorbic acid concentration and dietary density of ascorbic acid intake is depicted graphically (Figure 7.6).

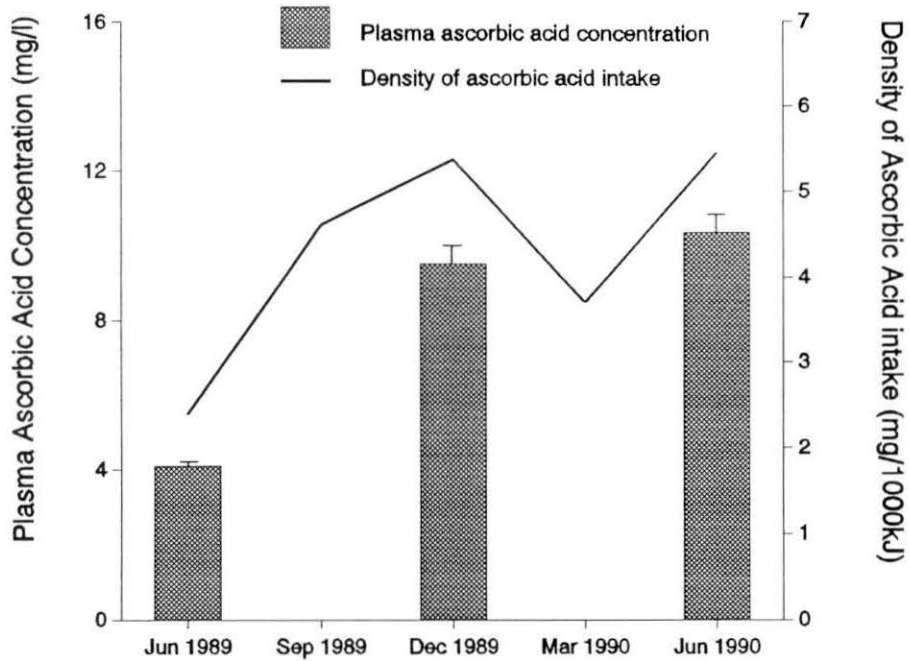


Figure 7.6. Dietary ascorbic acid intake and mean plasma ascorbic acid concentration over the intervention period

7.1.7.8. Plasma β -carotene concentration

The relationship between plasma β -carotene concentration and dietary intake of β -carotene over the intervention period, together with individual change in BMI, smoking status, kava and alcohol intake, was investigated by analysis of intra-individual variance (Table 7.14).

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variance), individual change in plasma β -carotene concentration was significantly explained by dietary density of β -carotene intake as measured by the store-turnover method. Individual change over time in BMI, smoking status, alcohol intake and kava consumption did not significantly affect the change in plasma β -carotene concentration over the intervention period.

After controlling for inter-individual variance, survey number fitted as an ordinal (linear) variable explained 20.7% of the remaining variation in plasma β -carotene concentration, of which dietary β -carotene intake alone explained 63.1%.

Table 7.14. Analysis of intra-individual variance of plasma β -carotene concentration over the intervention period

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	932935	131					
Subject ID	342524	72	590411	59	10007	2.58	< 0.01
Density of β -carotene intake	297830	71	44693	1	44693	11.50	< 0.005
Survey no. (ordinal)	271712	70	26119	1	26119	6.73	< 0.05
residual	-	-	271712	70	3882		

Therefore the dietary intake of β -carotene alone, as measured by the store-turnover method, explained most of the linear change in plasma concentration of β -carotene over the intervention period. The relationship between plasma β -carotene concentration and dietary density of β -carotene intake is depicted graphically (Figure 7.7).

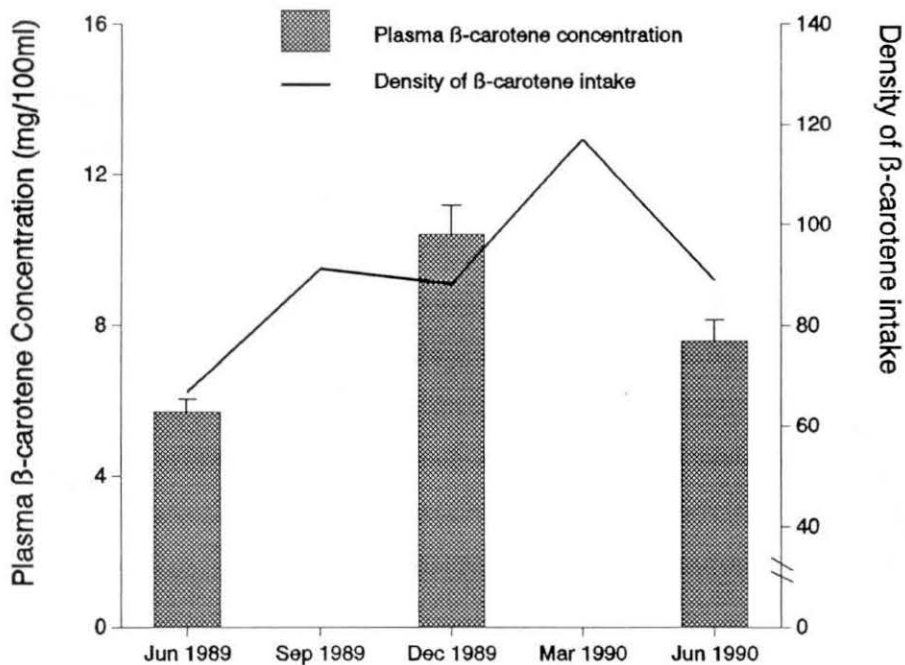


Figure 7.7. Dietary β -carotene intake and mean plasma β -carotene concentration over the intervention period

7.1.7.9. Plasma retinol concentration

The relationship between plasma retinol concentration and dietary density of total vitamin A intake, density of retinol intake and density of carotene intake over the intervention period, together with individual change in BMI, smoking status, kava and alcohol intake, was investigated by analysis of intra-individual variance.

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variance), individual change in plasma retinol concentration was not explained by change in density of total vitamin A intake ($F_{1,67}=0.78$, ns), density of retinol intake ($F_{1,67}=0.28$, ns) or density of carotene intake ($F_{1,67}=3.15$, ns) over the intervention period. Individual change in BMI, status of cigarette smoking, alcohol and kava consumption were not significantly associated with the small intra-individual change in plasma retinol concentration.

7.1.7.10. Plasma α -tocopherol concentration

The relationship between plasma α -tocopherol concentration and dietary density of α -tocopherol intake over the intervention period, together with individual change in BMI, smoking status, kava and alcohol intake, was investigated by analysis of intra-individual variance.

After controlling for inter-individual variance, neither change in density of α -tocopherol nor other variables significantly explained the change in intra-individual variance of plasma α -tocopherol over the intervention period.

However, as α -tocopherol is transported within the low density lipoprotein fraction of the blood, plasma levels are highly correlated with total plasma lipid concentration; plasma α -tocopherol concentration has been previously shown to reflect long term dietary intake of vitamin E once adjusted for total plasma cholesterol (section 1.7.4). Therefore additional analysis was conducted which first controlled for the effect of change in total serum cholesterol concentration on plasma α -tocopherol concentration over the intervention period (Table 7.15).

After controlling for subject identification code (fitted as a factor to adjust for inter-individual variance), and for change in individual serum cholesterol concentration, change

in the dietary density of α -tocopherol intake (as measured by the store-turnover method) significantly explained intra-individual change in the concentration of plasma α -tocopherol over the intervention period. Individual change over time in BMI, smoking status, alcohol intake and kava consumption did not significantly affect the concentration of plasma α -tocopherol over the intervention period.

After controlling for inter-individual variance, survey number fitted as an ordinal (linear) variable explained 18.8% of the remaining variation in plasma α -tocopherol concentration, of which density of dietary α -tocopherol intake alone explained 37.5%. After also controlling for change in individual serum cholesterol concentration over the intervention period, density of intake of α -tocopherol explained 99.6% of the remaining variance due to survey number.

Therefore, after controlling for total serum cholesterol concentration, density of dietary intake of α -tocopherol (as determined by the store-turnover method) effectively explained most of the linear change in intra-individual plasma α -tocopherol concentration over the intervention period.

Table 7.15. Analysis of intra-individual variance of plasma α -tocopherol concentration over the intervention period

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	1944.0	139					
Subject ID	512.9	79	1431	60	23.9	4.35	<0.001
Serum Cholesterol concentration	452.9	78	60.0	1	60.0	10.95	<0.001
Dietary α -tocopherol intake	420.8	77	32.1	1	32.1	5.86	<0.05
Survey no. (ordinal)	416.7	76	4.11	1	4.11	0.75	ns
residual	-	-	416.7	76	5.48		

The relationship between plasma α -tocopherol concentration adjusted for total serum cholesterol and the dietary density of α -tocopherol intake is depicted graphically (Figure 7.8).

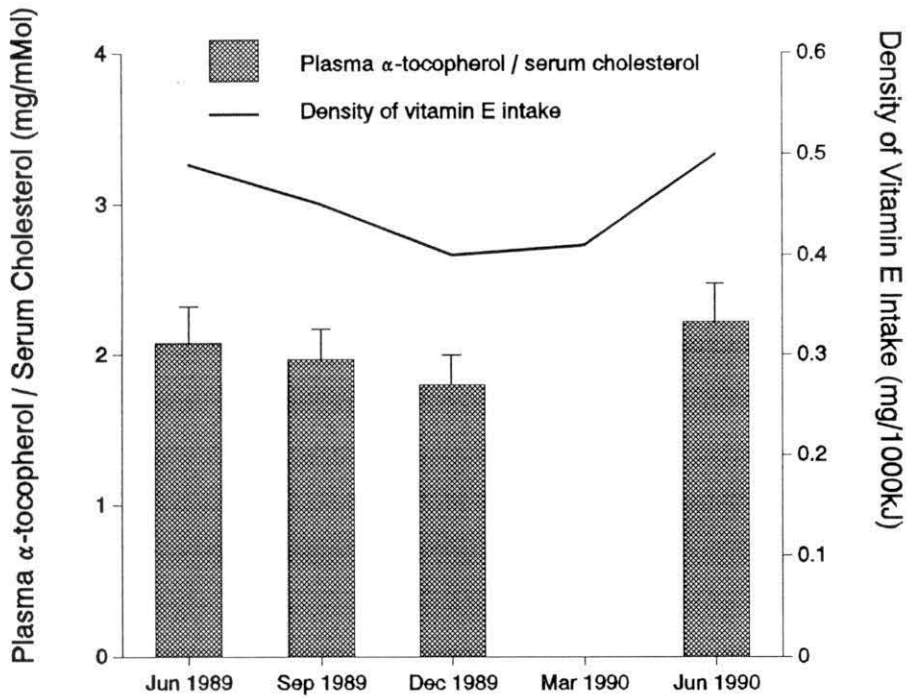


Figure 7.8. Dietary α -tocopherol intake and mean plasma α -tocopherol concentration over the intervention period

7.2. Further aspects of validity

7.2.1. Face validity

The results of the store-turnover method reflected previously reported qualitative descriptions of the diet in remote Aboriginal communities. Although the quantitative intakes of some foods (such as sugar) appeared high and others (such as fruit and vegetables) appeared low, results were of the same order of magnitude as previously described in other remote Aboriginal communities (section 1.8.11).

The consistency of dietary intake described in the control community (Chapter 5) also supported the face validity of the method.

7.2.2. Internal consistency

Investigation of various aspects of internal consistency suggested that there was a strong rational basis to the store-turnover method as a measure of dietary intake in remote, centralised Aboriginal communities.

7.2.2.1. Seasonal variation

There was indication of the effect of seasonality in the turnover of several specific foods and the nutrients they provided. There appeared to be a climatic basis for most of the observed trends, with an increase in cool beverages and ready-baked bread during the hot, humid months of the year and an increase in hot 'take-away' foods and cooking oils during the coolest months (Table 7.16).

In contrast, the turnover of some foods which would not be expected to vary with climate tended to be more consistent throughout the year (Table 7.17).

Table 7.16. Mean turnover of some specific foods affected by season per three month period (mean \pm se)

Food	Minjilang*		Control community	
	Wet	Dry	Wet	Dry
Cold beverages (litres)	6388 \pm 260	4363 \pm 212	9500 \pm 369	7244 \pm 303
Ice cream (litres)	1335 \pm 82	824 \pm 60	2397 \pm 81	1038 \pm 64
Hot take-away foods (kg)	975 \pm 62	650 \pm 46	872 \pm 61	717 \pm 63
Tomato sauce (kg)	98 \pm 9	65 \pm 4	157 \pm 10	90 \pm 8
Oatmeal (kg)	2.9 \pm 0.2	6.6 \pm 0.4	3.4 \pm 0.4	8.8 \pm 0.8

* excludes intervention year at Minjilang

Table 7.17.

Mean turnover of some specific foods not affected by season per three month period (mean±se)

Food	Minjilang*	Control community
Rice (kg)	264±17	378±28
Sweet biscuits (kg)	126±14	165±18
Jam (kg)	5.4±0.6	6.1±0.5
Tea leaf (kg)	166.2±10.7	92.1±5.4
Coffee (kg)	4.8±0.3	3.1±0.3
Salt (kg)	47.6±2.9	48.9±3.0

* excludes intervention year at Minjilang

The increase in the proportion of total energy derived from sugars at both communities during the warmest and most humid months of the year was associated with the increased turnover of cool sweetened beverages at that time. The per capita intake of sugar *per se* tended to remain relatively constant throughout the year.

Although there was no evidence of a seasonal effect on the proportion of total energy derived from complex carbohydrate at either community, the source of the complex carbohydrate tended to change throughout the year. The intake of flour increased during the coolest months when more cooking fires were used and outstations were visited, and ready baked bread was relatively more popular throughout the wet season.

The relative seasonal increase in the proportion of total energy intake derived from total fat during the cooler months of the year was associated with the increased turnover of fatty hot 'take-away' foods (particularly meat pies) and cooking oil (used for frying meat and damper) during these months.

The variation in the density of ascorbic acid intake at Minjilang was associated both with the increased availability of citrus fruit when in season (June, July and August) and the increased turnover of fruit juices during the hot 'build-up' months (September, October and November). Similarly, the seasonal variation of β -carotene at Minjilang corresponded to the relative increase in fruit juice intake and the increased availability of stone fruits during the hotter months; at all times of the year, however, yellow vegetables remained

the major source of β -carotene. Of all vitamins, the intake of β -carotene tended to be most variable in both communities. This has been a common finding in dietary surveys in many populations (section 1.7.2) and would be expected to arise from the variable intake of a limited number of foods which are exceptionally rich in β -carotene.

There tended to be a seasonal pattern in density of vitamin B₁₂, iron and zinc intake as measured by the store-turnover method. At both communities the intake of these nutrients tended to be higher during the wet season months between December and April. One logical explanation may have been the relatively greater availability/accessibility of traditional animal foods particularly fish, turtle, dugong and magpie geese, during the dry season, which would have reduced the demand for meat and meat products from the store. This effect was more marked at Minjilang than at the control community but appeared to diminish after 1989. There was no evidence of increased reliance on traditional animal foods at Minjilang prior to that time, however the council president (Mr Jumbo Goongiburra) did state that commercial fishermen had reduced barramundi numbers on the western side of the island following the 1988 season.

The effects of seasonality on store-turnover could have been due to either changing population within the community, to overstocking of non-perishable items or to real changes in dietary intake. The fact that apparent consumption of some foods remained consistent over the seasons when controlled for population change, while nutrient profiles changed over the seasons, suggested that real changes in the intake of specific foods did occur.

7.2.2.2. Associated food consumption patterns

The store-turnover method was able to reflect consistently the relationship between foods consumed in association with each other independently of season. At Minjilang and the control community respectively, the ratio of plain flour to baking powder was 146.6 ± 6.8 and 154.7 ± 7.0 and the ratio of sugar to tea leaf was 38.8 ± 1.8 and 42.4 ± 1.6 .

7.2.2.3. Store equipment and developments in food technology

Changes in both the total fat and the P:S ratio at the two communities appeared to be related to the introduction of new store equipment. P:S ratio decreased at Minjilang with the introduction of a 'take-away' section incorporating a new pie-warmer and rotisserie in

May 1988 (section 5.3.1). The P:S ratio at the control community also dropped following the installation of a bain-marie for display of deep fried foods at the community store in July 1987. These observations further supported the rational basis of store-turnover results.

The store-turnover method also reflected the impact of developments in food technology on the diet of Aboriginal communities; apparent intake of fruit juice increased at both communities following introduction of ultra-heat treated (UHT) fruit juice which did not require refrigeration (section 5.2.7).

7.2.2.4. Store managers

Many store managers were employed at either Minjilang or the control community during the five year period described. However two store managers were each employed at both communities for known terms during this period. Therefore opportunity existed to examine the relationship between the terms of appointment of these store managers and apparent dietary intake of the respective communities.

One store manager (Store Manager 'A') was particularly committed to both improving the nutritional quality of food available and training Aboriginal store workers. She had been employed at Minjilang from September 1987 to September 1988 and was also employed as locum manager at the control community from February to June 1989. These dates coincided with the periods of highest measured turnover of fresh fruit and vegetables (figure 5.1) and also the highest density of nutrients supplied by those foods prior to the commencement of the nutrition intervention project (folate (figure 5.16), ascorbic acid (figure 5.22), β -carotene (figure 5.24) and dietary fibre (figure 5.30)). During the period when both stores were managed by Store Manager 'A' there was also an increase in the turnover of wholegrain bread (figure 5.3) which contributed to a relative increase in the dietary density of both thiamine (figure 5.17) and dietary fibre (figure 5.30).

Another store manager (Store Manager 'B') was responsible for Minjilang store until June 1987 when he transferred to the control community as store manager until March 1989. The quantity and pattern of folate, β -carotene, ascorbic acid, thiamine and dietary fibre intake was similar in both stores at the time of his management and tended to be lower than during the period that store manager 'A' was responsible for each respective community store (figure 7.9; 7.10; 7.11).

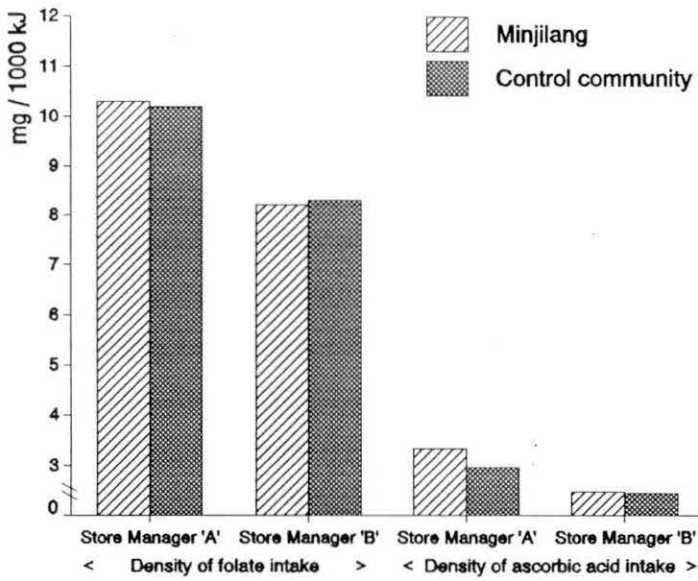


Figure 7.9. Density of folate and ascorbic acid intake at Minjilang and the control community when managed by store manager 'A' and store manager 'B'

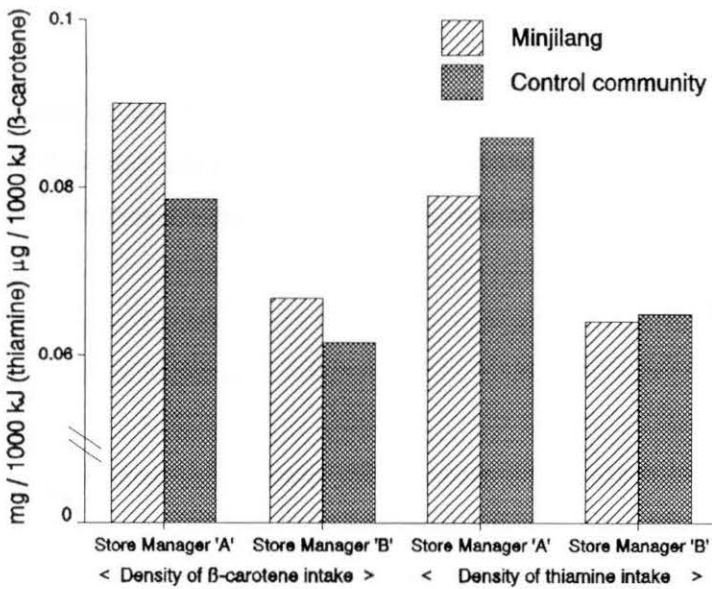


Figure 7.10. Density of beta-carotene and thiamine intake at Minjilang and the control community when managed by store manager 'A' and store manager 'B'

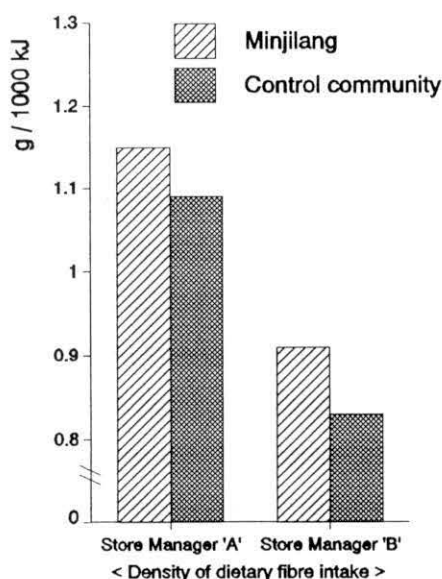


Figure 7.11. Density of dietary fibre intake at Minjilang and the control community when managed by store manager 'A' and store manager 'B'

Therefore the retrospective measure of store-turnover was not only able to reflect the effect of a different store manager, it suggested that change in store policy and/or food supply *could* influence the nutrient density of the diet in Aboriginal communities.

The association between the observed dietary variation at each community and the terms of appointment of individual store-managers over the five year period, further supported the notion that the store-turnover method was a useful approach to the quantitative consideration of dietary intake in remote Aboriginal communities.

7.3. Major points

Analysis of variance suggested that the prior changes in dietary intake of several vitamins as measured by the store-turnover method, including folate, vitamin B₆, β-carotene and ascorbic acid, significantly explained the changes in related biological variables over the intervention period (Table 7.18). For example, as determined by the store-turnover method, the change in density of dietary folate intake ($F_{1,148} = 151.2$, $p < 0.0001$) explained three quarters of the time-related change in red blood cell folate concentration over the intervention period. As expected, dietary intake of folate explained a greater percentage of

the intra-individual change in red blood cell folate concentration than serum folate concentration over the intervention period.

Furthermore, the plasma concentrations of those fat soluble vitamins which are known to be most resistant to dietary change (retinol, and α -tocopherol prior to controlling for serum cholesterol concentration), remained unaffected by the increased intake of those respective vitamins as determined by the store-turnover method.

Conversely, only a relatively small proportion of the seasonal change in both red blood cell and serum thiamine concentrations could be explained by the dietary intake of thiamine as measured by the store-turnover method; there was evidence that non-store foods were an important source of thiamine at Minjilang during the three month period prior to December 1991.

The decrease in serum cholesterol concentration over the intervention period could be statistically explained by the decrease in the reduction in the proportion of total energy intake derived from saturated fat, the increase in the ratio of polyunsaturated to saturated fatty acid intake (P/S ratio) and the increase in the density of dietary fibre intake. Due to the highly correlated nature of these dietary variables, two different models (Table 7.18) were developed in order to investigate their relationship with serum cholesterol concentration.

Change in proportional macronutrient intake, specifically reduction in the percentage of total energy intake derived from dietary fat, statistically explained the decrease in diastolic and systolic blood pressure and BMI over the intervention period (Table 7.18).

The fact that the change in store-turnover data correlated significantly with serial trends in biochemical data over the intervention period supported the notion that the store-turnover method was a valid method to measure the dietary intake of remote, centralised Aboriginal communities.

The face validity of the method and the relationship of the observed dietary variation at each community with season and with the terms of appointment of individual store-managers over the five year period, further supported this notion.

Table 7.18.

Summary of the relationship between change in biological variables and dietary intake as measured by the store-turnover method

Biological Variable	Dietary Variable	F-value	Seasonal Change %Explained	Linear Change %Explained
BMI (excluding diabetics)	% Energy as fat	$F_{1,133}=6.94$ ($p < 0.01$)	77.6	93.6
BMI (including diabetics)	% Energy as fat	$F_{1,154}=10.9$ ($p < 0.001$)	88.1	96.8
Diastolic Blood pressure	% Energy as fat	$F_{1,160}=20.7$ ($p < 0.001$)	82.4	99.6
Systolic Blood pressure	% Energy as fat	$F_{1,160}=21.6$ ($p < 0.001$)	76.4	98.8
Serum cholesterol* (first model)	% Energy as saturated fat	$F_{1,161}=7.84$ ($p < 0.001$)	32.2	39.5
"	P:S ratio	$F_{1,161}=11.1$ ($p < 0.001$)	45.5	55.8
Serum cholesterol* (second model)	Density of fibre	$F_{1,161}=19.8$ ($p < 0.001$)	81.1	99.1
HDL-cholesterol	nil	—	—	—
Red blood cell folate	Density of folate	$F_{1,151}=151.3$ ($p < 0.001$)	66.7	75.7
serum folate	Density of folate	$F_{1,125}=12.9$ ($p < 0.001$)	28.1	46.3
Serum vitamin B ₆	Vitamin B ₆	$F_{1,132}=49.6$ ($p < 0.001$)	98.9	99.0
Serum vitamin B ₁₂	Density of vitamin B ₁₂	$F_{1,132}=6.6$ ($p < 0.01$)	14.3	99.1
Red blood cell thiamine ^{***}	Density of thiamine	$F_{1,131}=19.6$ ($p < 0.001$)	7.2	12.9
Serum thiamine ^{***}	Density of thiamine	$F_{1,131}=43.8$ ($p < 0.001$)	26.6	97.5
Plasma vitamin C	Density of vitamin C	$F_{1,87}=9.57$ ($p < 0.005$)	—	99.2
Plasma retinol	nil	—	—	—
Plasma β-carotene	Density of carotene	$F_{1,70}=11.5$ ($p < 0.001$)	—	63.1
Plasma α-tocopherol	nil	—	—	—
Plasma α-tocopherol ^{***}	α-tocopherol	$F_{1,76}=6.58$ ($p < 0.05$)	—	99.6

* Two different models were developed in order to investigate the relationship between serum cholesterol concentration and dietary variables (section 7.1.3.1)

*** after controlling specifically for the third survey (section 7.1.7.5)

*** after controlling for change in serum cholesterol (section 7.1.7.10)

CHAPTER 8: DISCUSSION OF RESULTS AND IMPLICATIONS FOR FUTURE ABORIGINAL NUTRITION PROJECTS

This chapter provides a broader interpretation of all phases of the successful community-based nutrition intervention project at Minjilang including the role of the store-turnover method and details of the intervention strategies applied. This final chapter also considers developments arising from the project, implications for other community-based nutrition intervention projects in remote, centralised Aboriginal communities and further research questions.

8.1. Dietary intake

Following validation of the store-turnover method as a measure of dietary intake in remote, centralised Aboriginal communities (Chapter 7), it was justifiable to interpret and discuss the implications of the dietary results presented in Chapter 5 together with the biological results presented in Chapter 6.

8.1.1. Dietary intake prior to the Minjilang nutrition intervention project

Major results of the application of the store-turnover method at both Minjilang and the control community prior to June 1989 revealed general similarities to other Aboriginal communities (Chapter 3); intakes of energy, sugars and fat (particularly saturated fat) were excessive and nutrient densities were low. However, the diet at Minjilang in June 1989 was relatively lower in sugars and higher in fats than the control community and tended more towards the style of diet of non-Aboriginal Australia (section 3.5.2). Furthermore, a greater number of foods contributed to more than two percent of the total energy intake at Minjilang than at the control community (section 5.3.2). This supported the notion that the Minjilang diet was more varied and more 'western' than that of the control community. Various factors could have contributed to this difference. Firstly, it is known that European contact with the Iwadja and Marrgu of Croker Island was earlier and more intense than the control community (section 4.2.1). Secondly, the purpose of the missions established at both islands differed greatly. Croker Island mission was designed to totally assimilate "*part Aboriginal*" children (Cole, 1980:39) from other regions (section 4.2.1) and the local Iwadja were directly exposed to a large influx of people who were compelled

to adopt European cultural values (and diet). In contrast, at the control community there were few non-local people present at any time; despite the fact that the mission was established 24 years prior to that at Croker Island it is likely that there was less exposure to European cultural values. Thirdly, the employment of more local people at Minjilang may have contributed to the relative affluence of that community; it is possible that greater disposable income was available to be spent on food at Croker Island than at the control community.

In addition to the information disclosed in cross-sectional application of the store-turnover method, longitudinal results highlighted the marked seasonal variation in dietary intake (section 7.2.2.1). The effect of season implied that it was necessary to consider the time of year at which store-turnover was measured in interpretation of results. This was particularly so when the store-turnover method was employed in the monitoring or evaluation of a community-based nutrition intervention project; that is, the seasonal effects could confound results. For example, if pre-intervention store turnover data was collected for the three month period during the hottest months of the year, data collected during the coolest period of the year (six months later) would most likely indicate a reduction in the intake of sweetened carbonated beverages, but this would not necessarily reflect an impact of the intervention project. Therefore all subsequent discussion allowed for seasonal effects.

As in other remote, centralised Aboriginal communities (section 3.6.2.2), measurement of dietary intake suggested that dietary preferences for meat, fat and 'sweetness' had not changed from traditional times (section 1.2.3.1, section 1.5.3.1). Dietary preferences also tended to remain with the early introduced foods of flour, sugar and tea (section 1.5.2). The maintenance of conservative dietary preferences, when considered with the assumption that traditional Aboriginal beliefs did not encompass a relationship between diet and health (section 1.2.3.3), hinted at the difficulties which would confront efforts to improve nutrient intake. Dietary results suggested that innovative approaches, addressing traditional values, beliefs and attitudes, would be required to encourage consumption of a wider variety of nutritious foods.

However, longitudinal measure of store-turnover at Minjilang before June 1989 suggested that the community had already initiated dietary change prior to commencement of the intervention project. In particular there was evidence of decreasing intake of sugar *per se* over the previous four year period (section 5.2.2). This observation suggested that the

community may have already internalised an awareness of the hazards of a 'western' diet. Hence the reduction in sugar intake may have been not only a 'signpost' that the people of the Minjilang community were ready to act to improve their dietary intake in other areas, but also that they had already developed some appropriate strategies to improve their diet.

During the four years prior to June 1989, the proportion of total energy intake derived from both total and saturated fat had increased at Minjilang and immediately prior to the commencement of the intervention project, was closer to central Australian Aboriginal communities than other northern coastal communities (section 3.5.2). As sugar intake had also decreased at Minjilang over this period the dietary profile of the community tended to be both higher in the proportion of energy derived from fat and lower in the proportion of energy derived from sugar. Indeed the high intake of saturated fat at Minjilang immediately prior to June 1989 provided a rational explanation for the unusually high total serum cholesterol concentrations measured at that time (section 6.5.5). In fact the general lipid profile of the Minjilang community tended more towards that of non-Aboriginal Australia than other Aboriginal communities (section 8.2.2), and may have reflected the closer agreement between the dietary profile of Minjilang and non-Aboriginal Australia than other Aboriginal communities and wider Australia (section 3.5.2).

The fact that retrospective store-turnover was able to show the effect of different individual store managers on the dietary intake of the community supported the notion that change in store policy and/or food supply *could* influence the nutrient density of the diet (section 7.2.2.4). Although there was a trend for the sugar intake to decrease and the intake of saturated fat to increase at Minjilang prior to June 1989, there were fluctuations in these patterns associated with change in individual store managers. This confirmed that store managers still wield considerable power over the community food supply. Therefore, in addition to the need to address community values within the context of a community-based nutrition program, it was clear that store manager's beliefs and attitudes towards food and nutrition were an important consideration in the development of practical strategies to improve dietary intake.

The availability of store equipment, such as pie-warmers, bain-maries and rotisseries, and developments in food technology were also seen to influence the dietary intake of the communities (section 7.2.2.3).

8.1.2. Change in dietary intake over the intervention period

Longitudinal store-turnover data disclosed the relative seasonal changes in the turnover of targeted foods during the intervention period in Minjilang compared to the preceding four years (section 5.2). In summary, over the intervention year these changes included a marked increase in the consumption of fresh fruit and vegetables (from 83g to 183g per person per day) and wholemeal bread (from 14g to 33g per person per day), but a decrease in the turnover of fatty take-away foods, 'snack foods' and sugar (from 102g to 89g per person per day). The percentage of fat in available meat cuts was reduced by 37%. Although the store manager, on behalf of the community, constantly requested lean cuts of meat from the wholesale butchers, the most dramatic reduction occurred after the supplier was changed in early December 1989. There was also an increased proportion of nutritionally-preferred varieties of carbonated beverages (artificially sweetened) and cooking oils (polyunsaturated or monounsaturated) at Minjilang over the intervention period. These changes were in contrast to the relative stability of the turnover of these targeted foods at the control community from June 1989 to June 1990.

In summary, nutrient analysis of Minjilang store turnover data revealed a 33% decrease in the per capita intake of saturated fat, a 15% decrease in the per capita intake of sugars, a small increase in the per capita intake of dietary fibre and marked improvement in the density of intake of most vitamins over the intervention period (including a 52% increase in the per capita intake of folate and a doubling of the per capita intake of ascorbic acid).

The relative stability of the intake of sugar during the first nine months of the intervention project at Minjilang may have been due to resistance against further reduction of sugar intake following the efforts of the community during the previous four years (section 8.1.1). However, the source of sugars did change during this period, with a greater proportion of sugars being derived from fruit and vegetables and less from sweetened carbonated beverages (section 5.3.2). It was not until the final three months of the intervention period that the intake of sugar *per se* decreased; this reduction coincided with the provision of artificial sweeteners (brand name: *Equal*) at the store check-out counter. Unfortunately during the final six months of the project, sugar was also prominently displayed near the check-out, where the heavy two kilogram packages were readily toted from the store. It was felt that this practice actively promoted purchase of sugar. However, suggestions to replace the sugar on shelving within the main area of the store were not accepted by the store manager at the time.

That specific store manager, who was appointed to Minjilang in December 1989, had worked in community stores in remote Aboriginal communities for the previous 30 years, and had developed strong, personal opinions about the types of foods that Aboriginal people preferred (section 8.3.2.1). He was relatively unsupportive of the nutrition intervention project and was particularly reluctant to order large stocks of fruit and vegetables and wholemeal bread, fearing such items would remain unsold. In response, the community council arranged for a regular air charter to transport perishable foods from a mining town (Jabiru) on the mainland. Community members passed food orders and payment directly to the charter pilot. The council president asked the charter pilot to ensure that the invoices of foods supplied were included in the store turnover data. The tally of fresh produce supplied in this manner was: potatoes 30 kg; green vegetables 15 kg; other vegetables 50.3 kg; citrus fruit 32 kg; other fruit 242.2 kg; wholemeal bread 41 kg. By the end of January 1990 the store manager was forced to bow to economic pressure and restock these lines in the community store (section 8.3.2.1).

The purchase of cooking pots and equipment from the Minjilang store increased over the year. Evidence that cooking increased during the year was also provided by the rise in the turnover of dehydrated soups which were used predominantly as seasonings in stews. This subsequently, together with the rise in turnover of 'vegemite', contributed to the increase in density of sodium intake measured over the intervention period. (Salt was not specifically targeted during the intervention).

The introduction of salad sandwiches (section 8.3.2.4) helped explain the decline in high fat 'take-away' foods during the intervention period. Wholemeal bread used in the preparation of sandwiches accounted for over 75% of the total intake of wholemeal bread at Minjilang by June 1990. The popularity of wholemeal sandwiches hinted at the inaccuracy of assumptions which have been made about contemporary Aboriginal food preferences (section 8.3.2.1). However, wholemeal flour was not well accepted throughout the intervention period; suggested reasons for this included the perception that it was difficult to produce a 'good' damper from 'brown' flour, that it attracted weevils and that it was 'dirty'.

Store-turnover data was used to devise simple nutrition messages targeting key food items during the intervention project (section 8.3.1.10). Judging from various requests to store managers, it was also apparent that many community elders, particularly women, had beliefs about other foods which were considered '*strong*'; these included liver and skim

milk powder. Such beliefs may have developed from traditional values or as a result of past nutrition education programs. Various store managers also ordered stock which they considered 'healthy', including potassium salt (brand name: *Lite Salt*), glucose drinks (brand name: *Lucozade*) and special diet biscuits (brand name: *Limits*). As relationships developed, more people asked the researchers and the Senior Aboriginal Health Worker about the nutritional value of specific foods prior to purchase. In February 1990 a meeting was convened by Arnhem Land Progress Association (ALPA) in Darwin to enable store managers, researchers and nutritionists from the Northern Territory Department of Health and Community Services to meet and discuss such issues with view to formulating an ALPA nutrition policy (section 8.5).

The potential impact of a store nutrition policy was illustrated by the introduction and subsequent relatively high turnover of poly-unsaturated oil and margarine at the control community (section 5.2.8) following implementation of the ALPA nutrition policy (Addendum 3, A3.3) from March 1990.

Although the dietary intake of the community as a whole was shown to improve over the intervention period at Minjilang, food distribution patterns within the community were not addressed by application of the store-turnover method. It was therefore not possible to consider the dietary change in individual subjects throughout the project.

8.1.3. Intake of traditional foods at Minjilang

Although no formal attempt was made to quantify the individual intake of traditional bush foods during the year, many factors suggested that the mean per capita intake of traditional foods was low throughout the Minjilang community as a whole. These included problems with accessibility, the fact that many traditional foods were depleted in areas surrounding the township, that most bush foods were sought infrequently (and not always successfully) and that foods were widely shared within only a limited number of family groups. At best, even in the rich, diverse environments of Croker Island, some bush foods may have had a seasonal effect on the diet of the community. This was likely to be particularly so for nutrient dense foods (section 1.2.2.3) and those foods occasionally procured in high quantities over a relatively short period, such as the magpie geese shot

during the late dry period and the 11 turtles¹ captured in November (section 5.4.2). The seasonal patterns of hunting were directly in line with traditional observations (section 1.2.2.2). As observed in other communities, highly prized items, particularly meat and fat, were most frequently procured (section 1.5.3.2).

8.2. Biological indicators of health and nutritional status at Minjilang

8.2.1. Sample

The degree to which the Minjilang community felt involved and committed to this project was revealed by the high voluntary participation rates experienced (section 6.1). The high participation rates were remarkable given past problems with medical research in Aboriginal communities (section 1.9.4), traditional Aboriginal attitudes towards biological sampling (particularly in the context of medical research) (section 1.9.2), the supposed lack of an Aboriginal perception of the relationship between health and diet (section 1.2.3.3) and the longitudinal nature of the project involving five surveys at three month intervals (including two relatively invasive glucose tolerance tests).

Although encouragement was extended to all adult Minjilang residents in an attempt to ensure that each survey sample was complete, the wishes of those declining to participate, either directly or indirectly, were always respected immediately; no person was embarrassed by repeated invitation to take part in any screening and effort was taken to ensure that those refusing to participate did not feel excluded from either a close relationship with the researchers or the nutrition project *per se*. Therefore, the fact that subjects may have been unavailable within the community or declined to participate in biological sampling during a particular survey did not indicate that they were not actively attempting to improve their diet; that is, the numbers of subjects in each survey was not essentially related to dietary compliance within the community.

8.2.2. Risk factors of non-communicable disease: Initial findings at Minjilang in comparison to other studies

Initial serum cholesterol concentrations at Minjilang (5.93 ± 0.15 mmol/l) were

¹ The turtles may have increased serum cholesterol concentration (section 8.2.3.1) and thiamine levels (section 8.2.3) in those individuals consuming large quantities in November 1989.

surprisingly high compared to previous findings in most other remote communities (section 1.6.5.2). Very low serum cholesterol concentrations (3.9 ± 0.2 mmol/l) have been described for a small outstation community in north-east Arnhem Land (O'Dea *et al*, 1988b); similar results (3.9 ± 0.3 mmol/l) were also described in a group of healthy, lean, young Aboriginal men in the North-West Kimberley region (O'Dea *et al*, 1982). Relative to Caucasian concentrations, low total cholesterol concentrations have also been described in three small isolated desert communities (4.31 ± 0.17 mmol/l) (O'Dea *et al*, 1988a), in a decentralised Central Australian community (mean 5.4 ± 1.2 mmol/l (Gault, 1990:18-19) and in a small isolated population from Northern Australia (mean 4.49 ± 0.19 mmol/l) (O'Dea *et al*, 1990). However in these communities cholesterol concentrations were higher than those of the two more remote, less 'westernised' communities initially described above.

In comparison to the Minjilang results a similarly high mean serum cholesterol concentration (5.8 ± 0.2 mmol/l) has been described in one other location, a large central Australian community which had also been long-established, was relatively affluent and had been intensely exposed to non-Aboriginal values (and foods) for a similarly extended period (Patel *et al*, unpublished: in Gault, 1990:20). The early observation that cholesterol levels tend to rise with increasing urbanisation of Aborigines (Casley-Smith, 1959; Charnock *et al*, 1959) has been supported by more recent studies (Wise *et al*, 1976; Bastian, 1979). The results of several community-based studies illustrated a trend towards increased cholesterol levels across two age categories for both sexes associated with a measure of the degree of 'westernisation' of each community (Gault, 1990:20-21). Therefore the relatively high initial serum cholesterol concentrations at Minjilang supported the notion that the community had a more 'western' style of diet than other Aboriginal communities studied (section 8.1.1).

In a brief report from a study conducted at Yuendumu (Boyden and Agar, 1981), serum cholesterol level was found to be higher among 40 younger subjects aged 15 to 24 years, than 40 aged from 25 to 40 years, suggesting less exposure and/or resistance to dietary and other lifestyle change in older community members. At Minjilang (Addendum A2.5), the serum cholesterol concentrations of young men under 35 years (6.1 ± 0.3 mmol/l) were analogous to older men, but the serum cholesterol concentration of older women (6.3 ± 0.3 mmol/l) were surprisingly high particularly compared to that of younger women (5.1 ± 1.2

mmol/l)². These results supported the notion that the people at Minjilang had been exposed to lifestyle change for a relatively extended period.

Further, the observation that older women at Minjilang tended to have higher serum cholesterol concentrations than older men was in marked contrast to other studies in Aboriginal groups (section 1.6.5.2). However the preponderance of older women at Minjilang suggested that older men suffered relatively reduced longevity; it could be postulated that older men with the highest cholesterol concentrations may have died prior to the initiation of the intervention project at Minjilang.

Low-density lipoprotein (LDL) cholesterol and very-low-density lipoprotein (VLDL) cholesterol are positively correlated with ischaemic heart disease; high-density lipoprotein (HDL) cholesterol is negatively correlated with ischaemic heart disease (Miller and Miller, 1975; Gordon *et al*, 1977; Heiss *et al*, 1980; Castelli, 1984). A ratio of total cholesterol to HDL-cholesterol greater than 4.5 is believed to indicate increased risk of ischaemic heart disease (Castelli *et al*, 1986). At Minjilang the mean total cholesterol to HDL-cholesterol ratio (5.47 ± 0.19) was high relative to the recommended ratio. High total cholesterol:HDL-cholesterol ratios have also been described for two other remote communities (Sladden, 1987:120; Gault, 1990:14-15). However as total serum cholesterol concentrations were relatively low in these studies, the significance of the findings is unknown. Further data regarding total cholesterol:HDL-cholesterol is not available from other Aboriginal studies. However, in contrast to non-Aboriginal groups, gender differences were small when the actual HDL-cholesterol concentration was considered (Addendum A2.5); this has been a frequent finding in other Aboriginal studies (Sladden, 1987:120; Gault, 1990:14-15; O'Dea *et al*, 1990).

Although serum cholesterol levels have not generally been found to be elevated in Aboriginal communities, serum triglyceride concentrations (section 1.6.5.2) have been found consistently to be high in comparison with non-Aboriginal groups (Wise *et al*, 1976; O'Dea *et al*, 1982, 1988a, 1990; Gault, 1990:17). At Minjilang initial fasting serum triglyceride concentrations (1.81 ± 0.17 mmol/l) tended to be lower than in most other comparable Aboriginal communities, but high relative to non-Aboriginal groups. The fasting serum triglyceride concentrations were similar for each sex at Minjilang; but in all other studies serum triglyceride concentrations have been found to be higher in men than

² Although the range of cholesterol concentrations in younger women was much greater than for any other age/sex group

women. This difference was due to the relatively lower values for men at Minjilang (1.9 ± 0.5 mmol/l) compared to a mean of 2.7 ± 0.6 mmol/l for men in five other remote, centralised communities in the Northern Territory (Gault, 1990; O'Dea *et al*, 1990; Patel *et al*, unpublished; Lion *et al*, unpublished). The serum triglyceride concentrations for women at Minjilang (1.8 ± 0.2 mmol/l) were similar to those of women (1.7 ± 0.2 mmol/l) in the other five communities. As fasting serum triglyceride levels have been shown generally to be elevated in association with high alcohol consumption (Whitfield, 1981), and alcohol is more often consumed by men than women in Aboriginal communities (Watson *et al*, 1988), this difference may have reflected the fact that alcohol was formally prohibited from and rarely consumed at Minjilang. Fasting serum triglyceride levels are also associated with dietary intake of refined sugar (MacDonald, 1967). Therefore these results also suggested that the relative reduction of refined carbohydrate measured at Minjilang prior to June 1989 may have contributed already to a reduction in fasting serum triglyceride concentrations (particularly amongst the men) prior to the initiation of the nutrition intervention project. However, there was also the possibility that the older men with the highest fasting serum triglyceride concentrations may have died prior to commencement of the intervention project. Again, the results supported the notion that the lipid profile of the Minjilang community in June 1989 was closer to that of non-Aboriginal Australia (NHF, 1983, 1986) than other Aboriginal communities. As in other studies (Wise *et al*, 1976; O'Dea *et al*, 1982, 1988a, 1990; Gault, 1990:17) fasting serum triglyceride concentrations at Minjilang increased across non-diabetic, impaired glucose tolerance and diabetic groups (section 6.5.4) and were positively associated with BMI (section 6.6.6).

Although the BMI initially measured at Minjilang (23.3 ± 0.6 kg/m²) was similar to that of other remote, centralised Aboriginal communities (O'Dea *et al*, 1982, 1988a, 1990; O'Dea, 1984, 1987, 1988; Rutishauser and McKay, 1986; Gault, 1990:11; Lions *et al*, unpublished; Patel *et al*, unpublished) it was much higher than for nomadic groups. In comparison to the 'normal' Caucasian reference range of 20-25 kg/m² (Truswell, 1981), the mean BMI of nomadic adults was between 19 and 20 for men and between 18 and 20 for women with no apparent increase with age (Elphinstone, 1971; Abbie, 1971; 1975; White, 1985; O'Dea, 1987). More recently, BMI values of between 13.4 and 19.3 kg/m² (mean 17 kg/m²) have been described in apparently healthy people living a traditionally-orientated lifestyle in an outstation community in north-eastern Arnhem Land (O'Dea *et al*, 1988b). Such observations have given rise to doubts about the appropriateness of Caucasian BMI standards when applied to Aboriginal groups; in particular, Aboriginal

subjects deemed 'underweight' by Caucasian standards may not necessarily be undernourished. Certainly at Minjilang there was no evidence to suggest that 'underweight' subjects had consistently compromised vitamin status in comparison to subjects of 'normal' Caucasian BMI (sections 6.8, 6.10). Body composition studies have also shown that for a given BMI, Aboriginal women have more body fat than Caucasian women (Rutishauser and McKay, 1986; O'Dea, 1987).

In most studies in comparable communities, older Aboriginal women have been found to be more obese than older men (section 1.6.5.2). While this tendency was observed at Minjilang (the BMI for older women was $24.4 \pm 1.1 \text{ kg/m}^2$ and for older men it was $23.3 \pm 1.2 \text{ kg/m}^2$), young men had a much higher BMI ($24.2 \pm 1.1 \text{ kg/m}^2$) than young women ($20.9 \pm 1.4 \text{ kg/m}^2$). When the entire community was considered, the BMI of women tended to be lower than men.

However gender differences were small when the ratio of waist to hip circumference was considered; this has been a frequent finding in other studies (section 1.6.7). Also in parallel to other studies (O'Dea, 1987; Zimmet *et al*, 1986), android distribution of body fat at Minjilang (indicated by a waist circumference:hip circumference ratio of ≥ 1.0 for men, or ≥ 0.8 for women) was shown to be associated with hypertriglyceridaemia and impaired glucose tolerance (section 6.5.2, 6.5.3). The android body fat distribution of Aboriginals may help to explain the high prevalence of diabetes and risk factors for ischaemic heart disease observed in both sexes (Bastian, 1979; O'Dea, 1987; Wise *et al*, 1970; Stanton *et al*, 1985; Phillips and Kubisch, 1985; O'Dea *et al*, 1990).

The crude adult diabetes prevalence rate of 10.3% at Minjilang in June 1989 was within the range described for comparable communities (8-24%) (Winterbottom, 1961; Wise *et al*, 1970, 1976; Finlay-Jones and McCormish, 1972; Bastian, 1979; Duffy *et al*, 1981; O'Dea *et al*, 1982, 1988a, 1990; Gault, 1990:25) and higher than the rate (3.4%) for non-Aboriginal Australians (Glattaar *et al*, 1985) (section 1.6.6.2). In most surveys previously undiagnosed diabetics have been identified at a similar rate to that of known diabetics; the rate of identification of new cases of diabetes at Minjilang (two new cases compared with five known cases) was less than expected. This difference was likely to have arisen due to chance (particularly considering the small size of the population); however the apparent increased health consciousness of the community and the interest in non-communicable disease of the Senior Aboriginal Health Worker and past District Medical Officer may also have contributed to the relatively high proportion of 'known' diabetics in the community.

In the above studies the proportion of the total adult population with impaired glucose tolerance varied from 4.8% to 25%; at Minjilang this percentage was 8.7%. As consistently observed in previous studies, at Minjilang a greater number of diabetics and those with impaired glucose tolerance were women (section 6.5.3). The hyperinsulinaemia and elevated triglyceride concentrations described amongst those with impaired glucose tolerance (section 6.5.3) was consistent with underlying resistance to the glucose lowering effects of insulin (section 1.6.6.2) and paralleled findings of previous studies (Bastian, 1979; O'Dea *et al*, 1982, 1988a, 1988b, 1990).

Both mean systolic and diastolic blood pressures measured at Minjilang (section 6.5.6) tended to be lower than at other comparable communities (Wise *et al*, 1970; Edwards *et al*, 1976; Neilson and Williams, 1978; Bastian, 1979; Duffy *et al*, 1981; Simons *et al*, 1981; Thomson, 1984a; Phillips and Kubisch, 1985; Gault, 1990:31, McGrath *et al*, 1991:43-44), but were higher than described in nomadic groups (section 1.6.8). However the Northern Territory communities (Phillips and Kubisch, 1985; Gault, 1990:32-33) which had a higher prevalence of hypertension than Minjilang, also tended to have a greater proportion of subjects classified as 'overweight' by Caucasian standards. As with other studies, both systolic and diastolic blood pressures were found to be positively associated with BMI at Minjilang (section 6.6) and young women tended to have the lowest blood pressures of all age/sex groups (Addendum A2.5).

8.2.3. Changes in anthropometric and metabolic indicators of non-communicable disease over the intervention period: risk factors for cardiovascular disease

Analysis of variance indicated marked linear improvements in several risk factors for cardiovascular disease over the intervention period. These improvements were also supported by the results of paired t-tests in those 44 individuals of the original sample re-monitored in June 1990. Although an important risk factor of cardiovascular disease, the problem of diabetes and impaired glucose tolerance is discussed in detail in section 8.2.4.

8.2.3.1. Lipid profile

Of all risk factors for cardiovascular disease, the most dramatic improvement at Minjilang was observed in serum cholesterol concentration. Throughout the intervention year, mean serum cholesterol of the community decreased by over 12% (from 5.93 ± 0.19 mmol/l to 5.20 ± 0.15 mmol/l) (section 6.5.5). Effective large-scale dietary studies have achieved a

reduction of 10% to 15% of serum cholesterol concentration and reductions in serum cholesterol level have been shown previously to be proportional to dietary adherence (USDHHS, 1988:115). Several clinical trials have demonstrated that lowering plasma cholesterol reduces morbidity and mortality due to coronary heart disease (Goldman and Cook, 1984; USDHHS, 1988:120). The combined results of 11 randomised control dietary-intervention trials found that a 10% reduction in total cholesterol level was associated with a 15% reduction in ischaemic heart disease (IHD) incidence (LRCP, 1984). Based on these projections, the improvement in serum cholesterol concentration at Minjilang should correspond to a 20% reduction in the predicted risk of heart disease.

The finding that the reduction in mean serum cholesterol at Minjilang was related serially to the decreased intake of saturated fat, the increased ratio of poly-unsaturated fatty acids to saturated fatty acids and the increased density of intake of dietary fibre (section 7.1.3.1) was consistent with the relevant literature (section 1.7.4). Conversely the dietary intake of cholesterol *per se*, which actually increased due to the increased consumption of cholesterol rich hen's eggs over the intervention period (section 5.3.12), was not found to be associated with the change in mean serum cholesterol concentration. However, it is now believed that the type of dietary fat tends to have a larger and more consistent effect on serum cholesterol level than dietary cholesterol *per se* (Ernst and Levy, 1980; Diel and Mannerberg, 1981; Schonfeld *et al*, 1982; McNamara *et al*, 1987; Willett, 1990:345). Therefore this finding was consistent with more recent data.

The marked reduction in the mean serum cholesterol concentration that was apparent during the first three month period of the intervention intimated the rapid change in dietary habits which Minjilang residents were able to embrace.

In contrast to most traditional animal foods (section 1.2.2.3), turtle fat was found to be particularly high in saturated fatty acids (C12:0 and C14:0) (section 5.4.2, Addendum 2, Table A2.4). This suggested that those people who consumed the bulk of the turtles captured in November 1989 may have had an increased intake of saturated fatty acids at that time. Analysis of variance revealed that, in December 1989, there was a statistical difference ($F_{1,49}=6.94$, $p < 0.05$) between serum cholesterol concentrations of these 18 subjects (6.38 ± 1.04 mmol/l) and those 33 who did not regularly consume turtle (5.51 ± 1.16 mmol/l). Therefore dietary intake of turtle in some sections of the Minjilang community may have contributed to the relative increase in both the mean and standard deviation of total serum cholesterol concentration measured in December 1989.

The mean fasting serum triglyceride concentration of the community tended to decrease throughout the intervention period although the linear trend was not statistically significant. However the serum triglyceride level did remain relatively low compared with other community studies (section 8.2.2).

8.2.3.2. BMI and the ratio of waist to hip circumference

After controlling for inter-individual variance a small but significant reduction in mean BMI was observed throughout the intervention period (section 6.5.1). There was also a significant trend for those individuals (particularly older women) initially 'overweight' according to Caucasian standards to lose weight, and those initially 'underweight' (particularly younger women) by Caucasian standards to gain weight. As Caucasian standards of 'underweight' may not be appropriate for Aboriginal groups (section 8.2.2), the increasing BMI of 'underweight' subjects was not necessarily desirable.

8.2.3.3. Blood pressure

Both systolic and diastolic values decreased over the intervention period (section 6.5.6). One explanation for the decrease was that the subjects became more familiar with being measured over the course of the study and were less anxious in successive surveys. Under these circumstances it would be expected that the variance would also fall, particularly for systolic blood pressure. However such a reduction was not observed (Table 6.7). The decrease in blood pressure was also surprisingly less systematic in those attending every survey (Table 6.9). This suggested that there was a difference in response between individuals which may *not* have arisen out of familiarity with the measurement procedure.

A fall in blood pressure had been observed previously in association with weight loss and improved lipid and carbohydrate metabolism, when a small group of westernised Aboriginal adults temporarily reverted to traditional lifestyle (O'Dea, 1984, 1986a).

8.2.4. Changes in anthropometric and metabolic indicators of non-communicable disease over the intervention period: diabetes and impaired glucose tolerance

Those older women who lost weight did tend to have improved glucose tolerance over the

year (section 6.5.3). However glucose tolerance did not significantly alter for other subjects or for the community as a whole throughout the year and the fact that fasting insulin and glucose concentrations did not fall in any group suggested that insulin sensitivity did not improve. Increased insulin sensitivity is probably the key to improved metabolic control in NIDDM (section 1.6.6.2).

One important study has indicated that the main metabolic abnormalities of diabetes mellitus can be greatly improved and also potentially prevented (O'Dea, 1984). Metabolic changes included a decrease in fasting glucose, improved post-prandial glucose clearance, improved insulin response and reduction of fasting plasma triglyceride level. Although these improvements were achieved by reversion to a traditional hunter-gatherer lifestyle for seven weeks, this situation may not be desirable, possible or sustainable for most contemporary Aboriginal groups. O'Dea has emphasised the implications of these findings to public health. She stressed the importance of increased physical activity, low energy dense (low fat, high fibre) diet and weight control in the management and prevention of diabetes in Aboriginal people.

The fact that there was no systematic improvement in insulin sensitivity during the Minjilang study (despite increased exercise in some sections of the community (section 8.3.3), a reduction in the energy density of the diet (section 8.1.2) and a small but statistically significant weight loss (section 8.2.3.2)), suggested that the important health problem of impaired glucose tolerance and diabetes may need to be approached by different or more specific strategies; for example focus on groups with impaired glucose tolerance and those with a family history of diabetes, and to aim specifically for greater weight loss.

8.2.5. Change in other metabolic parameters over the intervention period

The very strong relationship between individual serum γ gt concentration and kava consumption (section 6.6.8) supported the results of previous studies (section 1.5.4), which had also demonstrated a dose relationship between the two variables. Although there was no difference between the proportion of subjects who participated in all surveys and the wider population with respect to kava consumption (section 6.1), the fact that there was a greater reduction in γ gt concentration in those participating in all surveys (section 6.3), suggested that they were less likely to consume kava in large quantities during the nutrition intervention project. Although alcohol consumption was also found to

be positively associated with serum γ gt concentration, the effect was less than that observed for kava (section 6.6.8). As had been suggested by the relatively low fasting serum triglyceride concentrations (section 8.2.2), alcohol consumption at Minjilang may have been less frequent than at other comparable communities.

8.2.6. Initial vitamin status and change over the intervention period

8.2.6.1. Folate

There was a very marked increase in the mean red blood cell folate concentration of the community over the intervention period, from $81.6 \pm 3.0 \mu\text{g/l}$ to $191.4 \pm 9.6 \mu\text{g/l}$; serum folate concentrations also rose over the intervention period from $2.1 \pm 0.2 \mu\text{g/l}$ to $2.8 \pm 0.2 \mu\text{g/l}$ (section 6.7.1).

Initial results of serum folate concentrations were similar to those (1.7 ± 0.2 to $2.5 \pm 0.2 \mu\text{g/l}$) described in comparable Aboriginal communities (Davis *et al*, 1965, 1975; Kamien *et al*, 1974; Nobile, 1974; Watson and Tozer, 1986; Cheek *et al*, 1989; Patel *et al*, unpublished; Lion *et al*, unpublished) (section 1.6.10). Unfortunately no published values for red blood cell folate concentrations were available for comparable communities, but results at Minjilang were much lower than those reported ($302 \pm 23 \mu\text{g/l}$) for a small, well-nourished, traditionally-orientated outstation group in north-east Arnhem land (O'Dea *et al*, 1988b). Serum folate levels also rose significantly from $2.5 \pm 0.2 \text{ ng/ml}$ to $3.0 \pm 0.2 \text{ ng/ml}$ in a small group of Aboriginal diabetics and others from the Kimberley, following seven weeks reversion to a traditional lifestyle (O'Dea *et al*, 1987).

The validity of the folate concentrations measured at Minjilang were supported by the relatively consistent results of the internal quality 'controls' (section 6.2.2) and the close agreement between repeated inter-batch and intra-batch assay (section 6.2.1). As indicated by previous studies (Craig *et al*, 1985), the initial low folate status of Minjilang was confirmed by the significant negative correlation between mean cell volume (MCV) and red blood cell folate (section 6.8.4). The serial relationship between decreasing MCV and increasing folate status (section 6.7.3), also supported the notion that the measured improvement in red blood cell folate concentration was legitimate. Moreover the small, significant improvements measured linearly over time in other haematological variables (mean haematocrit and red cell distribution width) were also subsequently found to reflect the increase in folate status (section 6.7.3). Although most haemoglobin concentrations

were surprisingly acceptable in the face of the initially poor folate status, as in many other contemporary communities haemoglobin concentrations were low compared to nomadic groups (section 1.6.9). It had been suggested that the effect of the low folate status may have been masked by iron deficiency (Craig *et al*, 1985; Herbert, 1987b) or the haemopoietic effect of heavy cigarette smoking (Nolan, 1990); however serum ferritin concentrations were not generally indicative of poor iron status. The increase in mean haemoglobin concentration observed during the year was shown to be strongly correlated with the serial increase in red blood cell folate status (section 6.7.3), suggesting that the initially poor folate status of the community *had* compromised haemoglobin concentrations at that time. However the major increase in haemoglobin concentration was observed during the three final months of the study, which suggested that haemoglobin responded slowly to the improving folate status.

Inter-individual analysis of both red blood cell and serum folate concentrations (section 6.8.1 and 6.8.2) supported previous studies highlighting the strong relationship between cigarette smoking and reduced folate status (Senti and Pilch, 1984). Indeed a consistent dose-related response was described for the relationship between serum folate concentration and the number of cigarettes smoked per day (section 6.8.2). However the strongest inverse association between red blood cell folate and smoking status was seen at the first survey (section 6.8.2), that is, when folate intakes were marginal (section 5.3.3).

Although red blood cell folate and serum folate concentrations were positively correlated over all surveys (section 6.7.1), in the first three month period serum folate concentrations actually reduced in the face of increasing red blood cell folate. This situation could have been related to active uptake of folate in haemopoiesis during recovery of long term folate deficiency (Davis, 1990), when folate was being catabolised at a greater rate than it had been absorbed; low serum folate is diagnostic only of negative folate balance, which precedes, and may not necessarily develop into folate depletion of tissues, indicating that "*at the time the sample was drawn and that probably, for the past two weeks or more, less folate has been absorbed than has been catabolised*" (Herbert, 1987b). Serum and red blood cell folate concentrations would only be expected to be positively correlated following a longer period of static dietary intake of folate; for example, as was observed (section 6.7.1) in June 1989 and December 1990 during the dietary study (section 5.3.3). Serum folate levels reflect the dietary intake immediately prior to blood sampling, whereas red blood cell folate levels tend to reflect dietary intake over a longer time frame (Hoffbrand *et al*, 1966; Bates *et al*, 1982), being dependent on folate status when the

erythroid cells reach maturation (Truswell, 1984a). Therefore, it was not surprising that the measure of mean dietary folate intake over the previous three month period determined using the store-turnover method was more strongly associated with red blood cell folate concentration (section 7.1.7.1) than serum folate concentration (section 7.1.7.2).

Over the intervention period at Minjilang both red blood cell folate and serum folate concentrations were found to be significantly positively correlated with plasma ascorbic acid and β -carotene concentrations (section 6.10). As the major dietary source of all these vitamins is fresh fruit and vegetables (Paul and Southgate, 1978), this observation supports the notion that folate status is a reflection of the dietary intake of these foods and suggests that folate status could be used as a 'marker' of fruit and vegetable intake in Aboriginal communities. In one previous study of 26 Aboriginal mothers (Nobile, 1974), serum folate concentrations were also found to be correlated with plasma vitamin C levels ($r=0.63$ $p<0.001$).

Poor folate status has been shown to be associated with excessive alcohol intake (Bonjour, 1980; Halsted, 1980). However, at Minjilang there was no association between red blood cell folate concentration and alcohol consumption (section 6.8.1). These results again suggested that excessive alcohol intake was not a particular problem at Minjilang. Although there was a positive correlation between serum folate concentration and alcohol consumption, the relationship was much stronger between low serum folate concentration and cigarette smoking (section 6.8.2).

These results appeared to have several clinical implications. Firstly, they confirmed that poor folate status is widespread in remote, centralised Aboriginal communities, which could have implications with respect to compromised immune function (section 1.6.3), low birth weights³ (section 1.6.3) and the high relative rate of neural tube defects⁴ amongst Aboriginal neonates in the Northern Territory (Connors, 1989). Secondly, results suggested that, although dietary factors were implicated predominantly, heavy cigarette smoking was also related to poor folate status. Thirdly, it was clearly demonstrated that

³ Folate supplementation to 'deprived' Indian mothers, has been shown to increase birth weight (Inyegar and Rajalakshmi, 1975). Folate status in non-anaemic pregnant women has also been shown to be correlated with birth weight, suggesting that growth rate of the foetus was affected even before anaemia due to the deficiency was detected in the mother (Whiteside *et al.*, 1968).

⁴ Maternal folate deficiency has been implicated in the development of foetal malformations, particularly neural tube defects (Smithells, 1976, 1983).

folate status could be improved remarkably by dietary means alone, that is, without medication (thereby enabling simultaneous benefits such as reduced serum cholesterol concentration). Finally, it would appear to be warranted to reconsider the widespread prescription (Barr, 1989) of the antibiotic *co-trimoxazol* (brand name: *Bactrim*), a known folate antagonist (Truswell, 1990), in Aboriginal communities. It is hoped that greater attention will be drawn to the widespread, frequently overlooked problem of low folate status in remote Aboriginal communities, particularly now that the problem has been shown to be amenable to rudimentary dietary intervention.

8.2.6.2. Thiamine

Of all results, those of thiamine proved to be the most complex to interpret due to the pronounced changes measured during survey three (December 1989). In particular the results of the red blood cell thiamine assays were much higher at survey three than at other times throughout the study (section 6.9.1). Further investigation suggested that the increase was due to individual change in dietary intake of non-store food sources of thiamine rather than due to laboratory artefact (section 7.1.7.5; section 7.1.7.6). Additional sources of thiamine available to some sections of the Minjilang community in December 1990 included green turtle (*Chelonia mydas*) (Section 5.4.1), a particularly rich source of thiamine at 0.25 mg/100 g (USDHEW, 1972).⁵

Similar unexplained seasonal changes in thiamine status have also been described in an east Arnhem Land community (Riley *et al*, 1990); compared with the 'normal' Caucasian reference range, none of the 156 subjects sampled in May 1988 but 65% of the 46 individuals sampled in November 1988 had low red blood cell thiamine concentrations.

The intake of thiamine as measured by the store-turnover method did significantly explain the individual change in thiamine status over the intervention period once adjusted for survey three. Less of the variance in red blood cell thiamine was explained by this measure of thiamine intake than that of serum thiamine concentration. Several factors can confound the relationship between dietary thiamine intake and thiamine status. For example, decreased thiamine absorption has been associated with low folate status and

⁵ For comparison, the thiamine content of rump steak is approximately 0.08 mg/100 g. The Aboriginal research assistant also consumed a large quantity of turtle meat during this period, which may explain the fact that both her serum and red blood cell thiamine concentrations were relatively high in December 1989, compared both to results of all other assays and those of the non-Aboriginal researcher.

tannic acid, found in tea (a popular beverage in Aboriginal communities) is a known thiamine antagonist (Wood, 1990). Thiamine status may be compromised by excessive alcohol intake, particularly in underweight persons (Wood and Breen, 1980; Wood, 1990); at Minjilang serum thiamine concentrations were found to be relatively low in underweight subjects who drank alcohol when it was available (section 6.10.3), but red blood cell thiamine was not found to be associated with alcohol consumption (section 6.10.4).

Given the high intake of refined carbohydrate described in Aboriginal communities, it was expected that thiamine status may have been adversely affected (Brin, 1976; Wood, 1985, 1990). Although the mean red blood cell thiamine concentration of the Minjilang population was within the reference range for Caucasians, 20% did initially have low red blood cell thiamine concentrations, but the similar proportion of subjects with relatively elevated serum thiamine concentrations was surprising. Again, similarly unaccountable results were also observed in previous studies in other communities; compared to the 'normal' Caucasian reference range, 42% of the 64 adults studied in the north east Arnhem land community in September 1987 had high serum thiamine concentrations (Riley *et al*, 1990), and at Utopia 8% of women and 18% of men had low values and 37% of the women and 8% of the men had high values (Gault, 1990:49). As Utopia is located in central Australia, short term dietary intake of turtle was unlikely to have contributed to the high concentrations observed!⁶.

There was no ready explanation for the lack of correlation observed between serum and red blood cell thiamine concentrations (section 6.9.1); neither was a correlation between serum and red cell thiamine levels observed in the previous study in north east Arnhem land (Riley *et al*, 1990).

Poor thiamine status was described for most age and sex groups in a "*part Aboriginal*" community (Kamien *et al*, 1974), but was particularly marked for women of child bearing age (Kamien *et al*, 1974; Nobile, 1974). Fortification of flour resulted in improvements in the thiamine status of this community (Kamien *et al*, 1975b). Although it was not fortified at the time of the Minjilang study, Australian flour used specifically in bread making is currently fortified with thiamine following the recommendations of the NHMRC Nutrition Committee (Wood, 1990). Given that flour, rather than bread, provides a large proportion

⁶ However, the sex differences at Utopia may suggest that women consumed a greater proportion of thiamine rich bush food prior to the study.

of carbohydrate intake in remote Aboriginal communities, it is unlikely that the fortification of bread with thiamine will have a great impact on the thiamine status of Aboriginal Australians in these communities.

8.2.6.3. Vitamin B₁₂

As in previous studies (Elphinstone, 1971; Davis and Pitney, 1957; Pitney, 1962; Davis *et al*, 1975; Holt *et al*, 1980; Gault, 1990:49) serum concentration of vitamin B₁₂ was consistently high (section 1.6.10) at Minjilang relative to the Caucasian reference range (section 6.9.1). In the past, as most vitamin B₁₂ was found to be 'bound' rather than available in the usual free form, the high levels of the vitamin were attributed to increased levels of vitamin B₁₂ binding protein which may be secondary to both acute and chronic infections (Davis and Pitney, 1957). As liver damage associated with alcoholism has also been associated with high vitamin B₁₂ levels (Jones and Mills, 1955; Kamien *et al*, 1974), high vitamin B₁₂ concentrations in Aboriginal groups have also been ascribed to excessive alcohol intake. However in one small study vitamin B₁₂ concentrations were unrelated to previous alcohol intake (O'Dea *et al*, 1987), and liver damage would not explain the consistently high levels in traditionally-orientated groups with no access to alcohol. However in the current study at Minjilang, nutrient intake of vitamin B₁₂ was shown to be positively correlated with observed fluctuations in the intra-individual changes in serum vitamin B₁₂ concentration (section 7.1.7.4). This suggested that there was a dietary component to this common observation. Traditional meat-orientated diets (section 1.2.2.2) would have provided an extremely rich dietary intake of vitamin B₁₂ (English, 1990). Compared to the dietary intake of other vitamins, relatively high intakes of vitamin B₁₂ have been shown to persist in the contemporary situation where diets remain meat-orientated (section 3.6.2.2). The unusual observation that 14 of 57 "part Aboriginal" people living in Bourke were found to have low or marginal levels of vitamin B₁₂ (Kamien *et al*, 1974) could have reflected the relatively low meat content of the diet in that setting. The hypothesis that high vitamin B₁₂ concentrations are related to the relatively high meat intake of Aboriginal groups was also supported by the significant rise in serum vitamin B₁₂ observed when a small Kimberley group reverted to traditional lifestyle and diet (O'Dea *et al*, 1987).

8.2.6.4. Vitamin B₆

As in previous studies in Aboriginal communities (Kamien *et al*, 1974; Nobile, 1974;

Davis *et al*, 1975) a large proportion of both male and female Minjilang adults initially had low serum concentrations of vitamin B₆ relative to Caucasian reference standards (section 6.9.1). Vitamin B₆ is found in a wide variety of food sources and in developed countries vitamin B₆ deficiency is uncommon, although some at-risk groups such as the elderly and adolescent girls have been shown to be affected (Hunter, 1990).

At Minjilang, serum vitamin B₆ concentration was shown to markedly increase during the intervention period from 41.4 ± 2.0 nmol/l to 65.0 ± 4.8 nmol/l (section 6.9.1). However the increasing variance of serum vitamin B₆ concentration (Table 6.23) suggested that there was a difference in response⁷ between individuals throughout the intervention period. The increase in serum vitamin B₆ concentration was less systematic in those attending every survey (Table 6.24). Serum vitamin B₆ concentrations (as was the case with serum folate, plasma β -carotene and ascorbic acid concentrations) were found to be lower in those who smoked cigarettes (section 6.10.1). However change in cigarette smoking could not explain the difference in individual response over the intervention period (section 7.1.7.3) and differential dietary intake of foods containing vitamin B₆ may have contributed to the difference in individual response. The increase in mean serum vitamin B₆ was positively associated with the increasing dietary intake of vitamin B₆ measured by the store-turnover method (section 7.1.7.3).

8.2.6.5. Ascorbic acid

As ascorbic acid is readily absorbed from the intestine and circulates freely between plasma and tissues (Hunter, 1990), ascorbic acid status is generally reflected by plasma ascorbic acid concentration (Hunter, 1990; Read, 1990). Very low plasma concentrations of ascorbic acid were initially measured at Minjilang; the mean concentration was marginal compared with 'normal' Caucasian reference standards (section 6.9.1). Similar results have been described in other Aboriginal communities (Jose and Welch, 1970; Hodges, 1960; Kamien *et al*, 1974; Nobile, 1974). Ascorbic acid status improved significantly at Minjilang throughout the intervention period and plasma concentrations increased from 4.10 ± 0.34 mg/l to 10.34 ± 2.49 mg/l (section 6.9.1). However the increasing variance of plasma ascorbic acid concentration (Table 6.23) suggested that there

⁷ Differences in response could have occurred in either dietary change (i.e. compliance) or physiological response to dietary change.

was a difference in response⁸ between individuals throughout the intervention project. The increase in plasma ascorbic acid was less systematic in those attending every survey (Table 6.24). Differences in response may have arisen due to differential dietary change in ascorbic acid intake as change in cigarette smoking could not explain the difference in individual response over the intervention period (section 7.1.7.7). Results suggested that the ascorbic acid status of most of the adults of the Minjilang community remained low compared to the 'normal' Caucasian reference range. This occurred despite the fact that the intake of ascorbic acid as measured by the store-turnover method remained higher than the recommended intake throughout the intervention period (section 5.3.6) and a strong relationship between the increased plasma ascorbic acid concentration and dietary intake of vitamin C over the intervention period was described (section 7.1.7.7). As reported in other studies (Hunter, 1990), plasma ascorbic acid concentrations were found to be much reduced in those who smoked cigarettes (section 6.10.5); as most subjects smoked heavily this could help explain the very low levels of plasma ascorbic acid revealed. Vitamin C status could also have been compromised by the high prevalence of chronic and acute infections (Hunter, 1990) frequently described in similar Aboriginal communities.

Biochemical status of vitamin C has been shown to closely reflect recent, rather than long-term, dietary intake (Woodhill and Nobile, 1971; Hunter, 1990; Zador *et al*, 1990). At Minjilang, plasma ascorbic acid concentration was shown to increase in positive correlation with dietary intake of ascorbic acid during the previous three month period as measured by the store-turnover method (section 7.1.7.7). However, a stronger association was observed between red blood cell folate concentration and dietary intake of folate during the previous three month period as measured by the store-turnover method (section 7.1.7.1). This would be expected as red blood cell folate concentration is known to reflect dietary intake of the relevant vitamin over a longer term than plasma ascorbic acid concentration (section 8.2.6.1).

Despite dramatic fluctuations in the availability of the vegetable component of the traditional diet, little evidence of scurvy has been recorded for traditional Aboriginal groups (Hodges, 1960; Billington, 1960; McArthur, 1960b). Liver has been shown to be a good dietary source of vitamin C and may have been a useful source of ascorbic acid in traditional times (O'Dea *et al*, 1987); it may also be a useful source of vitamin C in communities which do not have ready access to fruit and vegetable supplies.

⁸ Differences in response could have occurred in either compliance or physiological response to dietary change.

8.2.6.6. Vitamin A

Recent interest in vitamin A status of Aboriginal groups has been raised by postulated associations between vitamin A consumption and childhood survival in third world countries (Sommer *et al*, 1883, 1986; Tarwotjo *et al*, 1987; Gadomski and Kjolhede, 1988); it has been speculated (Rutishauser, 1990a) that Aboriginals are the only Australian group who appear to have a consistently low intake of vitamin A. Vitamin A is stored in the liver and plasma levels do not accurately reflect true retinol status over the normal range of liver vitamin A concentration. However plasma retinol may be a more sensitive indicator of vitamin A status in populations with a prolonged low intake of vitamin A (Hunter, 1990). Therefore plasma retinol would be expected to be generally low if Aboriginal groups had a consistently low intake of vitamin A. However, as with all other available studies in Aboriginal communities (Kamien *et al*, 1974; Nobile, 1974; O'Dea *et al*, 1987; Cheek *et al*, 1989), there was no evidence of low vitamin A status at Minjilang. Indeed, most adults had consistently high concentrations of plasma retinol relative to the 'normal' Caucasian reference range throughout the entire intervention period (section 6.9.2). Plasma retinol concentration (and plasma α -tocopherol concentration) was also found to be relatively high in those who drank kava (section 6.10.6); there was no ready explanation for this observation.

As plasma levels do not accurately reflect retinol status over the normal range of liver vitamin A concentration (Hunter, 1990), it was not surprising that there was no significant increase in plasma retinol concentration over the intervention period despite the measured increase in the density of dietary intake of retinol, vitamin A equivalents and β -carotene over the intervention period at Minjilang.

In a previous study (O'Dea *et al*, 1987), serum retinol fell significantly following reversion to traditional lifestyle for a small group of diabetics ($465 \pm 36 \mu\text{g/l}$ to $404 \pm 19 \mu\text{g/l}$); however both measurements were still acceptable compared with the 'normal' adult Caucasian reference range.

In contrast to the acceptable plasma retinol concentrations described for Aboriginal groups, initial plasma β -carotene concentrations at Minjilang were low compared with the 'normal' Caucasian reference range (section 6.9.2). This has been a frequent finding in comparable studies (Kamien *et al*, 1974; Nobile, 1974; O'Dea *et al*, 1987; Cheek *et al*, 1989), and probably reflects the low intake of carotenoid-containing fruit and vegetables.

Plasma carotenoid concentrations are considered to be a sensitive measure of recent dietary carotenoid intake as they are not closely regulated by homeostatic mechanisms (Hunter, 1990). Large inter-individual variations and high intra-individual repeatability in serum β -carotene levels have been documented (Willett *et al*, 1983). Mean plasma β -carotene concentrations did improve during the intervention project and rose from 5.7 ± 0.5 mg/100 ml to 7.6 ± 1.1 mg/100 ml (section 6.9.2). However, the increasing variance of plasma β -carotene concentration (Table 6.23) suggested that there was a difference in response⁹ between individuals throughout the intervention project. The increase in plasma β -carotene concentration was less systematic in those attending every survey (Table 6.24). As with other studies (Hunter, 1990), a strong negative relationship between plasma β -carotene concentration and cigarette smoking was described (section 6.10.8), but changes in cigarette smoking could not explain the difference in individual response over the intervention period (section 7.1.7.8). It is again likely that the different individual response may have arisen from differential dietary change; some subjects may have eaten more carotenoid containing fruit and vegetables than others. There was also evidence that mean plasma α -carotene concentrations increased during the final six months of the study (section 6.9.2).

In confirmation of the known relationship between the two (Hunter, 1990), the improvement in plasma β -carotene concentration at Minjilang was positively associated with the increasing intake of dietary β -carotene intake as measured by the store-turnover method (section 7.1.7.8).

8.2.6.7. α -tocopherol

In line with the high intake of dietary fat and high serum cholesterol concentrations described, the plasma concentrations of α -tocopherol were higher at Minjilang compared with studies in other Aboriginal groups (Kamien *et al*, 1974; Nobile, 1974; O'Dea *et al*, 1987).

Unlike vitamin A, there is no principle storage organ for vitamin E and plasma levels of α -tocopherol have been shown to be associated with α -tocopherol intake (Hunter, 1990). However, as α -tocopherol is transported in blood within the low density lipoprotein

⁹ Differences in response could have occurred in either dietary change (ie. compliance) or physiological response to dietary change.

faction, plasma tocopherol levels tend to be highly correlated with the total plasma lipid concentration (Hunter, 1990; Roberts, 1990) and adjustment for total plasma cholesterol concentration has been shown to increase the correlation between dietary intake and plasma levels of α -tocopherol (Willet *et al*, 1983). At Minjilang there was a relative increase in the ratio of α -tocopherol to total serum cholesterol (section 6.9.2). Change in the density of dietary intake of vitamin E was shown to be positively associated with plasma α -tocopherol concentration after controlling for change in total serum cholesterol concentration (section 7.1.7.9).

8.3. Intervention strategies

Both the initial store turnover and biological screening results were used to design intervention strategies with the community. Strategies were modified and developed according to successive results of the surveys conducted at three monthly intervals. In order to maximise the potential for change several strategies were applied simultaneously. These addressed two major issues: increasing motivation of community members and promoting an increased variety of food choice.

8.3.1. Increasing motivation of community members

There appeared to be a high degree of knowledge and awareness of 'western' nutrition information at Minjilang (particularly amongst older women), prior to the commencement of the formal intervention project. This was supported by evidence of dietary differences between Minjilang and the control community prior to June 1989. In particular, longitudinal store turnover data indicated that the intake of sugar had already decreased at Minjilang over the period from March 1986 to June 1989 (section 8.1.1). Therefore it appeared that, rather than needing more information about food and nutrition, Minjilang residents were already aware of major 'western' nutrition concepts and were at the stage where they required practical support in order to act on this knowledge.

8.3.1.1. Initiation of the project by the community

The project was initiated in response to a direct request from the senior Aboriginal Health Worker (Mrs Daisy Yarmirr) on behalf of those adults living at the community who wished to be screened for risk of coronary heart disease. The project developed entirely from within the community itself rather than being applied from external agencies.

The initial request reflected the increased community awareness of non-communicable disease, and appeared to arise due to three main reasons:

- 1: Concern, particularly amongst the older women, about the sudden fatal myocardial infarctions of two young men who were related to the major land owning group at Croker Island
- 2: The previous District Medical Officer (Dr Peter Thorn) had a special interest in non-communicable disease and had assessed the lipid profiles of several prominent community members, where clinically indicated, over the past four years; follow-up counselling had been provided by a nutritionist (myself)
- 3: The three Aboriginal Health Workers at Minjilang had extensive clinical skills and experience, and a comprehensive understanding of the (non-Aboriginal) principles of nutrition; they expressed an interest in the practice of preventative, rather than curative medicine

In March 1989 I was invited to Minjilang to discuss the request for community screening with the twelve members of the community council. At the meeting the Senior Aboriginal Health Worker and an influential elder of the community (Mrs Ilitjilli Lamilami) requested that the prospective cholesterol screening be extended, for voluntary participants, to include other biochemical, anthropometric and haematological measurements of health and nutritional status, so that people had the opportunity to be "*checked right through*". The council formally agreed to adopt this approach. It was agreed that, in the event of any abnormalities being uncovered as a result of the screening, I would "*sit down*" in the community to work with both the relevant family groups and the store for one year. The council also requested that tests should be repeated so that people could "*keep a look out*" on their progress. Accommodation for the researcher and the potential employment of a community member to help with the project were also raised by the council. Plans were made to commence screening during the first week of June 1989.

This meeting marked the only formal gathering to discuss the project; subsequent developments, such as the three monthly monitoring of biochemical, anthropometric and haematological measurements, evolved only after the more traditionally-orientated and protracted process of informal family group discussion and consensus.

8.3.1.2. Direction of the project by the community elders

In traditional Aboriginal society knowledge and authority rests with tribal elders. This was very apparent at Minjilang where the senior members of the land-owning groups were responsible for all major administrative decisions. Although Minjilang was not managed as an official 'community-controlled' Aboriginal health service, the delivery of health care was directed by the senior Aboriginal Health Worker who was immediately answerable to the broader community through kinship responsibilities. The relatively infrequent medical visits and the absence of non-Aboriginal health staff based at Croker Island ensured that the day to day control of health care delivery remained within the community. The more influential members of the Minjilang community council said that they did not want non-Aboriginal health staff to reside at Croker Island (as may be the case with the formal development of a 'community-controlled' health service) and felt that they had adequate control over the current health system by virtue of the unique standing of the Senior Aboriginal Health Worker and their control over the '*Grants-in-aid*' funding scheme of the NTDHCS (section 4.2.1). The strong, cohesive nature of the Minjilang community and the independent nature of the Menzies School of Health Research also ensured that management of the nutrition intervention project was vested within traditional power structures. Researchers looked directly to the community elders for direction ensuring that 'ownership' of the project remained within the community.

8.3.1.3. The development of relationships with community members and all family groups

Given that traditionally-orientated Aboriginal people may view food in terms of "*social metabolism*" (Stacy, 1977) rather than biological metabolism (section 1.2.3.3; section 1.9.1.1), it was relevant to consider the nature of the relationship between community members and researchers. This relationship was assisted by the fact that both myself and the Aboriginal research assistant had been known to community members at Minjilang for several years prior to the commencement of the nutrition intervention project. The Aboriginal research assistant was also related by marriage to one family group at Minjilang. However it was necessary for the Aboriginal research assistant to actively foster relationships with other extended family groups to avoid accusations of 'favouritism'. From a traditional perspective there were also potential problems as the project involved working very closely with participants of both sexes (section 1.9.2). Several precautions were taken to avoid the development of misunderstandings. The two

researchers resided within the community for extended periods during the intervention year, enabling the development of close personal and professional relationships with many residents. In turn these strong relationships advanced the development of mutual trust and the sharing of information within an atmosphere of active co-operation.

8.3.1.4. The identification of both traditional and contemporary beliefs and attitudes related to health and nutrition within the community

Strategies were designed to reinforce and build on the existing food and health knowledge of community members. At all times the cultural and nutritional value of traditional foods was supported. People were encouraged to choose foods from the store which were most like bush foods, such as fresh fruit and vegetables and lean meat. In discussion, foods were not classified into the groupings used in past nutrition education programs (Shelley *et al*, 1981; Shelley, 1982) (section 1.9.1.1). Instead foods were classified according to traditional categories (section 1.2.3.2), when the community deemed such division expedient. This approach dealt directly with those food messages derived from store turnover data, which at first impression appeared to directly contradict traditional food values and preferences. For example, the adage '*choose lean meat, discarding visible fat*' would appear to oppose the traditional value afforded to fat (section 1.2.3.1). However, people readily distinguished between the type of visible fat found on traditional foods (which they referred to as "*soft fat*") and the type of fat on store purchased meat ("*hard fat*"). Tribal elders, in particular the wife of the author Lamilami (1974), also drew on relevant traditional stories highlighting over-consumption associated with greed (Addendum 4); these stories were interpreted by the community as warnings about the over-consumption of fat and sugar. Therefore 'teaching methods', in both cultural dimensions, followed the traditional Aboriginal style, being contextual, informal, holistic and practical (section 1.2.1).

8.3.1.5. The employment of an Aboriginal research assistant/nutrition worker

In line with the many potential benefits related to the employment of indigenous people in health care projects (WHO, 1986b:1-2) an Aboriginal research assistant was employed by MSHR specifically to work on the nutrition intervention project. The nutrition worker (Ms Annie Bonson) is an extremely popular personality and well-known within 'Top End' communities. She is committed to the concept of health through nutrition having personally experienced the benefits of improved diet and functioned (unintentionally) as a

vivacious role model for the young women in the community. More importantly, she was accepted by the community as a non-threatening person who immediately understood and accepted local cultural values and was sensitive to direction by the community in a way which would have been extraordinary in a non-Aboriginal person. She was also able to convey greater insight into community perspectives to myself and translate non-Aboriginal concepts to the community in a meaningful way.

8.3.1.6. Employment of community members during the screening and monitoring process

Although no community resident wished to be formally employed to work full-time on the project, at the request of the community seven different individuals were employed by MSHR for short periods during the phases of biological survey. Employees were chosen by the community from different family groups. Only one had previous formal training as an Aboriginal health worker. Duties included: driving the clinic vehicle to collect and return volunteers for survey; measuring blood pressure and pulse using automated equipment; preparing glucose loads for glucose tolerance tests; separating blood samples; centrifuging blood samples; labelling sample tubes; taking prepared samples to the airport; preparing breakfasts for subjects following survey. There were obvious benefits in involving local people in this manner. Employees learnt new skills, acquired increased status within the community and valued being paid for these duties. Volunteers also appeared more relaxed and willing to approach and remain in the clinic when staffed by kinfolk rather than non-Aboriginal health professionals. The survey process also became a genuine community activity with opportunities for the exchange of information. For example, many subjects expressed the desire to observe, and sometimes assist with, the separation and handling of their own blood samples; this provided the opportunity to show and discuss the implications of lipemic samples to subjects and their families. Apart from the two researchers from MSHR, no external health professionals were involved in the survey in March 1990; previous employment of community members lead directly to this important step as local Aboriginal Health Workers and residents had developed confidence in their ability to work together in a clinical setting. The Senior Aboriginal Health Worker was very proud and community self-esteem was particularly high at that time. Even though there was inadequate supervision resulting in insufficient mixing of several haematological samples (section 6.7), the benefits in the process of the conduct of that survey appeared to overwhelmingly outweigh the negative elements.

8.3.1.7. Prompt return in an appropriate form, of the individual results of all biochemical, anthropometric and haematological monitoring

The value of prompt return of results to individual volunteers cannot be over-emphasised, particularly in the face of the record of past research in Aboriginal communities (section 1.9.4). Subjects presented at each survey because they wanted to discover how they were progressing during the intervention project; they wanted their own results as soon as possible and would not have re-presented at subsequent surveys if their needs were not met. Every effort was made to successfully ensure that feedback booklets (Addendum 3, A3.1) were distributed to subjects within two weeks of each survey. The booklets provided a physical representation of individual results. They appeared to be highly valued and were carefully stored in family residences. However, the most effective component of feedback appeared to be the verbal explanation of the meaning of the results in a family group setting. Various metaphors were used to assist in the explanation of concepts and results. For example, the analogy of a pipe coated with mineral deposits (with which people were very familiar) was used to illustrate the idea of atherosclerosis and elevated serum lipid concentrations. Individuals were keen to compare their own results with past measurements. Within family groups there was also a tendency for people to compete against each other.

8.3.1.8. Prompt display and discussion of community results following each period of dietary and biological survey

In addition to individual results, the results of each survey were presented on a community basis (Addendum 3, A3.1). The results of each store turnover were also presented in the form of a community booklet (Addendum 3, A3.1). In addition to being distributed within the community these booklets were mounted on prominent walls at the store, the clinic and the council. Aboriginal Health Workers and community elders appeared to be most interested in the community results.

8.3.1.9. Involvement of children through practical nutrition sessions and activities at school

There was no formal attempt to target children in the intervention project. However we were asked by community elders to talk formally to the school children about the community surveys, results and the nutrition project in general. The school teachers were

very obliging. Practical food preparation had been a component of school-based activities for the previous three years. These activities continued but were extended to include educational visits to the store, food tastings, food games and poster-making for display in public areas. Several senior women also commenced teaching an Iwadja studies program at the school which incorporated traditional foods. In May 1990 both parents and school children requested that the children be included in the community survey. Measurements of anthropometric parameters and haemoglobin concentration were subsequently made, compared with previous clinical data and reported elsewhere (Lee *et al*, in preparation).

Given that a large proportion of the Minjilang population were children (section 4.2.1), the marked improvement in store turnover data over the intervention period suggested that the diet of children residing in the community may also have changed. Certainly children were observed eating the increased variety of food available at the store; sandwiches and fruit appeared to be particularly popular. These observations suggested that children were looking to their parents for guidance in food choice, as would have been the case in traditional times. Several older women actually guided their grandchildren through the store, pointing out the foods which had been labelled with '*good heart food*' and '*strong blood food*' shelf-talkers (section 8.3.2.3).

8.3.1.10. The use of store turnover data to devise simple dietary messages

Store turnover data was used as a rational basis in the development of specific nutrition messages targeting key foods items at Minjilang. These messages were used as a guide to help people improve their diet, and were incorporated into the above strategies in an attempt to render them more culturally appropriate. Specific messages were:

- Increase consumption of fruit and vegetables
- Increase consumption of wholemeal bread and wholegrain cereals like porridge
- Reduce consumption of refined sugar *per se*
- Reduce consumption of sweetened carbonated beverages ("*sweet cool drinks*")
- Choose lean meat, discarding visible fat
- Eat sandwiches instead of pies

8.3.2. Promoting an increased variety of food choice

The store provided a natural focus for increasing the variety of food available to the Minjilang community. In contrast to the endeavours of several past nutrition programs (section 1.9.1) there was no attempt to manipulate the pricing structure of foods available in the store. This was an agreed approach between community representatives of the ALPA executive, the initial store manager, interested community members (including Aboriginal Health Workers) and the researchers. It was based on the premise that more existential principles governed the use of money by traditionally-orientated Aboriginal people than those which affect European concepts of budgeting (section 1.5.3.4; 1.9.1.2) and evidence that 'price elasticity' in Aboriginal community stores differs greatly from the non-Aboriginal setting (McMillan, 1991). It was felt that if there was sufficient cash in hand for customers to purchase a desired item, then that item would be bought regardless of either its price or the amount of money remaining; if there was not sufficient cash it could be 'borrowed' from kinfolk, acquired by gambling (section 1.5.3.4) or eventually obtained from the next salary payment or social security entitlement. Therefore the subsidisation of, for example, fruit by increasing the price of '*Coca Cola*' would not necessarily ensure an increased turnover of fruit; rather '*Coca Cola*' could be preferentially purchased leaving less change for nutritionally-preferred foods. Therefore, rather than follow potentially ill-founded and patronising changes to the store pricing policy (determined centrally by ALPA), it was agreed that, together with efforts to increase community motivation for dietary change (section 8.3.1), provision of a wide range of good quality, nutritious foods was the most appropriate approach. However, it was clearly not sufficient to merely stock new foods on the shelves beside familiar products; nutritionally-preferred foods required identification, introduction through tastings and general promotion.

8.3.2.1. The provision of a wide variety of foods in the store

A wide variety of food choice was maintained in the Minjilang store. In particular, items such as sweetened carbonated beverages and high fat 'take-away' foods (such as pies) were constantly available; poly-unsaturated and eventually mono-unsaturated vegetable oils were displayed alongside previously stocked brands. Therefore improvements in dietary intake actually occurred as a result of *choice* of suitable foods, rather than as a result of limited options.

Some store managers appeared to assume that Aboriginal consumers were less discriminating than non-Aboriginal consumers. However, poor quality food items, particularly fruit and vegetables, were not acceptable to people who, traditionally, enjoyed an extensive supply of very fresh foods (section 1.2.3.1). Spoiled foods tended to remain unpurchased and fuelled comments such as "*Aboriginal people do not like fruit*", which were used to justify lack of stock at some periods throughout the intervention year, in particular from November 1989 to January 1990 (section 8.1.2). The views of the store manager during this period also embraced such beliefs as "*Aboriginal people do not like wholemeal bread*" and "*Aboriginal people prefer 'Coca Cola' to diet drinks*". However, as has been seen, the community council was able to independently supply those foods which were temporarily unavailable at the Minjilang store. When the store manager was forced to bow to economic pressure, the people of Minjilang realised their consumer powers for the first time. All subsequent community requests for specific foods were met. In the past it was unlikely that the community would have confronted the store manager, but would have waited passively for him to leave for another settlement and hoped to have secured the services of a more sympathetic employee.

8.3.2.2. Visual displays

Senior women produced a prominent display at the entrance to the store which illustrated the amount of fat and sugar present in different foods; lard and white granulated sugar were used to represent those dietary components. The relative quantities of each were weighed, placed in transparent plastic containers and exhibited beside the appropriate food. The display proved particularly useful in highlighting the sources of 'hidden' fats and sugars in foods such as potato crisps, cakes and biscuits. The Aboriginal store workers maintained the display.

A colourful display of a wide variety of fruit and vegetables was also sustained within the store. Aboriginal store workers also ensured that target foods, such as wholemeal bread and diet cool drinks, were constantly stocked on the shelves beside standard lines.

The actual lay-out of foods in the store was also likely to affect purchasing patterns. Although store managers were not always amenable to re-organisation of the store shelves (section 8.1.2), attempts were made to display target foods at eye level and at the entrance to cash register check-outs. Vertical glass-fronted refrigerated cabinets for additional display of nutritious perishable foods were ordered during the year but did not arrive until

completion of the project.

8.3.2.3. The use of 'shelf-talkers'

Red, heart-shaped cardboard signs were cut from cardboard, labelled '*good heart food*' and '*strong blood food*' and adhered to the shelf beneath the appropriate 'target' foods in the store. These 'shelf-talkers' aided community recognition of nutritious foods and were of particular benefit to illiterate and visually impaired shoppers.

The fact that the original cardboard signs survived in excellent condition throughout the entire year, illustrated the value afforded to them by the community. This was particularly revealing as several 'shelf-talkers' were located within the grasp of young children; on many occasions store workers and researchers overheard adults direct children not to touch the 'special' signs.

8.3.2.4. The preparation and tasting of a wide variety of 'target' foods and suitable recipes in the store

Of those prepared foods introduced to the community during the year, wholemeal salad sandwiches proved to be the most popular. Initially sandwiches were prepared by the researchers and made available at no cost to the community as a promotional exercise. Subsequently Aboriginal store workers made the sandwiches each morning. The store manager also calculated that there was greater profit to be earned from the preparation and sale of sandwiches than from the purchase and sale of high fat 'take-away' foods, such as meat pies. Occasionally other 'one-pot' dishes such as vegetable stew and rice were prepared by store-workers and sold in the store. Recipes using target foods and written in accessible English were also available at the store.

8.3.2.5. Maintenance of good relationships with store employees

As has been seen (section 8.1.1; 8.3.2.1) store managers appeared to be particularly powerful determinants of food supply in remote, centralised Aboriginal communities. Therefore it was extremely important that there was a good relationship between individual store managers, community consumers and community-based health professionals and, in this study, between store managers and nutrition researchers. Such relationships have been hindered by negative stereo-typing of both store managers and

nutritionists. In addition, the valuable resource of Aboriginal store workers would not appear to have been afforded professional recognition in the past.

During the year of the intervention the profits of the Minjilang community store increased (McMillan, 1990), practically illustrating that the provision of nutritious food was compatible with lucrative business practices. This point clearly illustrated the benefits to be obtained by mutual co-operation.

Researchers spent many hours in direct consultation with the four different store managers employed during the year. The Aboriginal research assistant voluntarily worked in the store, pricing goods, stocking shelves and making displays. Excellent personal relationships were established with the two Aboriginal store workers who had been employed for several years. Both had acquired intimate knowledge of the workings of the store (and the desires of community consumers), and had many innovative ideas about ways to display and promote nutritious foods.

8.3.3. Other strategies

Various community development projects were also supported during the intervention year.

During the year subjects were constantly encouraged to increase exercise. For most older people walking was the preferred option. Rather than use the council vehicle, people were encouraged to walk to the store by the initiation of a grocery delivery service. Younger men and women commenced evening basketball games; the school basketball court was upgraded by the NT Education Department following a submission by the community. A successful council submission to the NT Department of Youth Sport and Recreation also ensured funding for the development of a new football oval during the year. Both submissions were supported in writing by the researchers who outlined the early positive results of the nutrition intervention project and the community endeavours to improve health.

Fruit and vegetable gardens were established surrounding four houses during the year, although, with the exception of two watermelons, no produce had matured by the conclusion of the formal nutrition intervention project. The gardens were fenced against assault by children, dogs and the pet community pigs. Varieties planted included bananas,

papaya, sugar cane and cassava. Gardening skills had been acquired during the mission years. Researchers assisted by providing seeds and corms from Darwin.

According to the Aboriginal store workers, several young men who previously entered the store only to purchase tobacco, 'cool drinks' and 'take-away' foods, began to purchase other foods and groceries for their extended family during the intervention year.

8.4. Cost-benefits of the Minjilang nutrition intervention project

Although there was no attempt to cost specific intervention strategies individually, over the twelve month period the Minjilang project, including salaries, cost in the order of \$65,000¹⁰ (Addendum 3, A3.4). The cost of the nutrition intervention project was relatively trivial when compared with the high costs incurred in the delivery of clinically-orientated, high technology health care such as aerial ambulance services, clinical medical visits to the community, hospital-based services and admissions. Furthermore, the validation of the store-turnover method has obviated the need for frequent monitoring of costly biochemical parameters in future community-based nutrition intervention projects and would therefore assist greatly in additional budgetary constraint in other projects.

Measurable benefits to the Minjilang community included improvements in health outcomes, such as reduced risk factors for coronary heart disease and improved vitamin status. However, from the community perspective, there were several advantages which were not so readily measurable. Firstly, most Minjilang adults volunteered¹¹ that they felt "*stronger*" at the completion of the project than they had a year earlier (this may have been a feasible result of improved folate and haemoglobin status); several older subjects, particularly those older women who had experienced weight loss during the year, stated that they no longer experienced "*short wind*" on exertion. Secondly, the improved nutritional status of the community and the decrease in both prevalence and degree of risk factors for heart disease, may have reduced the number of hospital admissions¹², hence

¹⁰ Costs of biochemical assays were kept to a minimum during the study as several analytical laboratories involved charged nominal fees or donated staff, time and reagents to the project in view of its public health significance. Additional air travel was also provided by the NTDHCS at no cost to the project (of approximate commercial value \$1,800). The costs of the *entire* project which extended over a four year period, (involving pilot studies, the development of the store-turnover method, purchase of equipment, analysis and production of reports) actually cost in the order of \$ 200,000.

¹¹ Formal process evaluation was conducted throughout the intervention project, but details are to be published elsewhere as they are not central to the major thesis

¹² Relevant data collection continues

avoiding the social disruption which underpins such events in remote communities. Thirdly, the community experienced a real sense of pride in their ability to take their health into their own hands. That is, the success of the project directly addressed the failure psychology which has beset past health and nutrition projects in Aboriginal communities.

8.5. Developments arising from the project and implications for other community-based nutrition intervention projects in remote, centralised Aboriginal communities

As one development of the project, a nutrition policy has been implemented in several community stores managed by Arnhem Land Progress Association (ALPA), the Aboriginal owned enterprise responsible for the Minjilang store (Addendum 3, A3.3). ALPA now also employs an Aboriginal store worker in each community to focus on nutrition-related issues; these store workers are trained as 'good food people' by NTDHCS nutritionists (Lions *et al*, 1991). One of the store workers at Minjilang has been appointed as a 'good food person' and has continued to work with the community in the development of many of the initiatives of the project (The Health Report, ABC Radio 16/9/91). The NTDHCS has also developed a 'diabetes story project' which incorporates traditional story-telling and learning styles to specifically address the issue of impaired glucose tolerance in Aboriginal communities (Boyes *et al*, 1991). Both programs could be evaluated at an outcome level by application of the store-turnover method.

A further six remote, centralised Aboriginal communities have formally requested assistance with specific projects, based on the Minjilang model, to address the high prevalence of nutrition-related health problems. Relevant information has been extended to the communities and to the responsible health care delivery organisations (both community-controlled and government regulated). Developments have been frequently thwarted by limited availability of funds.

In April 1991, a national conference on Aboriginal nutrition in remote and rural communities was convened by MSHR at the request of the Australian Health Ministers Advisory Committee (AHMAC) in December 1990 and was sponsored by the CDCSH (MSHR, 1991). The conference demonstrated the value of a bi-cultural, multi-disciplinary forum for discussing nutritional problems and potential solutions in Aboriginal communities using methods which were culturally relevant, practical and scientifically

sound. The conference recommended on practical ways to help Aboriginal people to disseminate and act upon available nutrition information. The Minjilang nutrition intervention project provided a model study for this purpose.

8.6. Further research questions

As there was no attempt to evaluate independently the mix of intervention strategies applied simultaneously in this study, it is essential that they be ranked individually with respect to cost-effectiveness, sustainability and generalisability. The co-ordinated evaluation of these individual strategies would enable the recommendation of potentially successful, cost-effective and sustainable community-based strategies suitable for wide-spread implementation by other remote, centralised Aboriginal communities.

Evaluation of intervention strategies at an outcome level typically requires a comparative measurement of pre- and post-intervention change. As discernible changes in health indicators are not expected to be apparent over the relatively short time frame considered in most intervention projects in Aboriginal communities, it is expected that the outcome of principle interest would be quantitative change in dietary intake. With the validation of the store-turnover method, there is now an acceptable method for measuring dietary intake for this purpose. Moreover, the validation of the store-turnover method has obviated the need for monitoring of costly biochemical indicators of health and nutritional status to evaluate strategies in the long term.

8.6.1. Generalisability

Several features of the Minjilang project warrant discussion in the context of the generalisability of the project. Certainly some aspects of the Minjilang community may be considered pre-requisites for the development of projects in other communities. Firstly, the Iwadja and Marrgu of Croker Island had obtained inalienable freehold title to their land (granted under the NT Land Rights Act, 1976). Traditional beliefs and values were still strong; cultural pride was clearly evident. There was a high degree of social cohesion and stable, traditionally-based power structures still operated within the community. Few non-Aboriginals attempted to interfere with the management or administrative processes of the community, particularly within the sphere of health care delivery. Although kava was available regularly at the time of the study, the community had elected to officially prohibit alcohol under the NT Liquor Act. Many of these features are also evident in other

remote, centralised Aboriginal communities, particularly within the Northern Territory and the north-west region of South Australia.

It is also unlikely that the characteristics of the organisations (such as ALPA and MSHR) and of the individuals providing technical support to Minjilang were entirely unique. Certainly the Aboriginal research assistant was particularly enthusiastic and competent, most store managers were very co-operative, and I had been privileged to be actively involved in Aboriginal health issues for over ten years and had been taught much about Aboriginal culture. There are many committed and sympathetic individuals, particularly those working for Aboriginal community-controlled organisations, who would be capable and willing to support communities in their endeavours to improve dietary intake. This was evidenced by the enthusiasm and interest exhibited at the national conference on Aboriginal nutrition in remote and rural communities (MSHR, 1991).

Of primary importance was the fact that the people of Minjilang actually initiated the project. It is plausible that where similar requests for specific assistance are made by other communities, many aspects of the Minjilang project may be directly relevant.

8.6.2. Sustainability

It could be postulated that improvements in dietary intake and the resultant changes in nutritional status measured throughout the nutrition intervention project at Minjilang may not be sustainable within the community. In particular it was speculated by other non-Aboriginal health professionals that the thrust of the project would diminish once the researchers withdrew from Minjilang. This patronising view ignored the feelings of 'ownership' of the project within the community. Since the formal project was completed in June 1990, there is anecdotal evidence that several aspects of the project have been continued and developed within the community (The Health Report, ABC Radio 16/9/91). However application of the store-turnover method and/or repeated measure of the biological indicators of health and nutritional status of the community would be required to objectively appraise sustainability.

8.6.3. Cost-effectiveness

In future projects, the relative cost of each individual intervention strategy could be directly compared against the measures of effectiveness as determined by changes in

community dietary intake using the store-turnover method. Where available, the relative cost of each individual intervention strategy could also be directly compared against change in anthropometric, haematological and biochemical health indicators.

8.7. Conclusions

Firstly, the study demonstrated that it was possible to measure dietary intake in remote, centralised Aboriginal communities by systematic collection and analysis of invoice data from the community store (the store-turnover method).

Secondly, the store-turnover method was validated congruently against biological indicators of nutritional status and by appraisal of the face validity of the method.

Thirdly, the store-turnover method identified practical nutrition intervention strategies which were used as a rational basis for the development of a community-based nutrition intervention project.

Fourthly, the store-turnover method was shown to be a sensitive indicator of the effectiveness of the strategies applied during the intervention project at an outcome level.

Fifthly, the study has provided a successful model for future Aboriginal community-based nutrition intervention projects.

In summary, the successful development, validation, testing and application of the store-turnover dietary survey method has produced an appropriate tool to measure dietary intake quantitatively in remote, centralised Aboriginal communities; the method can be used to produce objective data which have wide implications for future health and nutrition programs in Aboriginal communities.

The project has further demonstrated that, where communities are involved in *all* stages of the development, implementation and evaluation of nutrition intervention projects and the projects are *genuinely* community-based (that is, community-initiated, community-directed and community-monitored), improvements in nutritional health and the reduction in some risk factors of non-communicable disease *are* possible.

ADDENDUM 1: Forms and equipment used in dietary studies and community surveys

A1.1. Example of food frequency questionnaire*




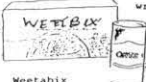



















Food	How many times per 'pay period'	Eaten only on 'pay day'	Eaten everyday	Other comments
Porridge				
Weetbix				
Cornflakes				
Baby cereal				
Other breakfast cereal				
Flour				
Bread (white)				
Bread (wholemeal)				
Bread (mixed grain)				
Rice				
Pasta				
Dry biscuits				
Sweet biscuits				
Golden syrup/honey				
Jam				
Sugar				
Tinned baked beans				
Tinned spaghetti				
Butter				
Margarine (poly-unsaturated)				
Margarine (other)				
Cooking oil (poly-unsaturated)				
Cooking oil (other)				
Dripping				
Peanut butter				
Cordial				
Cordial (diet)				
Cool drinks				
Cool drinks (diet)				
Fruit juice drink				
Fruit juice				
Tea				
Coffee				
Milo/ovaltine				
Beef (fresh cuts)				
Beef (mince)				
Lamb				
Pork				
Chicken (whole)				
Chicken (pieces)				
Tinned corn beef				
Tinned corn beef with cereal				
Tinned stew				
Tinned fish				
Eggs				
Cheese				
Frozen fish				
Fish				
Magpie geese				
Dugong				
Turtle				
Kangaroo				
Wallaby				
Perentie				
Goanna				
Rabbit				

* Not designed to be self administered

A1.1. Food frequency questionnaire (continued)

Food	How many times per 'pay period'	Eaten only on 'pay day'	Eaten everyday	Other comments
Milk (fresh)				
Milk (dried)				
Milk (condensed)				
Milk (flavoured)				
Tinned baby dinner				
Tinned baby cereal				
Confectionery				
Chocolate				
Potato crisps				
Extruded snacks				
Icecream				
Frozen yoghurt				
Flavoured yoghurt				
Meat pie				
Sausage roll				
Pastic				
Fried fish				
Fried chips				
Chicko/spring roll				
Dim Sims				
Hot tinned snacks				
Dried soup				
'Instant' meals				
Prepared sandwich				
Prepared stew				
Fruit				
Apples				
Oranges				
Bananas				
Mandarins				
Pears				
Peaches				
Mangoes				
Cherries				
others..				
Fruit (tinned)				
Vegetables (fresh)				
Potatoes				
Onions				
Cabbage				
Carrot				
Pumpkin				
Silverbeet				
Zucchini				
Capsicum				
Mushrooms				
others..				
Vegetables (tinned)				
Vegetables (frozen)				
Other foods...				

A1.2. Food frequency booklet designed with Aboriginal Health Worker: Example of layout

<p> write the number of eggs you eat in a day. e.g. 1 egg. 2 eggs.</p> <p>eggs:</p> <hr/> <p> write the number of slices you eat in a day. e.g. 1 slice of bacon. 1½ slice of bacon. 2 slices of bacon.</p> <p>bacon:</p> <hr/> <p> write the number of slices you eat in a day. e.g. 1 slice of bread. 1½ slices of bread. 2 slices of bread.</p> <p>wholemeal bread:</p> <hr/> <p> write the number of weetabix you eat in a day or a bowl of oats in a day. e.g. 1 weetabix 2 weetabix and oats. or 1 bowl of oats.</p> <hr/> <p> write the number of time you use vegemite or peanut butter on your bread. e.g. 1 spread of vegemite 1 spread of peanut butter.</p> <p>vegemite and peanut butter:</p> <hr/> <p> write the number of teaspoons of sugar you use in a day. e.g. 1 teaspoon of sugar 1½ teaspoon of sugar 2 teaspoon of sugar 2½ teaspoon of sugar</p> <p>sugar:</p>	<p> write the number of fish you eat in the day. e.g. 1 fish. 1½ fish. 2 fish. 2½ fish.</p> <hr/> <p> write the part of the chicken you have eaten in the day. e.g. (L) for leg. (W) for wing. (B) for breast.</p> <hr/> <p> write the numbers of chops you eat in the day. e.g. 1 chop. 1½ chops. 2 chops. 2½chops.</p> <hr/> <p> write which part of the crab you have eaten. e.g. (B) body. (L) legs.</p> <hr/> <p> write which part of the goose you have eaten. e.g. (B) Breast (L) legs (N) Neck (W) Wing</p> <hr/> <p> write the number of steaks you eat in the day. e.g. 1 steak. 2 steak meat with fat. (Y) for fat. meat with no fat. (N) for no fat.</p>
<p> write the number of cups you drink in a day. e.g. 1 cup of juice. 1½ cup of juice. 2 cups of juice.</p> <p>fruit juice:</p> <hr/> <p> write the number of cans you drink in a day. e.g. 1 can of diet drink 1½ can of diet drink</p> <p>diet cool drink.</p> <hr/> <p> write the number of cool drinks you have in a day. (coke, Fanta, etc..) e.g. 1can of cool drink. 2can of cool drink.</p> <hr/> <p> write the number of a cup a tea or coffee drink in a day. 1 cup of tea 1 cup of coffee</p> <p>Tea and Coffee:</p> <hr/> <p> write the number of wine you drink in a day or cans you drink in a day. e.g. 1 glass of wine 1 can of beer.</p> <p>wine and beer.</p>	<p> Tick the box if you have eaten a sandwich made from the store today.</p> <hr/> <p> Write the number of slices you eat in a day. e.g. 1 slice 1½ slices 2 slices</p> <hr/> <p> Tick the box every time you eat a pie in a day.</p> <hr/> <p> Tick the box every time you eat a sausage roll in a day.</p> <hr/> <p> write the number of times you spread butter on your bread or use butter for cooking. butter: (teaspoon of butter)</p> <hr/> <p> write the number of times you spread margarine on your bread or use margarine for cooking. (teaspoon of margarine for cooking).</p>

A1.3. Section of store turnover tabulation spreadsheet compiled from store invoice data

Code	Food	Bulk unit	Supplied (g or ml)	1989		
				8/2	21/2	7/3
7	Custard powder	375 g X 12	4500	-	-	-
1010	Flour (plain)	16 kg X 1	16000	12	10	16
1010	"	1 kg X 12	12000	4	3	4
1010	"	1 kg X 15	15000	1	1	-
1011	Flour (self-raising)	1 kg X 12	12000	1	1	1
1012	Flour (wholemeal)	1 kg X 12	1200	-	1	-
17	Oatmeal	750 g X 24	1800	-	-	-
19	Rice (white)	1 kg X 12	1200	2	2	3
19	"	500 g X 12	6000	2	1	1
5017	Rice (brown)	1 kg X 12	1200	-	-	-
26	Pasta	500 g X 36	1800	-	-	-
28	Tinned Spaghetti	440 g X 24	10560	-	-	1
569	Baked beans	450 g X 24	10800	-	-	-
1005	Cornflakes	500 g X 18	9000	1	-	-
57	Weetbix	375 g X 24	9000	2	1	2
57	"	750 g X 12	9000	-	1	1
70	Biscuits (sweet)	200 g X 20	4000	4	2	4
70	"	250 g X 20	5000	2	2	-
70	"	500 g X 16	8000	1	-	-
73	Biscuits (dry)	250 g X 20	5000	-	1	-
83	Cake	400 g X 12	4800	5	3	2
107	Icecream	2 l X 6	12000	10	2	-
107	"	5 l X 1	5000	-	2	-
107	"	100g X24 X12	57600	4	1	3
115	Tinned rice pudding	450 g X 12	10800	2	-	2
130	UHT milk	1 l X 12	12000	-	2	2
130	"	500 ml X 24	12000	-	-	-
132	Condensed milk	400 g X 24	9600	1	1	1
135	Dried milk	1 kg X 6	6000	7	7	7
135	"	300 g X 24	7200	3	2	3
1000	Margarine (PU)	500 g X 24	12000	4	4	4
1000	"	1 kg X 12	12000	-	-	-
195	Vegetable oil	750 ml X 12	9000	1	1	-
195	"	2 l X 6	12000	-	1	1
196	Vegetable oil (PU)	750 ml X 12	9000	-	-	-
196	"	2 l X 6	12000	-	-	-
838	Peanut butter	375 g X 12	4500	-	-	-
843	Sugar	2 kg X 12	2400	2	2	2
843	"	1 kg X 15	15000	20	16	20
1065	Cool drinks (sweet)	1.25 l X 12	15000	5	7	5
1065	"	375 ml X 24	9000	84	68	64
1065	"	500 ml X 24	12000	14	12	16
1066	Cool drinks (diet)	1.25 l X 12	15000	-	-	-
1065	"	375 ml X 24	9000	2	2	1

etc.

A1.4. Examples of consent forms used for Minjilang surveys

a. June 1989

CONSENT

I give permission for the Menzies School of Health Research to use a small amount of my blood (total = 27.5 ml) to check for diabetes, heart, liver disease and some vitamin levels, and to measure my height, weight, arm, hip and waist and blood pressure.

I understand that the results of these tests will be made available to me as soon as possible.

Signed.....

Witnessed.....
(MSHR)

Date.....

b. September 1989, December 1989, March 1990 and June 1990

CONSENT

I give permission for the Menzies School of Health Research to use a small amount of my blood (total = 25 ml) to monitor for diabetes, heart disease and some vitamin levels, and to measure my height, weight, arm, hip and waist and blood pressure for comparison with previous measurements.

I understand that the results of these tests will be made available to me as soon as possible.

Signed.....

Witnessed.....
(MSHR)

Date.....

A1.5. Equipment and materials required each survey (n = approx 60 adults)

a. To collect bloods (fasting 25 ml)

Syringes 60 x 20 ml
Needles 60 x size 21 gauge 9
Cleansing swabs x 60
Cottonwool swabs x 60
Elastoplast x 60
Sharps containers
 large x 1
 small x 2
Autoclave bags x 6
Adhesive numbers for blood tubes (1 to 60 x 30 each)
Gloves x 2 boxes (small and medium)
Tourniquet x 2

b. To process bloods

*collecting tubes
 120 x 10 ml Plain (yellow top)
 120 x 2.5 ml Fluoride (red top)
 120 x 1 ml paediatric haematology tubes (pink top)
 60 x 2.5 ml EDTA
 60 x 5 ml Heparin (Orange top)
*processing/transport
 180 x 5 ml plain glass (white screw tops)
 180 x 5 ml plain plastic (yellow top)
Centrifuge
Alfoil
Trichloroacetic acid x 300 ml
Polystyrene test tubes racks x 10
Plastic pasteur pipettes x 400
(autoclave bags and polystyrene racks as above)

c. To store/transport samples

Icebricks
 'blue' water based x 20
 'red' formula (-12°C) x 4
Small polystyrene eskies x 20
1 large esky
Masking tape
Waterproof markers x 4
Temperature indicators

d. Anthropometric measurement

Fibreglass Tape measure x 2
Standardised adult scales x 1
Stadiometer x 1

f. Blood pressure

Automatic blood pressure machine x 1 (Dynamap)

g. Miscellaneous

Waterproof, absorbent cloths
Sellotape dispensers x 2
Large cardboard boxes x 5
Biros
Felt pens (waterproof)
Torch
Tissues
Digital clock
Pathology forms
 haematology
 biochemistry work sheet
Plastic bags
Applicator sticks (1 box)
Gilson pipette (1 ml) and blue tips

A1.6. Example of data sheet used for Minjilang surveys

(MSHR letterhead)

MINJILANG HEALTH AND NUTRITION PROJECT.
HEALTH SURVEY

LAB NO.....
PREVIOUS IDCOD.....

SURVEY NUMBER.....

DATE:.....

NAME:.....

SEX M/F

WEIGHT.....kg. HEIGHT.....cm.

WAIST.....cm. HIPS.....cm.

MID-UPPER ARM CIRCUMFERENCE:.....cm.

BLOOD PRESSURE:...../.....mm Hg

PULSE.....

QUESTIONS.

Do you smoke cigarettes?.....Y/N.

If yes, how many cigarettes do you smoke a day?.....

Do you drink alcohol?.....Y/N.

If yes, when was the last time you drank alcohol?

.....

Do you drink kava?.....Y/N.

If yes, when was the last time you drank kava?

.....

ADDENDUM 2: Additional data and analysis, Minjilang survey

Table A2.1a: Minjilang population, determined by three monthly census

Age/Sex Group	June 1989 n (%)	September 1989 n (%)	December 1989 n (%)	March 1990 n (%)	June 1990 n (%)
<=3 yrs, both sexes	6 (4.3)	8 (6.3)	10 (6.7)	8 (6.5)	9 (6.0)
4-7 yrs, both sexes	12 (8.6)	11 (8.6)	13 (8.7)	10 (8.2)	15 (10.0)
7-18 yrs, males	25 (18.0)	20 (15.6)	26 (17.3)	18 (14.7)	26 (17.3)
7-18 yrs, females	24 (35.3)	22 (17.2)	23 (15.3)	20 (16.4)	28 (18.7)
>=18 yrs, men	30 (21.6)	26 (20.3)	31 (20.7)	27 (22.1)	32 (21.4)
>=18 yrs, women	36 (25.9)	34 (26.6)	39 (26.0)	32 (26.2)	30 (20.0)
pregnant women	1 (0.7)	2 (1.6)	2 (1.3)	2 (1.6)	3 (2.0)
breastfeeding women	5 (3.6)	5 (3.9)	7 (4.7)	5 (4.2)	7 (4.6)
Total	139	128	151	122	154

Table A2.1b: Minjilang population, determined by fortnightly roll call/checklist (mean \pm se)

Age/Sex Group	June 1989	September 1989	December 1989	March 1990	June 1990
Mean population of previous three months	-	152(3.4)	169(2.7)	160(4.1)	150(3.3)

Table A2.1c: Control community population, estimated by Health Centre Staff at Control Community

Age/Sex Group	June 1989	September 1989	December 1989	March 1990	June 1990
Approximate population	360	300	290	300	300

Table A2.2a. Recommended nutrient density for age/sex groups

Nutrient	< =3 yrs both sexes	4-7 yrs both sexes	7-18 yrs males	7-18 yrs females	adult men	adult women	pregnant women	breastfeeding women
Thiamine (mg/1000 kJ)	0.09	0.10	0.11	0.12	0.10	0.10	0.11	0.11
Riboflavin(mg/1000 kJ)	0.14	0.15	0.17	0.18	0.16	0.14	0.16	0.16
Niacin (mg/1000 kJ)	1.70	1.67	1.89	2.06	1.73	1.55	1.60	1.70
Vitamin B ₆ (mg/1000 kJ)	0.13	0.15	0.17	0.17	0.15	0.14	0.13	0.18
Folate (µg/1000 kJ)	17.9	13.9	18.9	23.0	18.2	23.8	42.6	33.0
Vitamin B ₁₂ (µg/1000 kJ)	0.18	0.21	0.19	0.23	0.18	0.24	0.32	0.24
Vitamin C (mg/1000 kJ)	5.36	4.17	2.83	3.45	3.64	3.57	6.38	5.66
Vitamin A (eq/1000 kJ)	53.6	48.6	68.4	83.3	68.2	89.3	79.8	113.2
Vitamin E (mg/1000 kJ)	0.89	0.83	0.99	1.03	0.91	0.83	0.75	0.90
Iron (mg/1000 kJ)	1.25	0.97	1.08	1.32	0.64	1.67	3.09	1.32
Zinc (mg/1000 kJ)	0.80	0.83	1.13	1.38	1.09	1.43	1.70	1.70
Calcium (mg/1000 kJ)	125	111	113	115	73	95	117	113

Table A2.2b. Recommended nutrient density, Minjilang*

Nutrient	Recommended density
Thiamine	0.10 mg/1000 kJ
Riboflavin	0.16 mg/1000 kJ
Niacin	1.77 mg/1000 kJ
Vitamin B ₆	0.15 mg/1000 kJ
Folate	21.0 µg/1000 kJ
Vitamin B ₁₂	0.21 µg/1000 kJ
Vitamin C	3.74 mg/1000 kJ
Vitamin A	74.8 retinol eq/1000 kJ
Vitamin E	0.92 mg/1000 kJ
Iron	1.19 mg/1000 kJ
Zinc	1.22 mg/1000 kJ
Calcium	102 mg/1000 kJ

* Based on Table A2.1a and Table A2.2a

Table A2.3a. Traditional vegetable foods obtained at least once at Croker Island between June 1989 and June 1990

Iwadja name	European name	Part eaten	habitat
<u>Available all year</u>			
<i>marrwadj</i>	lily	stem, seed	Swamp, lagoons, rivers bulb, leaves
<i>mudala</i>	pandanus	nut	Coastal dunes, open forest, Swamp, lagoons and rivers
<u>Wet season (October to May)</u>			
<i>lurrigala</i>	cashew*	nut	Coastal dunes, Rainforest
<i>irriyala</i>	red apple	fruit	Coastal dunes, Rainforest Open forest
<i>erwal</i>	white plum	fruit	Swamp, lagoons, rivers Coastal dunes, open forest, Rainforest
<i>warrwardang</i>	white apple	fruit	Coastal dunes, open forest
<i>garbiyoyo</i>	cherry plum	fruit	Rainforest
<i>yamba</i>	billygoat plum	fruit	Coastal dunes, open forest, Rainforest
<u>Dry season (May to October)</u>			
May to October			
<i>lungun</i>	long yam	root	Rainforest
May to August			
<i>kanirr</i> <i>kurldi</i>	cheeky yam*	root	Rainforest
<i>kunkunubaj</i>	yam	root	Rainforest
March to December			
<i>marrunj</i>	kentia palm	shoot	Forest, Rainforest

* Requires special preparation

Table A2.3b. Traditional animal foods obtained at least once at Croker Island between June 1989 and June 1990.

Iwadja name	European name	habitat
<u>Available all year</u>		
<i>weelan</i> <i>wimbarr</i> <i>kubulak</i> <i>bortlor</i>	honey	Open Forest
<i>mandali</i>	schnapper	Sea, lagoons, rivers
<i>eagaman</i>	catfish	Sea, rivers
<i>woodar</i> <i>weedee</i>	mullet	sea
<i>jinumbu</i>	kingfish	sea
<i>arrayi</i>	oyster	coast
<i>kurrurdalk</i>	crab	coast
<i>muminga</i>	clamshell	coast
<i>kubiya</i>	cockle	coast
<i>marndingunjunj</i>	dugong	sea
<i>manbiri</i>	green-back turtle	sea
<i>urwa</i>	flat-back turtle	sea
<i>munjar</i>	longneck turtle	swamps, rivers
<i>warrgarrk</i> <i>galdj</i>	goanna	widespread
<i>kundaman</i>	frilled neck lizard	widespread
<i>wadbadj</i>	mussel	swamps, lagoon, rivers
<u>Dry season</u>		
<i>mangkawuli</i>	barramundi	sea
<i>weeloo</i>	catfish	sea
<u>Wet season</u>		
<i>mangkawuli</i>	barramundi	rivers
<i>kalakalak</i>	magpie geese	swamps

Table A2.4. Fatty acid analysis of some traditional foods used at Minjilang from June 1989 to June 1990

Sample Lipids	Total	12:0	14:0	14:1	16:0	16:1	18:0	18:1	18:2 w6	18:3	20:1	20:2	20:3 w6	20:4 w6	20:5 w3	22:4 w6	22:5 w6	22:5 w3	22:6 w3
Barramundi (n=2)	0.68	-	0.5	-	19.5	1.4	11.0	15.5	2.5	0.4	-	0.4	0.5	15.0	1.4	1.7	3.2	2.6	12.4
Turtle fat (n=3)	83.5	17.0	10.8	0.6	18.3	6.9	5.1	32.7	1.6	1.0	-	-	0.2	1.3	0.7	0.4	-	1.3	0.2
Turtle meat	1.23	3.5	3.1	-	16.2	2.7	10.2	28.2	3.9	1.0	0.5	0.4	0.9	14.2	4.3	1.9	-	3.6	0.8
Goose fat	34.9	-	0.6	0.1	26.3	5.1	6.4	55.8	3.9	0.2	-	-	-	0.4	-	-	-	-	-
Dugong meat	1.6	0.1	4.9	0.2	27.4	3.8	4.4	46.8	3.1	1.9	0.1	-	0.2	2.5	0.5	0.2	-	0.8	0.1
Dugong fat	71.4	0.1	8.0	0.3	27.1	3.7	3.0	53.5	0.8	1.4	0.6	0.1	-	0.2	-	0.1	-	0.2	-
Topside (store purchased)	1.21	-	1.1	0.3	24.1	1.5	17.7	30.7	8.6	1.8	0.8	-	1.1	3.8	1.1	0.2	-	1.7	-

Table A2.5. Anthropometric, metabolic and haematological data Minjilang, June 1989

	men		women	
	< 35 yrs	> 35 yrs	< 35 yrs	> 35 yrs
n	16	17	14	21
Age (yr)	24.0±1.1	47.0±2.6	25.9±1.3	49.2±4.6
BMI (kg/m ²)	24.1±1.1	23.3±1.2	20.9±1.4	24.4±1.1
Waist:Hip	0.89±0.02	0.96±0.02	0.83±0.02	0.89±0.01
Mid-arm Circumference(cm)	28.5±0.9	28.1±0.7	24.0±1.1	25.8±0.9
Diastolic B.P. (mm Hg)	73.0±2.6	74.1±2.1	66.7±2.1	73.4±2.2
Systolic B.P. (mm Hg)	127.4±3.7	122.0±3.1	109.8±3.1	128.2±4.5
Pulse (beats/min)	80.1±3.0	84.6±2.5	85.3±2.8	78.0±2.2
Cholesterol (mmol/l)	6.1±0.3	6.1±0.3	5.1±1.2	6.3±0.3
HDL-cholesterol (mmol/l)	1.1±0.06	1.2±0.07	1.1±0.06	1.1±0.05
Triglyceride (mmol/l)	1.7±0.2	2.1±0.7	1.5±0.2	2.0±0.1
Fasting insulin (mu/l)	9.7±1.3	16.4±5.6	18.9±4.9	19.8±2.7
2hr insulin (mu/l)	18.1±2.9	25.3±4.3	59.9±15.0	75.3±10.0
Fasting glucose (mmol/l)	4.9±0.1	6.3±0.6	4.7±0.1	6.2±0.5
2hr glucose (mmol/l)	4.7±0.2	8.1±1.3	5.5±0.4	9.1±1.2
S. fructosamine(mmol/l)	219.1±4.3	253.5±20.2	212.1±5.4	238.1±12.2
Ferritin (µg/l)	84.4±12.6	199.7±35.6	43.2±10.6	125.8±20.3
Serum protein (g/l)	80.4±0.9	82.5±0.9	80.8±1.7	83.9±1.3
Serum albumin (g/l)	43.4±0.5	42.2±0.7	41.5±0.8	41.9±0.4
γGT (U/l)	167.3±70.4	85.1±18.1	47.8±10.8	69.3±12.3
Haemoglobin (g/l)	156.6±2.0	149.4±3.2	132.3±2.4	140.7±2.4
MCV (fL)	88.0±0.8	91.1±1.1	88.5±1.3	89.7±0.9
Haematocrit	0.48±0.01	0.46±0.01	0.41±0.01	0.43±0.01
RDW (%)	14.0±0.3	14.6±0.3	14.4±0.3	14.3±0.3
Serum folate (µg/l)	1.82±0.22	2.80±0.54	1.87±0.25	1.90±0.19
Red cell folate (µg/l)	94.4±5.9	82.4±7.7	77.0±6.0	78.1±4.0
Serum thiamine (µg/l)	7.6±0.6	8.0±0.8	6.9±0.7	7.3±0.3
Red cell thiamine (µg/l)	65.6±3.1	59.0±3.8	56.5±4.5	64.3±3.1
Serum pyridoxal (nmol/l)	41.7±5.2	45.4±5.1	40.4±2.0	38.7±2.9
Serum cobalamin (ng/l)	631±51	847±44	755±56	774±19
Pl. ascorbic acid (mg/l)	4.13±0.57	3.57±0.52	4.19±1.07	4.25±0.73
Plasma retinol (mg/100ml)	56.6±7.0	73.4±5.2	55.6±4.3	61.4±2.5
Pl. α-tocopherol (mg/l)	12.3±0.8	12.7±1.2	11.0±1.2	12.9±0.6
Pl. α-carotene (mg/100ml)	5.2±1.8	1.9±0.9	1.0±0.5	0.4±0.1
Pl. β-carotene (mg/100ml)	45.9±6.9	74.4±20.5	53.8±7.7	55.5±4.9

Table A2.6. Anthropometric and metabolic data Minjilang, June 1989 (excluding diabetics)

	men		women	
	< 35 yrs	> 35 yrs	< 35 yrs	> 35 yrs
n	16	14	14	18
BMI (kg/m ²)	24.1±1.1	22.7±1.3	20.9±1.4	22.9±1.1
Weight (kg)	70.9±3.4	66.5±4.2	55.8±3.9	60.7±3.2
Waist:Hip	0.89±0.02	0.95±0.02	0.83±0.02	0.88±0.01
Triglyceride (mmol/l)	1.7±0.2	2.2±0.8	1.5±0.2	1.9±0.2
Fasting insulin (mu/l)	9.7±1.3	9.7±1.3	18.9±4.9	20.1±3.3
2hr insulin (mu/l)	18.1±2.9	19.6±2.4	59.9±15.0	80.6±11.1
Fasting glucose (mmol/l)	4.9±0.1	5.4±0.2	4.7±0.1	5.4±0.2
2hr glucose (mmol/l)	4.7±0.2	5.8±0.4	5.5±0.4	7.1±0.6
S. fructosamine (mmol/l)	219±4.3	222±3.1	212±5.4	219±5.5
Cholesterol (mmol/l)	6.1±0.3	6.2±0.4	5.1±1.2	6.2±0.3
HDL-cholesterol (mmol/l)	1.1±0.06	1.19±0.06	1.1±0.06	1.09±0.06
Cholesterol:HDL	5.6±0.4	5.4±0.5	4.6±1.6	6.0±0.4

Table A2.7. Anthropometric, metabolic and haematological data Minjilang, June 1990

n	men		women	
	< 35 yrs	> 35 yrs	< 35 yrs	> 35 yrs
	11	11	10	14
Age (yr)	24.6±1.7	45.7±2.6	24.7±1.4	49.0±3.0
BMI (kg/m ²)	22.3±1.0	23.1±1.6	22.1±1.6	25.9±1.5
Waist:Hip	0.85±0.02	0.93±0.02	0.80±0.01	0.87±0.01
Mid-arm Circumference(cm)	28.2±0.7	29.1±0.8	25.5±1.3	28.6±1.1
Diastolic B.P. (mm Hg)	70.9±2.0	69.3±2.4	61.3±1.7	68.3±2.5
Systolic B.P. (mm Hg)	118.3±2.7	119.0±2.7	105.9±2.5	117.2±4.9
Pulse (beats/min)	78.6±3.9	78.9±4.6	77.1±2.7	73.6±3.6
Cholesterol (mmol/l)	5.06±0.27	5.40±0.36	4.56±0.24	5.66±0.25
HDL-cholesterol (mmol/l)	1.02±0.05	1.04±0.06	1.00±0.08	0.99±0.06
Triglyceride (mmol/l)	1.33±0.71	1.41±0.88	1.16±0.85	2.26±0.90
Fasting insulin (mu/l)	10.0±1.5	11.4±1.9	26.0±6.8	21.3±3.2
2hr insulin (mu/l)	22.1±7.8	18.7±3.8	49.3±15.4	59.0±14.5
Fasting glucose (mmol/l)	5.1±0.2	7.0±1.2	5.1±0.2	6.5±1.4
2hr glucose (mmol/l)	6.6±0.6	8.8±3.0	6.1±0.5	9.2±1.1
S. fructosamine (mmol/l)	232.9±6.7	277.5±30.7	219.6±8.0	240.7±11.7
Ferritin (µg/l)	161.2±39.4	241.8±34.1	57.6±4.3	167.4±5.0
Serum protein (g/l)	78.6±2.1	81.1±1.4	78.9±2.3	81.2±0.9
Serum albumin (g/l)	42.2±1.1	42.1±0.9	40.8±1.2	39.7±0.5
γGT (U/l)	119.0±74.4	67.4±13.0	33.7±5.5	59.4±15.5
Haemoglobin (g/l)	157.6±2.0	152.0±3.9	136.6±1.9	141.1±3.0
MCV (fL)	88.5±0.8	87.6±1.1	85.0±1.6	86.8±0.9
Haematocrit	0.47±0.01	0.45±0.01	0.40±0.01	0.41±0.01
RDW (%)	13.5±0.3	14.2±0.4	13.7±0.3	13.8±0.3
Serum folate (µg/l)	2.6±0.6	3.6±0.5	3.0±0.4	2.3±0.3
Red cell folate (µg/l)	188.6±23.7	198.9±18.8	203.1±38.9	235.1±49.3
Serum thiamine (µg/l)	6.1±0.5	9.0±0.9	8.0±0.9	11.6±0.8
Red cell thiamine (µg/l)	58.5±2.8	55.1±2.6	57.3±5.1	54.1±3.3
Serum pyridoxal (nmol/l)	67.0±9.2	74.5±11.0	48.0±5.6	68.4±9.8
Serum cobalamin (ng/l)	679.5±55.7	108.0±31.0	700.0±52.5	770.4±45.3
Pl. ascorbic acid (mg/l)	9.34±4.29	10.35±5.35	14.90±7.28	7.46±3.23
Pl. retinol (mg/100ml)	67.2±9.0	80.9±6.4	62.9±5.7	63.7±6.1
Pl. α-tocopherol (mg/l)	11.0±1.2	12.3±1.1	9.9±0.6	12.9±1.7
Pl. α-carotene (mg/100ml)	16.9±5.3	8.1±1.4	12.8±2.2	12.9±5.0
Pl. β-carotene (mg/100ml)	52.0±13.9	80.0±31.9	82.7±19.7	84.3±19.3

Table A2.8. Anthropometric and metabolic data Minjilang, June 1990 (excluding diabetics)

n	men		women	
	< 35 yrs	> 35 yrs	< 35 yrs	> 35 yrs
	11	9	10	11
BMI (kg/m ²)	22.3±1.0	22.3±1.9	22.1±1.6	25.9±1.5
Weight (kg)	66.1±3.9	64.1±5.9	57.8±4.6	63.7±3.8
Waist:Hip	0.85±0.02	0.92±0.03	0.80±0.01	0.86±0.02
Triglyceride (mmol/l)	1.33±0.15	1.41±0.24	1.16±0.28	2.26±0.27
Fasting insulin (mu/l)	10.0±1.5	9.5±1.7	26.0±6.8	18.0±3.1
2hr insulin (mu/l)	22.1±7.8	18.6±5.0	49.3±15.4	67.4±18.1
Fasting glucose (mmol/l)	5.1±0.2	5.2±0.2	5.1±0.2	5.3±0.2
2hr glucose (mmol/l)	6.6±0.6	4.3±0.4	6.1±0.5	7.3±0.6
S. fructosamine (mmol/l)	232.9±6.7	233±6.5	219.6±8.0	221±3.5
Cholesterol (mmol/l)	5.1±0.3	5.2±0.4	4.6±0.2	5.4±0.3
HDL-cholesterol (mmol/l)	1.02±0.05	1.04±0.08	1.00±0.08	0.94±0.05
Cholesterol:HDL	5.1±0.3	5.3±0.7	4.9±0.5	6.0±0.4

Table A2.9. Anthropometric, biochemical and haematological data by age and sex groups pre-intervention (June 1989) and post-intervention (June 1990) Minjilang (mean \pm se)

	men < 35 years			> =35 years			women < 35 years			> 35 years		
	n	value	T	n	value	T	n	value	T	n	value	T
BMI (kg/m ²)	10	22.4 \pm 1.1 22.3 \pm 1.1	-0.19	11	23.0 \pm 1.7 23.1 \pm 1.6	0.16	9	22.3 \pm 1.9 22.2 \pm 1.8	-0.32	13	26.9 \pm 1.6 26.1 \pm 1.6	-3.81**
Weight (kg)	10	66.2 \pm 4.1 66.3 \pm 4.3	0.15	11	65.7 \pm 5.1 65.9 \pm 4.9	0.29	9	58.9 \pm 5.4 58.6 \pm 5.0	-0.26	13	71.2 \pm 4.3 69.1 \pm 4.3	-3.82*
Waist:Hip	10	0.87 \pm 0.02 0.85 \pm 0.02	-1.32	11	0.95 \pm 0.02 0.93 \pm 0.02	-1.13	9	0.81 \pm 0.02 0.81 \pm 0.01	-0.56	13	0.90 \pm 0.02 0.87 \pm 0.02	-3.86*
Systolic B.P. (mm Hg)	9	124.2 \pm 2.9 118.4 \pm 3.0	-2.54*	11	118.9 \pm 3.2 119.0 \pm 2.7	0.03	10	107.7 \pm 3.3 105.3 \pm 2.7	-0.72	13	127.0 \pm 5.3 119.6 \pm 4.7	-2.76*
Diastolic B.P. (mm Hg)	9	69.9 \pm 3.5 70.9 \pm 2.3	-0.24	11	73.5 \pm 3.0 69.3 \pm 2.5	2.39*	10	66.8 \pm 2.2 61.1 \pm 1.9	-2.6*	13	73.2 \pm 2.7 69.3 \pm 2.4	-1.43
Cholesterol (mmol/l)	10	5.5 \pm 0.3 5.1 \pm 0.3	-2.18	11	6.1 \pm 0.3 5.4 \pm 0.4	-3.56***	10	4.8 \pm 0.4 4.5 \pm 0.3	-1.02	13	6.5 \pm 0.2 5.8 \pm 0.2	-3.61**
HDL-cholesterol (mmol/l)	10	1.2 \pm 0.1 1.0 \pm 0.1	-1.67	11	1.16 \pm 0.08 1.04 \pm 0.06	-2.11	10	1.13 \pm 0.05 0.98 \pm 0.09	-1.37	13	1.06 \pm 0.07 0.98 \pm 0.07	-1.30
Cholesterol/HDL	10	5.0 \pm 0.4 5.0 \pm 0.3	-0.10	11	5.5 \pm 0.5 5.5 \pm 0.6	-0.2	10	4.3 \pm 0.3 5.0 \pm 1.8	1.58	13	6.4 \pm 0.4 6.2 \pm 0.4	-0.55
Triglyceride (mmol/l)	9	1.5 \pm 0.2 1.3 \pm 0.2	-1.44	10	2.4 \pm 1.1 1.4 \pm 0.3	1.1	8	1.5 \pm 0.2 1.2 \pm 0.3	-1.38	13	2.0 \pm 0.2 2.3 \pm 0.2	2.07
Fasting insulin (mu/l)	9	10.3 \pm 1.5 10.3 \pm 1.6	0	10	9.5 \pm 1.4 11.4 \pm 1.9	1.3	8	14.1 \pm 2.9 26.0 \pm 6.7	1.92	13	21.8 \pm 3.0 22.5 \pm 3.2	0.17

* p = < 0.05 ** p = < 0.01 *** p = < 0.005 † p = < 0.001 †† p = < 0.0005 ††† p = < 0.0001

Table A2.9. (continued)

	men < 35 years			> = 35 years			women < 35 years			> 35 years		
	n	value	T	n	value	T	n	value	T	n	value	T
2hr insulin (μ u/l)	7	19.7 \pm 6.2 22.9 \pm 8.7	0.88	9	22.1 \pm 4.1 18.7 \pm 3.8	-0.78	8	59.8 \pm 20.0 49.3 \pm 15.4	-1.16	12	82.6 \pm 15.3 60.4 \pm 15.7	-1.18
Fasting glucose (mmol/l)	9	4.8 \pm 0.2 5.1 \pm 0.2	1.2	10	6.6 \pm 0.9 7.0 \pm 1.2	0.62	8	4.7 \pm 0.1 5.2 \pm 0.2	2.37*	13	6.8 \pm 0.8 6.6 \pm 0.7	-0.64
2hr glucose (mmol/l)	6	5.0 \pm 0.4 6.9 \pm 0.8	3.01*	9	8.6 \pm 2.3 8.8 \pm 3.0	0.26	8	5.8 \pm 0.5 6.3 \pm 0.6	0.83	11	11.9 \pm 1.9 9.9 \pm 1.2	-2.40*
Serum fructosamine (mmol/l)	9	220 \pm 5.3 235 \pm 7.0	1.97	10	260 \pm 26 278 \pm 31	2.1	8	209 \pm 6.3 220 \pm 8.0	1.22	13	240 \pm 15 243 \pm 12	0.33
γ GT (U/l)	10	153 \pm 107 128 \pm 82	-0.94	11	78 \pm 18 67 \pm 13	-1.48	10	49.8 \pm 14.9 35.4 \pm 5.8	-1.32	13	77.2 \pm 18.3 61.7 \pm 16.6	-3.82**
Haemoglobin (g/l)	10	157 \pm 2.4 157 \pm 2.2	0.25	11	150 \pm 3 152 \pm 4	0.88	10	132.5 \pm 2.7 136.9 \pm 2.1	2.05	13	140.0 \pm 3.3 141.2 \pm 3.4	0.88
MCV (fL)	10	88.9 \pm 1.0 88.5 \pm 0.9	-0.68	11	89.4 \pm 1.3 87.6 \pm 1.1	-2.37*	10	87.8 \pm 1.8 85.3 \pm 1.7	-3.7***	13	90.2 \pm 1.0 87.1 \pm 1.0	-7.25***
Haematocrit	10	0.48 \pm 0.01 0.47 \pm 0.01	-1.68	11	0.46 \pm 0.01 0.45 \pm 0.01	-1.92	10	0.41 \pm 0.01 0.41 \pm 0.01	-0.68	13	0.43 \pm 0.01 0.41 \pm 0.01	-2.66*
RDW (%)	10	13.7 \pm 0.2 13.3 \pm 0.3	-2.18	11	14.3 \pm 0.4 14.2 \pm 0.4	-0.41	10	14.8 \pm 0.4 13.7 \pm 0.4	-3.02*	13	14.1 \pm 0.33 13.8 \pm 0.3	-1.08

* p = < 0.05 ** p = < 0.01 *** p = < 0.005 ' p = < 0.001 " p = < 0.0005 "" p = < 0.0001

Table A2.9. (continued)

	men <35 years			≥35 years			women <35 years			≥35 years		
	n	value	T	n	value	T	n	value	T	n	value	T
Serum folate ($\mu\text{g/l}$)	9	1.71±0.26 2.77±0.59	1.75	11	2.6±0.6 3.6±0.5	1.97	10	1.7±0.2 3.0±0.4	3.01*	13	2.1±0.3 2.4±0.3	1.20
Red cell folate ($\mu\text{g/l}$)	8	88.5±3.4 192.3±26.5	4.09***	11	88.1±10.1 198.9±18.8	6.77*	10	74.4±6.7 235.1±58.3	2.80*	13	82.2±5.6 185.3±11.3	8.49***
Serum thiamine ($\mu\text{g/l}$)	9	6.2±0.3 5.7±0.4	-0.89	11	8.3±1.1 9.0±1.0	0.77	10	7.1±1.0 8.0±1.1	0.58	13	7.1±0.5 11.9±1.4	3.66**
Red cell thiamine ($\mu\text{g/l}$)	9	66.0±3.3 58.7±2.6	-1.87	11	57.6±4.4 55.1±2.6	-0.71	10	58.9±5.9 57.3±5.1	-0.19	13	66.5±4.4 54.2±3.7	-3.25*
Serum pyridoxal (nmol/l)	9	44.9±5.3 68.4±10.0	3.7**	11	48.5±7.4 74.5±11.0	2.43*	10	39.9±2.9 48.0±5.6	1.37	13	38.5±3.6 70.9±10.3	3.79**
Serum cobalamin (ng/l)	9	576.1±70.4 670.6±61.4	1.04	11	845±38 805±31	-1.29	10	703±65 700±52	-0.05	13	750±19 768±49	0.36
Plasma ascorbic acid (mg/l)	9	4.17±0.78 10.09±4.72	1.20	11	3.31±0.70 10.35±5.35	1.46	10	4.19±1.07 15.3±8.03	1.41	13	4.25±0.73 8.03±3.44	1.16
Plasma retinol (mg/100ml)	9	63.7±4.8 68.5±10.0	0.59	10	74.6±5.5 80.5±6.4	1.42	10	55.0±4.9 64.3±6.2	1.86	13	60.0±3.1 65.1±6.5	0.91
Plasma α -tocopherol (mg/l)	9	12.0±1.1 11.3±1.3	-0.68	10	13.9±1.5 12.3±1.0	-2.6*	10	9.9±1.2 9.8±0.7	-0.17	13	13.4±0.7 13.4±1.8	0.00
Plasma α -carotene (mg/100ml)	9	36.4±21.2 167.8±41.5	2.85*	10	23.5±12.1 81.0±14.3	4.49***	10	10.3±6.0 125.0±24.5	4.99***	12	5.0±3.6 136.7±58.9	2.38*
Plasma β -carotene (mg/100ml)	9	52.1±8.7 52.2±15.5	0.01	9	73.0±23.0 84.4±35.3	0.75	10	51.9±9.0 75.0±20.1	1.41	13	52.2±6.1 87.7±20.6	2.17*

* p < 0.05 ** p < 0.01 *** p < 0.005 † p < 0.001 †† p < 0.0005 ††† p < 0.0001

Table A2.10. Analysis of inter-individual variance of BMI (excluding diabetics)

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	4126.7	199					
Survey no. (ordinal)	4126.3	198	0.393	1	0.391	1.00	ns
Survey no. (factor)	4087.2	195	39.16	3	13.05	33.52	<0.001
Age	4085.3	194	1.868	1	1.868	4.80	<0.05
Sex	4073.3	193	11.99	1	11.99	30.80	<0.001
Serum triglyceride concentration	3999.6	192	73.68	1	73.68	188.0	<0.001
Kava	3678.1	191	321.6	1	321.6	817.9	<0.001
Alcohol	3580	190	98.0	1	98.0	250.0	<0.001
Cigarettes	3075.5	189	504.6	1	504.6	1,287	<0.001
Age*Triglyc.	3045.8	188	29.67	1	29.67	75.69	<0.001
Sex*Alcohol	3026.1	187	19.68	1	19.68	50.20	<0.001
Sex*Cigarettes	2955.4	186	70.72	1	70.72	180.4	<0.001
Age*Alcohol	2855.4	185	100.0	1	100.0	255.1	<0.001
Subject ID	48.93	125	2806	60	46.76	119.3	<0.001
residual	-	-	48.93	125	0.392		

Table A2.11. Analysis of inter-individual variance of diastolic blood pressure

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	19107	233					
Survey no. (ordinal)	17991	232	1116	1	1116	27.7	<0.001
Survey no. (factor)	17460	229	531	3	177	4.4	<0.01
Sex	16433	228	1027	1	1027	25.5	<0.001
Age	14617	227	1816	1	1816	45.7	<0.001
BMI	14330	226	287	1	287	7.12	<0.01
Sex*Age	13194	225	1136	1	1136	28.2	<0.001
Subject ID	6403.5	159	6790	66	102.9	2.56	<0.001
residual	-	-	6403.5	159	40.3		

Table A2.12. Analysis of inter-individual variance of systolic blood pressure

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	59481	233					
Survey no. (ordinal)	56560	232	2921	1	2921	45.3	< 0.001
Survey no. (factor)	55655	229	905.1	3	301.7	4.68	< 0.005
Sex	53852	228	1803	1	1803	28.0	< 0.001
Age	44720	227	9132	1	9132	141.7	< 0.001
BMI	41847	226	2873	1	2873	44.6	< 0.001
Alcohol	40949	225	897.7	1	897.7	13.93	< 0.005
Sex*Age	39226	224	1723	1	1723	26.7	< 0.001
Subject ID	10185	158	29041	66	440.0	6.83	< 0.01
residual	-	-	10185	158	64.46		

Table A2.13. Analysis of inter-individual variance of total serum cholesterol concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	340.51	237					
Survey no. (ordinal)	329.22	236	11.30	1	11.30	37.1	< 0.001
Survey no. (factor)	323.38	233	5.837	3	1.95	6.41	< 0.001
Age	313.64	232	9.739	1	9.739	32.0	< 0.001
Sex	305.42	231	8.222	1	8.222	27.02	< 0.001
BMI	290.68	230	14.74	1	14.74	48.44	< 0.001
HDL	279.80	229	10.87	1	10.87	35.72	< 0.001
Age*Sex	262.70	228	17.10	1	17.10	56.19	< 0.001
HDL*Age	255.07	226	4.42	1	4.42	14.53	< 0.001
Subject ID	47.78	157	207.3	69	3.00	9.86	< 0.005
residual	-	-	47.78	157	0.3043		

Table A2.14. Analysis of inter-individual variance of total serum cholesterol concentration (including fasting serum triglyceride concentration)

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	318.42	231					
Survey no. (ordinal)	308.11	230	10.32	1	10.32	35.5	<0.001
Survey no. (factor)	304.01	227	4.10	3	1.365	4.69	<0.005
Age	295.62	226	8.39	1	8.39	28.8	<0.001
Sex	290.51	225	5.11	1	5.11	17.6	<0.001
BMI	276.15	224	14.36	1	14.36	49.3	<0.001
HDL	263.99	223	12.16	1	12.16	41.8	<0.001
Serum triglyceride concentration	219.23	222	44.76	1	44.76	153.8	<0.001
Age*Sex	208.43	221	10.80	1	10.80	37.1	<0.001
BMI*Triglyc.	203.22	220	5.22	1	5.22	17.92	<0.001
HDL*Age	100.87	219	2.35	1	2.35	8.07	<0.01
Subject ID	43.96	151	156.9	68	2.31	7.94	<0.001
residual	-	-	43.96	151	0.291		

Table A2.15. Analysis of inter-individual variance of HDL-cholesterol concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	13.09	237					
Survey no. (ordinal)	12.86	236	0.232	1	0.232	9.13	<0.005
Survey no. (factor)	12.60	233	0.257	3	0.086	3.55	<0.05
Sex	12.58	232	0.024	1	0.024	0.99	ns
Age	12.57	231	0.006	1	0.006	0.23	ns
BMI	12.47	230	0.106	1	0.106	4.39	<0.05
Serum cholesterol concentration	12.00	229	0.466	1	0.466	19.3	<0.001
Sex*Age	11.82	228	0.186	1	0.186	7.72	<0.01
Sex*BMI	11.56	227	0.226	1	0.226	9.38	<0.005
Subject ID	3.81	158	7.776	69	0.113	4.67	<0.001
residual	-	-	3.81	158	0.024		

Table A2.16. Analysis of inter-individual variance of HDL-cholesterol concentration (including fasting serum triglyceride concentration)

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	12.57	231					
Survey no. (ordinal)	12.31	230	0.268	1	0.268	11.27	<0.001
Survey no. (factor)	12.03	227	0.275	3	0.092	3.87	<0.05
Sex	11.98	226	0.050	1	0.050	2.10	ns
Age	11.97	225	0.005	1	0.005	0.01	ns
BMI	11.92	224	0.065	1	0.065	2.73	ns
Serum cholesterol concentration	11.39	223	0.524	1	0.524	22.15	<0.001
Serum triglyceride concentration	10.63	222	0.759	1	0.759	32.06	<0.001
Sex*Age	10.52	221	0.116	1	0.116	4.88	<0.05
Sex*BMI	10.32	220	0.197	1	0.197	8.31	<0.01
Subject ID	3.60	152	6.72	68	0.099	4.18	<0.001
residual	-	-	3.60	152	0.024		

Table A2.17. Analysis of inter-individual variance of serum triglyceride concentration (excluding diabetics)

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	207.78	205					
Survey no. (ordinal)	206.44	204	1.339	1	1.339	2.05	ns
Survey no. (factor)	203.04	201	3.397	3	1.130	1.73	ns
Age	200.83	200	2.216	1	2.216	3.40	<0.05
Sex	200.83	199	0.001	1	0.001	0.02	ns
BMI	196.18	198	4.648	1	4.648	7.13	<0.01
Alcohol	195.51	197	0.673	1	0.673	1.03	ns
Cigarettes	184.42	196	11.09	1	11.09	17.0	<0.001
Sex*Alcohol	181.08	195	3.337	1	3.337	5.12	<0.05
Sex*Age	176.93	194	4.149	1	4.149	6.36	<0.05
Subject ID	86.08	132	90.85	62	1.47	2.25	
residual	-	-	86.08	132	0.652		

Table A2.18. Analysis of inter-individual variance of serum triglyceride concentration (including serum cholesterol concentrations; excluding diabetics)

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	207.78	205					
Survey no. (ordinal)	206.44	204	1.339	1	1.339	2.10	ns
Survey no. (factor)	203.04	201	3.397	3	1.130	1.77	ns
Sex	203.02	200	0.025	1	0.025	0.04	ns
BMI	198.20	199	4.818	1	4.818	7.56	<0.01
Serum cholesterol concentration	174.61	198	23.59	1	23.59	37.0	<0.001
HDL	159.99	197	14.62	1	14.62	22.95	<0.001
Alcohol	158.95	196	1.043	1	1.043	1.64	ns
Cigarettes	151.65	195	7.296	1	7.296	11.5	<0.001
Sex*Alcohol	146.3	194	5.379	1	5.379	19.9	<0.001
Subject ID	82.83	130	63.44	64	1.01	1.59	<0.05
residual	-	-	82.83	130	0.637		

Table A2.19. Analysis of inter-individual variance of two-hour plasma glucose concentration (excluding diabetics)

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	232.45	72					
Survey no. (ordinal)	229.48	70	2.97	1	2.97	2.04	ns
Age	220.51	70	8.97	1	8.97	6.14	<0.05
Sex	188.50	69	32.01	1	32.01	21.92	<0.001
BMI	174.53	68	13.97	1	13.97	9.57	<0.005
2 hr insulin	131.68	67	42.85	1	42.85	29.35	<0.001
Fasting glucose	114.00	66	17.68	1	17.68	12.11	<0.001
Subject ID	35.05	24	78.95	42	1.88	1.29	ns
residual	-	-	35.05	24	1.46		

Table A2.20. Analysis of inter-individual variance of serum γ gt concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	2544305	231					
Survey no. (ordinal)	2532651	230	11654	1	11654	21.3	<0.001
Survey no. (factor)	2503510	227	29142	3	9714	17.7	<0.001
Sex	2457918	226	45592	1	45592	83.2	<0.001
Age	2440704	225	17214	1	17214	31.4	<0.001
BMI	2383524	224	57180	1	57180	104.4	<0.001
Serum triglyceride concentration	2203521	223	180003	1	180003	320.0	<0.001
Kava	1911465	222	292057	1	292057	328.5	<0.001
Alcohol	1887625	221	23839	1	23839	43.5	<0.001
Cigarettes	1872289	220	15337	1	15337	28.0	<0.001
BMI*Kava	1749897	219	122392	1	122392	223.4	<0.001
BMI*Sex	1701801	218	48096	1	48096	87.8	<0.001
Kava*Sex	1662450	217	39351	1	39351	71.8	<0.001
Trig.*Alcohol	1655006	216	7444	1	7444	13.6	<0.001
Subject ID	80543	147	1574463	69	22818	41.6	<0.001
residual	-	-	80543	147	547.9		

Table A2.21. Analysis of inter-individual variance of red blood cell folate concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	638565	223					
Survey no. (ordinal)	387396	222	251169	1	251169	206.6	<0.001
Survey no. (Factor)	350071	219	37324	3	12441	10.2	<0.001
Sex	346079	218	3993	1	3993	3.28	ns
Cigarettes	325161	217	20918	1	20918	17.2	<0.005
Cigarettes*Sex	305990	216	19171	1	19171	15.8	<0.005
Subject ID	179921	148	124951	67	1864.9	1.53	<0.05
residual	-	-	179921	148	1215.7		

Table A2.22. Analysis of inter-individual variance of serum folate concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	376.07	196					
Survey no. (ordinal)	354.03	195	22.03	1	22.03	27.4	<0.001
Survey no. (factor)	341.04	193	12.99	2	6.50	8.1	<0.001
Sex	334.55	192	6.50	1	6.50	8.1	<0.005
Age	320.45	191	14.09	1	14.09	17.5	<0.001
Cigarettes	279.79	190	40.67	1	40.67	50.5	<0.001
Alcohol	260.39	189	19.40	1	19.40	24.1	<0.001
Cigarettes*sex	244.29	188	16.10	1	16.10	20.0	<0.001
Cigarettes*age	231.55	187	12.74	1	12.74	15.8	<0.001
Subject ID	97.5	121	134.1	66	2.03	2.52	<0.005
residual	-	-	97.5	121	0.81		

Table A2.23. Analysis of inter-individual variance of haemoglobin concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	36020	209					
Survey no. (ordinal)	36019	1	1.7	1	1.7	0.06	ns
Survey no. (factor)	35685	205	333.5	3	111.2	4.1	<0.01
Sex	24225	204	11460	1	11460	419.5	<0.001
Cigarettes	23012	203	1213	1	1213	44.4	<0.001
Alcohol	22643	202	369.1	1	369.1	13.5	<0.005
RBC Folate	20909	201	1734	1	1734	63.5	<0.001
Sex*cigarettes	20364	200	544.8	1	544.8	19.9	<0.001
Sex*Alcohol	19515	199	849.2	1	849.2	31.1	<0.001
RBCFol*Survey (ordinal)	18795	1	719.6	1	719.6	26.4	<0.001
RBCFol*Survey (factor)	17468	195	1328	3	442.7	16.2	<0.001
Subject ID	3497	128	13971	67	208.5	7.6	<0.001
residual	-	-	3497	128	27.3		

Table A2.24. Analysis of inter-individual variance of mean cell volume

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	3401.2	209					
Survey no. (ordinal)	3282.9	208	118.3	1	118.3	72.4	<0.001
Survey no. (factor)	3220.8	205	62.1	3	20.7	12.67	<0.001
Sex	3199.8	204	21.0	1	21.0	12.84	<0.001
Kava	2876.9	203	323.0	1	323.0	197.7	<0.001
Cigarettes	2867.8	202	9.11	1	9.11	5.57	<0.05
Alcohol	2865.4	201	2.33	1	2.33	1.42	ns
RBC folate	2857.2	200	8.23	1	8.23	5.04	<0.05
Sex*Cigarettes	2774.4	199	82.8	1	82.8	50.7	<0.001
Sex*Alcohol	2655.6	198	118.8	1	118.8	72.7	<0.001
Cigs*Alcohol	2619.3	197	36.3	1	36.3	22.2	<0.001
Sex*Kava	2552.0	196	67.3	1	67.3	41.2	<0.001
RBCFol.*Survey (ordinal)	2475.5	195	76.5	1	76.5	46.8	<0.001
RBCFol.*Survey (factor)	2415.0	192	60.5	3	20.2	12.3	<0.001
Subject ID	204.2	125	2211	67	33.0	20.2	<0.001
residual	-	-	204.2	125	1.63		

Table A2.25. Analysis of inter-individual variance of haematocrit

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	0.31508	209					
Survey no. (ordinal)	0.30590	208	0.00918	1	0.00918	38.25	<0.001
Survey no. (factor)	0.30091	205	0.00499	3	0.00167	6.96	<0.001
Sex	0.20236	204	0.09855	1	0.09855	410.6	<0.001
Cigarettes	0.19508	203	0.00728	1	0.00728	30.33	<0.001
Alcohol	0.19141	202	0.00368	1	0.00368	15.33	<0.001
Age	0.19108	201	0.00032	1	0.00032	1.34	ns
RBC Folate	0.17714	200	0.01395	1	0.01395	57.9	<0.001
Sex*Age	0.15662	199	0.02051	1	0.02051	85.13	<0.001
Sex*Cigarettes	0.15217	198	0.00445	1	0.00445	18.47	<0.001
Sex*Alcohol	0.14797	197	0.00420	1	0.00420	17.50	<0.001
Cigs*Alcohol	0.14580	196	0.00217	1	0.00217	9.04	<0.005
RBCFol.*Survey (ordinal)	0.13970	195	0.00610	1	0.00610	25.4	<0.001
RBCFol.*Survey (factor)	0.13283	192	0.00687	3	0.00229	9.54	<0.001
Subject ID	0.03060	127	0.10220	65	0.01572	65.5	<0.001
residual	-	-	0.03060	127	0.00024		

Table A2.26. Analysis of inter-individual variance of red cell distribution width

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	258.8	209					
Survey no. (ordinal)	252.5	208	6.37	1	6.37	17.4	<0.001
Survey no. (factor)	230.2	205	22.3	3	7.43	20.3	<0.001
Age	226.8	204	3.37	1	3.37	29.5	<0.001
Sex	226.2	203	0.63	1	0.63	1.71	ns
Cigarettes	221.2	202	4.95	1	4.95	13.5	<0.001
Alcohol	220.6	201	0.61	1	0.61	1.66	ns
Kava	220.4	200	0.28	1	0.28	0.76	ns
RBC Folate	215.2	199	5.11	1	5.11	13.9	<0.001
Cigarettes*Age	209.1	198	6.15	1	6.15	16.8	<0.001
Kava*Age	206.8	197	2.25	1	2.25	6.14	<0.01
Sex*Cigarettes	196.9	196	9.90	1	9.90	27.0	<0.001
Sex*Age	188.5	195	8.50	1	8.50	23.2	<0.001
Sex*Alcohol	185.7	194	2.74	1	2.74	7.47	<0.01
RBCFol.*Survey (ordinal)	185.4	193	0.27	1	0.27	0.74	ns
RBCFol.*Survey (factor)	181.6	191	3.83	3	1.28	3.50	<0.05
Subject ID	45.83	125	135.8	65	2.09	5.70	<0.001
residual	-	-	45.83	125	0.367		

Table A2.27. Analysis of inter-individual variance of serum ferritin concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	3124859	213					
Survey no. (ordinal)	3078231	212	46628	1	46628	21.4	<0.001
Survey no. (factor)	2981520	209	96711	3	32237	14.8	<0.001
Sex	2855125	208	126396	1	126396	57.9	<0.001
Age	244016	207	414964	1	414964	190.1	<0.001
BMI	2053721	206	386440	1	386440	177.0	<0.001
Cigarettes	2004638	205	49083	1	49083	22.48	<0.001
Kava	1995294	204	9344	1	9344	4.28	<0.05
Sex*BMI	1947810	203	47485	1	47485	21.75	<0.001
Sex*Cigarettes	1931987	202	15823	1	15823	7.25	<0.01
Sex*Kava	1912451	201	19536	1	19536	8.95	<0.005
Subject ID	292541	134	1619910	67	24178	11.07	<0.005
residual	-	-	292541	134	2183		

Table A2.28. Analysis of inter-individual variance of serum vitamin B₆ concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	123619	206					
Survey no. (ordinal)	111114	205	12505	1	12505	38.7	<0.001
Survey no. (factor)	104450	203	6664	2	3332	10.3	<0.001
Sex	102274	202	2176	1	2176	6.7	<0.01
Cigarettes	97283	201	4990	1	4990	15.4	<0.001
BMI	92418	200	4866	1	4866	15.1	<0.001
Cigarettes*Sex	87294	199	5124	1	5124	15.9	<0.001
Subject I.D.	41680	129	45614	70	651.6	2.0	<0.005
residual	-	-	41680	129	323.1		

Table A2.29. Analysis of inter-individual variance of serum vitamin B₁₂ concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	11140270	206					
Survey no. (ordinal)	11090252	205	50018	1	50018	1.77	ns
Survey no. (factor)	10364744	203	725508	2	362754	12.8	<0.001
Age	9752862	202	611882	1	611882	21.6	<0.001
Sex	9683125	201	69737	1	69737	2.46	ns
Alcohol	9189609	200	493516	1	493516	17.4	<0.001
Age*Sex	8746319	199	443290	1	443290	15.7	<0.001
Alcohol*Sex	8506640	198	239679	1	239679	8.46	<0.005
Subject ID	3681749	130	4824891	68	70954	2.51	<0.005
residual	-	-	3681749	130	28321		

Table A2.30. Analysis of inter-individual variance of serum thiamine concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	2639.5	206					
Survey no. (ordinal)	2455.5	205	184.0	1	184.0	34.5	<0.001
Survey no. (Factor)	1666.6	203	788.9	2	394.5	73.9	<0.001
BMI	1505.4	202	161.2	1	161.2	30.2	<0.001
Alcohol	1489.1	201	16.36	1	16.36	3.06	ns
BMI*Alcohol	1443.7	200	45.33	1	45.33	8.49	<0.005
Subject ID	688.4	129	755.3	71	10.6	1.99	<0.005
residual	-	-	688.4	129	5.34		

Table A2.31. Analysis of inter-individual variance of red blood cell thiamine concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	143567	205					
Survey no. (ordinal)	143368	204	199.4	1	199.4	0.81	ns
Survey no. (Factor)	68150	202	75218	2	37609	151.8	< 0.001
Sex	68115	201	34.3	1	34.3	0.138	ns
Cigarettes	67842	200	273.4	1	273.4	1.10	ns
Sex*Cigarettes	66609	199	1233	1	1233	4.98	< 0.05
Subject ID	31960	129	34649	70	495.0	2.0	< 0.01
residual	-	-	31960	129	247.8		

Table A2.32. Analysis of inter-individual variance of plasma ascorbic acid concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	2632168	158					
Survey no. (ordinal)	2512705	157	119463	1	119463	8.70	< 0.005
Survey no. (factor)	2494113	156	18592	1	18592	1.35	ns
BMI	2427440	155	66673	1	66673	4.83	< 0.05
Cigarettes	2253513	154	173927	1	173927	12.6	< 0.001
Cigarettes*BMI	2111542	153	141972	1	141972	10.29	< 0.005
Subject ID	1152866	84	958676	69	11413	0.83	ns
residual	-	-	1152866	84	13725		

Table A2.33. Analysis of inter-individual variance of plasma retinol concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	5279278	131					
Survey no. (ordinal)	5260208	130	19071	1	19071	1.32	ns
Survey no. (factor)	5213548	129	46660	1	46660	3.23	ns
Sex	5097183	128	116365	1	116365	8.05	<0.01
Age	5079289	127	17894	1	17894	1.24	ns
BMI	5029142	126	50148	1	50148	3.47	ns
Cigarettes	4986423	125	42719	1	42719	2.96	ns
Kava	4774971	124	211452	1	211452	14.63	<0.001
Sex*Cigarettes	4550879	123	224092	1	224092	15.50	<0.001
BMI*Cigarettes	4393049	122	157831	1	157831	10.92	<0.001
Age*Cigarettes	4248689	121	144360	1	144360	9.99	<0.005
Subject ID	924956	64	3323733	57	58311	4.03	<0.001
residual	-	-	924956	64	14452		

Table A2.34. Analysis of inter-individual variance of plasma α -tocopherol concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	1944.0	139					
Survey no. (ordinal)	1928.6	138	15.4	1	15.4	2.74	ns
Survey no. (factor)	1875.5	137	53.2	1	53.2	9.45	<0.005
Cholesterol	1257.2	136	618.3	1	618.3	109.9	<0.0001
BMI	1135.6	135	121.6	1	121.6	21.61	<0.001
Kava	1095.7	134	39.92	1	39.92	7.09	<0.05
Subject ID	416.5	74	679.1	60	11.3	2.01	<0.01
residual	-	-	416.5	74	5.63		

Table A2.35. Analysis of inter-individual variance of plasma β -carotene concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	932935	131					
Survey no. (ordinal)	909648	130	23287	1	23287	5.80	<0.05
Survey no. (factor)	858131	129	51517	1	51517	12.84	<0.005
Cigarettes	732527	128	125605	1	125605	31.30	<0.001
Alcohol	721968	127	10559	1	10559	2.63	ns
Cigs.*Alcohol	705321	126	16647	1	16647	4.15	<0.05
Subject ID	268900	67	436420	59	7396.9	1.84	<0.01
residual	-	-	268900	67	4013		

Table A2.36. Analysis of intra-individual variance of haemoglobin concentration

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	36020	209					
Subject ID	4011.4	141	32009	68	470.7	16.9	<0.001
RBC folate	3918.1	140	93.24	1	93.24	3.35	ns
Survey no. (ordinal)	3907.3	139	10.82	1	10.82	0.39	ns
Survey no. (factor)	3787.9	136	119.4	3	39.8	1.46	ns
RBCFol.*Survey (ordinal)	3657.3	135	130.6	1	130.6	4.69	<0.05
RBCFol.*Survey (factor)	3592.9	132	64.39	3	21.46	0.77	ns
residual	-	-	3787.9	136	27.85		

Table A2.37. Analysis of intra-individual variance of mean cell volume

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	3401.2	209					
Subject ID	390.95	141	3010	68	44.26	25.5	< 0.001
RBC folate	346.74	140	44.21	1	44.21	25.4	< 0.001
Kava (factor)	329.57	139	17.18	1	17.18	9.90	< 0.001
Survey no. (ordinal)	297.1	138	32.4	1	32.4	18.6	< 0.001
Survey no. (factor)	242.4	135	54.7	3	18.2	10.5	< 0.001
RBCFol.*Survey (ordinal)	242.3	134	0.17	1	0.17	0.10	ns
RBCFol.*Survey (factor)	227.9	131	14.4	3	4.80	2.76	< 0.05
residual	-	-	227.9	131	1.739		

Table A2.38. Analysis of intra-individual variance of haematocrit

Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	0.31508	209					
Subject ID	0.04252	141	0.27260	68	0.00400	17.02	< 0.001
RBC folate	0.04009	140	0.00242	1	0.00242	10.30	< 0.005
Survey no. (ordinal)	0.03733	139	0.00277	1	0.00277	11.78	< 0.001
Survey no. (factor)	0.03422	136	0.00311	3	0.00104	4.33	< 0.01
RBCFol.*Survey (ordinal)	0.03149	135	0.00272	1	0.00272	11.58	< 0.001
RBCFol.*Survey (factor)	0.031002	132	0.00049	3	0.00016	0.69	ns
residual	-	-	0.031002	132	0.00024		

Table A2.39. Analysis of intra-individual variance of red cell distribution width

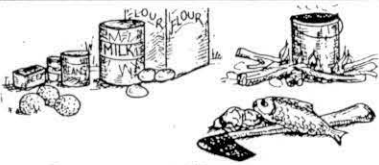














Variable	Deviance ss	Total df	Change ss	df	mean sq.	F	p
Fit	258.82	209					
Subject ID	65.11	141	193.7	68	2.85	<0.001	
RBC folate	63.02	140	2.10	1	2.10	<0.001	
Kava	61.19	139	1.83	1	1.83	<0.001	
Survey no. (ordinal)	61.06	138	0.13	1	0.13	ns	
Survey no. (factor)	48.35	135	12.7	3	4.23	<0.001	
residual	-	-	48.35	135	0.358		

ADDENDUM 3: Examples of written feedback of individual and community results, duty guidelines and outline of training programme for the Aboriginal research assistant, ALPA nutrition policy and summary of project budget.

A3.1. Examples of written feedback of results to the community and individuals at Minjilang

Results were fed back to the individuals and the community in a similar style every three months from June 1989 to June 1990.

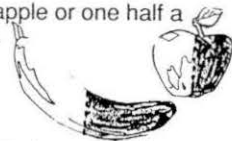
A3.1a. Community feedback booklet: Dietary intake June 1989

 <p style="text-align: center;">Community Nutrition Report. Minjilang. June 1989.</p> <p style="text-align: center;">Mandy Lee and Ann Bonson Menzies School of Health Research.</p>  <p style="text-align: left;">Page 1</p>	 <p>Some people at Minjilang are worried about the number of people getting sick with diabetes and heart trouble. In the old days, Aboriginal people didn't get diabetes or heart trouble - and people didn't get too fat. Change in diet and exercise can cause these problems. When people eat too much fat (from fatty meat, take-away foods and cooking oil) or too much sugar (from sugar and cool drinks) and too little fibre (from wholemeal bread and cereals and fruit and vegetables) they can get FAT and might get diabetes or heart disease. We need to know more about the types and amounts of food that people are eating so we can help people to stay healthy. Nutrition workers from the Menzies School of Health Research visited Minjilang and looked at the foods people buy. This book tells the result of this research.</p>  <p style="text-align: left;">Page 2</p>
<p>NUMBER OF DIFFERENT FOODS:</p> <p>Minjilang sells 86 different types of foods which is about the same as most other community stores in the Top End. This means that there is a variety of foods for people in Minjilang to choose.</p> <p>TYPES OF DIFFERENT FOODS:</p> <p>Compared with other places in Australia, people in Minjilang eat:</p> <ul style="list-style-type: none"> more sugar  more cool drinks  more snack foods  more meat pies  more cooking oil  more tinned meat  less fruit and less vegetables  <p style="text-align: left;">Page 3</p>	<p>FAT</p> <p>At Minjilang, people are eating too much fat.</p> <p>This fat comes from:</p> <ul style="list-style-type: none"> Fatty meats and chickens :  Too much fat on the meat. Potato chips and snacks:  1 packet everyday. Meat Pies:  1 pie every three days. Cooking oil:  Too much fried food. <p style="text-align: left;">Page 4</p> <p style="text-align: right;">(Continued over..)</p>

FRUIT AND VEGETABLES:

At Minjilang, people are eating :

Only one half an apple or one half a banana everyday.



Only one half a carrot or one half a potato everyday.



So people are not getting enough fibre or vitamins, like vitamin C and folic acid.

Page 5

SUGAR

At Minjilang, people are using nearly four times the amount of sugar recommended.

This sugar comes from:

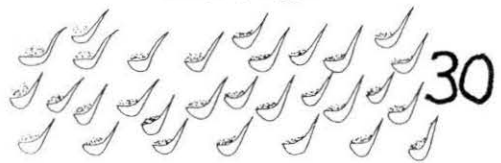
Sugar:
30 teaspoons everyday.



Cool drinks:
More than one can everyday.



Lollies:
Five lollies everyday.

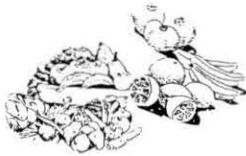


Page 6

This nutrition research tells us that to stay healthy in Minjilang it would help to:

EAT MORE:

- Fruit



-Vegetables



-Wholemeal bread and cereals
(like porridge and weetbix).



Page 7

It would also help to



EAT LESS:

- FAT

Cut the fat off meat.



Do not fry food.

Do not eat take-away foods (pies).



-SUGAR

Use less sugar.



Drink less cool drinks. (Try diet drinks or water instead).

Use bush foods whenever possible.



Page 8

**DIABETES
AND
HEART DISEASE:
MINJILANG
June 1989.**



Mandy Lee and Ann Bonson,
Menzies School of Health Research.

Page 1

WHAT THIS BOOK IS ABOUT:

This book is about Minjilang people and diabetes and heart trouble.

Some people at Minjilang are worried about people getting sick from diabetes and heart trouble.



Long time ago, Aboriginal people use to get a lot of exercise by hunting and gathering bush tucker. Aboriginal people didn't get diabetes or heart trouble.



Now people buy a lot of store foods. Now we are eating too much sugar and too much fat and getting a little bit of exercise and a little bit of bush tucker.

The Community asked Menzies School of Health Research to help people to stay healthy and to find out how many people had diabetes and could get heart trouble.

Page 2

At Minjilang we weighed everyone and also took some blood to see how fatty it was and to look at how much sugar was in the blood:



Too much sugar in the blood means a person has **DIABETES:**

Too much fat in the blood means a person could get **HEART PROBLEMS:**

This book tells Minjilang people what we found out and how we can stay healthy.

Page 3

WHY PEOPLE GET DIABETES AND HEART DISEASE:



People who get too little exercise and eat too much fat (from fatty store meat and margarine) or too much sugar (from sugar and cool drinks) and too little fibre (from wholemeal bread and cereals and fruit and vegetables) can get fat and might get diabetes or heart disease.

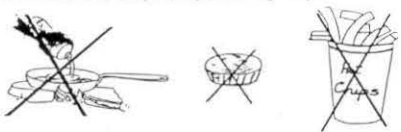


Page 4


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WHY PEOPLE GET FATTY BLOOD:


People get a lot of fat called **CHOLESTEROL** in their blood if they eat too much fat (from fatty store meat and fatty foods like pies, chips and margarine).



People get a lot of fat called **TRIGLYCERIDE** in their blood if they eat too much sugar (from sugar and cool drinks) and drinks too much grog.



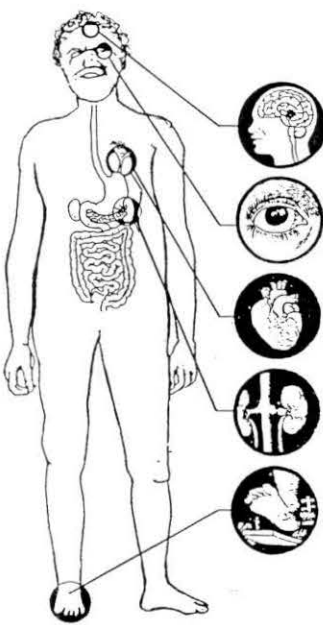
People who get fatty blood might get **HEART DISEASE**








Heart-Attack

Page 5

HEALTH PROBLEMS FOR DIABETICS



-  **Stroke**
-  **Blindness**
-  **Heart-Attack**
-  **Kidney-Disease**
-  **Foot-Problems**

Page 6

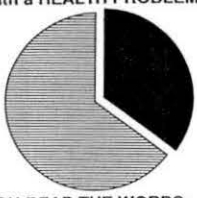
WHAT WE FOUND OUT AT MINJILANG:

- More older people than young people are **FAT**.
- More older people than young people have **DIABETES**.
- More women than men have **DIABETES**.
- More older people than young people have **FATTY BLOOD**.
- More men than women have **FATTY BLOOD**.

Page 7

HOW TO USE THIS BOOK:

The dark part of the circle shows the number of people with a **HEALTH PROBLEM**

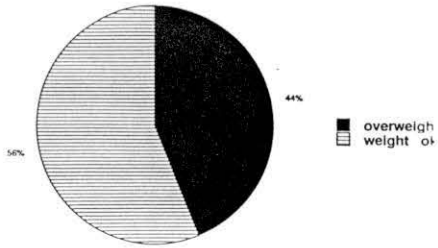


WHEN YOU READ THE WORDS:

None	It means	0%
Only some.....	It means	1% - 20%
Some.....	It means	21% - 40%
About half.....	It means	41% - 60%
A lot of.....	It means	61% - 70%
Most.....	It means	71% - 99%
All.....	It means	100%

Page 8 (Continued over..)

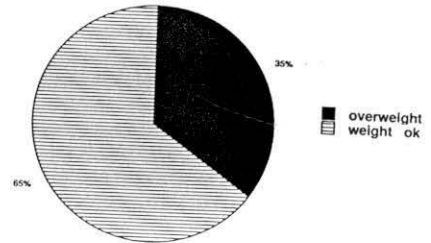
FAT YOUNG MEN:



About half of the young men are FAT.

Page 9

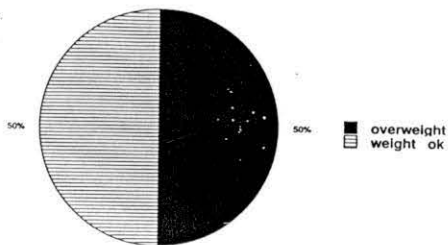
FAT OLDER MEN:



Some of the older men are FAT:

Page 10

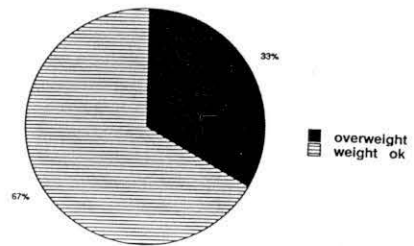
FAT YOUNG WOMEN:



About half the young women are FAT.

Page 11

FAT OLDER WOMEN:

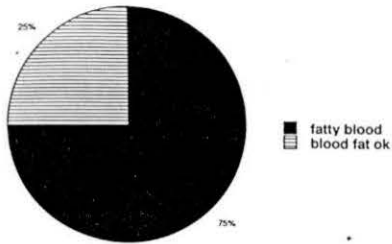


Some of the older women are FAT.

Page 12

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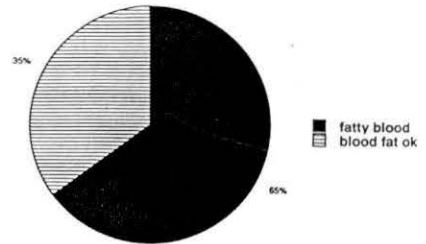
FATTY BLOOD IN YOUNG MEN:



Most of the young men have alot of fat called CHOLESTEROL in their blood.

Page 13

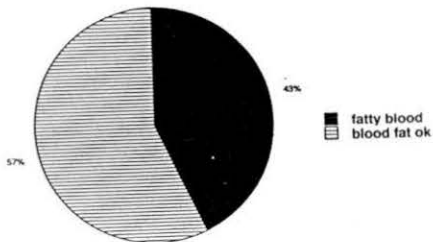
FATTY BLOOD IN OLDER MEN:



A lot of the older men have a lot of fat called CHOLESTEROL in their blood.

Page 14

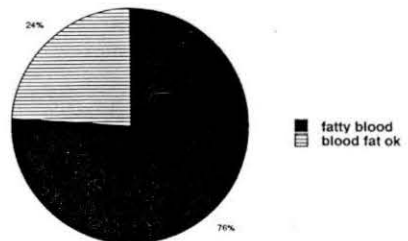
FATTY BLOOD IN YOUNG WOMEN:



About half of the young women have a lot of fat called CHOLESTEROL in their blood.

Page 15

FATTY BLOOD IN OLDER WOMEN:

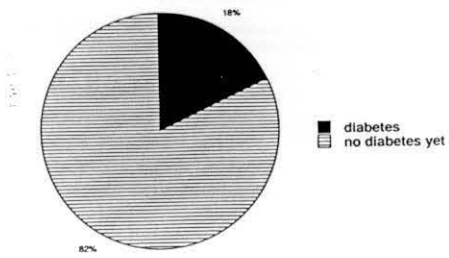


Most of the older women have a lot of fat called CHOLESTEROL in their blood.

Page 16

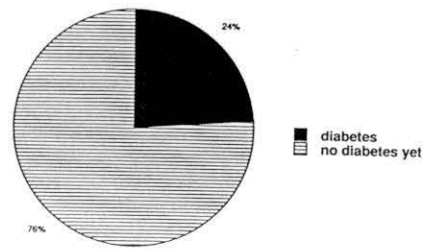
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DIABETES IN OLDER MEN:



Only some of the older men have a lot of sugar in their blood.

DIABETES IN OLDER WOMEN:



Some of the older women have a lot of sugar in their blood.

***** MENZIES SCHOOL OF HEALTH RESEARCH *****

***** COMMUNITY X FOOD AND HEALTH PROJECT *****

PERSON X

This book tells YOU about YOUR health in SEPTEMBER 1989.

CHOLESTEROL.

Cholesterol is a type of fat in the blood. Some people have too much cholesterol in the blood. Too much cholesterol can cause heart trouble.

In May the cholesterol in your blood was 5.5
This was a bit TOO much cholesterol.

Now the cholesterol in your blood is 5.1
This means that your cholesterol is getting better.

Now your cholesterol is OK.

To keep your cholesterol OK. EAT LESS FAT:

Cut all the fat off meat
Grill, bake or boil foods - do not fry foods
Try not to eat take away foods, like pies

Page 1

TRIGLYCERIDES.

Triglycerides are other types of fat in the blood. If people eat too much sugar or drink too much grog they can get fatty blood from triglycerides. Fatty blood can cause heart trouble.

In May the triglyceride in your blood was 2.7
This was TOO much triglyceride.

Now the triglyceride in your blood is 2.3
This means that your triglyceride is getting better.

But you still have TOO much triglyceride.

You CAN get rid of this triglyceride.

To get rid of triglyceride : EAT LESS SUGAR:
Do not use any sugar or jam or honey.
Use diet cool drinks only, like Diet Coke.
Do not drink grog.

DIABETES.

A person with diabetes has too much sugar in the blood. Too much sugar in the blood can cause trouble in the heart, kidneys, veins, skin, nerves and eyes.

You have got diabetes.

It is very important to look after your body so you do not have trouble with your diabetes.

Your diabetes is getting better.

TO CONTROL DIABETES:

Eat less fat, pies and fatty meat
Eat less sugar
Eat more fresh fruit and vegetables
Eat more wholemeal bread and cereals, like weetbix

Page 2

LIVER TROUBLE.

Some people can get liver trouble from drinking grog and kava.

In May you had liver trouble.
You do not have liver trouble anymore.

Do not drink too much grog or kava.

HEART PROTECTION MEASUREMENT.

We measured the HDL in your blood. This HDL can protect your heart.

In May your HDL was too low.

Your HDL is still too low.

Help protect your heart by walking and by other exercise like basketball, hunting, gardening, and dancing.

YOUR WEIGHT.

In May you were overweight.
You have lost weight.
Your weight is now good.

Look at the last page to see more about your weight and measurements. If you do not feel good about your weight, we can help you, and weigh you every week at the health centre.

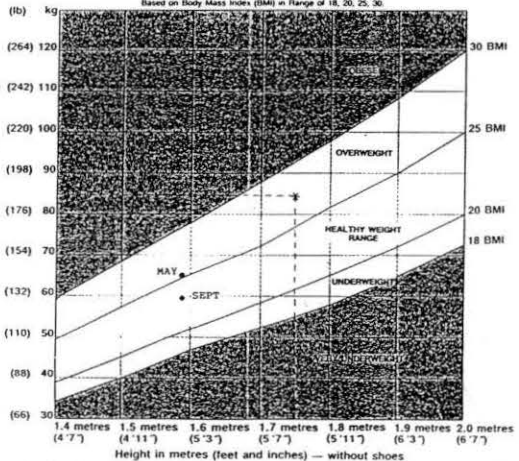
Page 3

WEIGHT FOR HEIGHT CHART
(For Men and Women from 18 years onward)



NAME: PERSON X I.D. No.: _____
COMMUNITY: _____ D.O.B.: ____/____/19__

Based on Body Mass Index (BMI) in Range of 18, 20, 25, 30



Reproduced with the permission of the Australian Nutrition Foundation Inc.

IN MAY: DATE: 25/5/19 89 HEIGHT: 158.5 cm WEIGHT: 64.1 kg WAIST: 89 cm HIP: 103 cm BMI: 25.3

IN SEPT: DATE: 4/9/19 89 HEIGHT: 158.5 cm WEIGHT: 59.6 kg WAIST: 80 cm HIP: 97 cm BMI: 23.7

Page 4

FOOD AND HEALTH

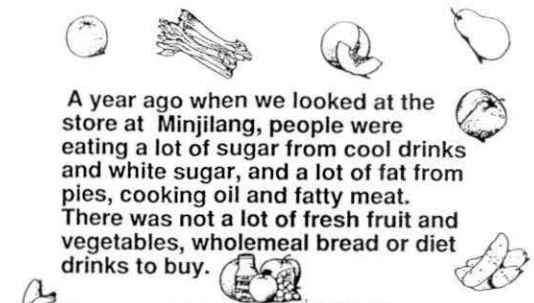
MINJILANG

JUNE 1989 - JUNE 1990



Mandy Lee & Ann Bonson
MENZIES SCHOOL OF HEALTH
RESEARCH

Page 1



A year ago when we looked at the store at Minjilang, people were eating a lot of sugar from cool drinks and white sugar, and a lot of fat from pies, cooking oil and fatty meat. There was not a lot of fresh fruit and vegetables, wholemeal bread or diet drinks to buy.

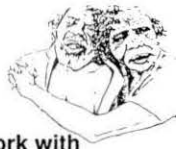
A year ago everybody at Minjilang was weighed, measured and had their blood checked.

Most people had too much cholesterol and too much triglyceride in their blood. Some people had diabetes. A lot of people had low folic acid levels which can make weak blood.

When people at Minjilang got their results back, everybody wanted to eat healthier food. The store got more fruit and vegetables, wholemeal bread and diet drinks.



Page 2



A year ago we began to work with people at Minjilang to help fight diabetes and heart trouble.

Long time ago people did not get diabetes or heart trouble. People used to get a lot of exercise and eat bush tucker to keep strong.

Now we are sitting down and eating store tucker.

Too much fat or sugar from store tucker can make people get fat and can also make too much fat and sugar in the blood.

Too much fat in the blood means a person could get HEART TROUBLE.

Too much sugar in the blood means a person has DIABETES.

Page 3

People tried to get more exercise, and some people stopped smoking.

Every three months people had another health check to see if they were getting better.



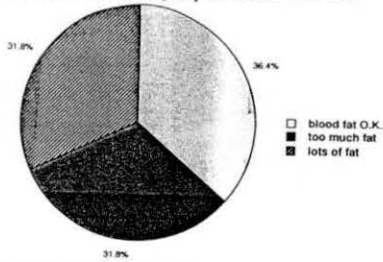
During the year the food in the store improved and people's health started to improve too!!



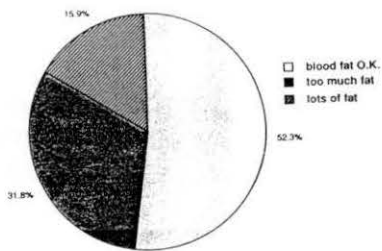
Page 4

(Continued over..)

Last year a lot of people had too much cholesterol (fat) in their blood.

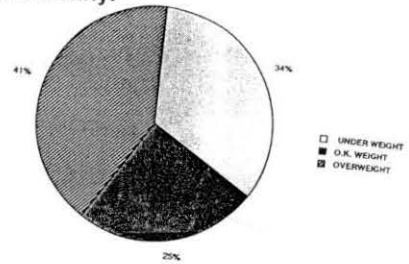


Now most people have less cholesterol (fat) in their blood.

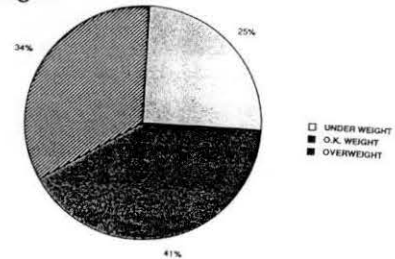


Page 5

Last year a lot of people were too fat or too skinny.

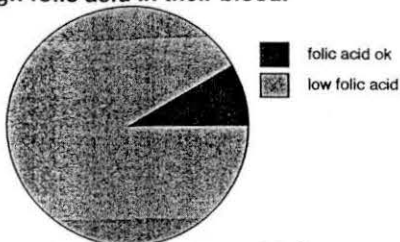


Now more people have a healthy weight.

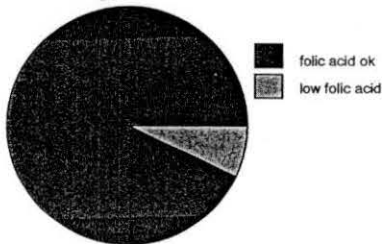


Page 6

Last year a lot of people did not have enough folic acid in their blood.



Now most people have lots of folic acid and strong blood.

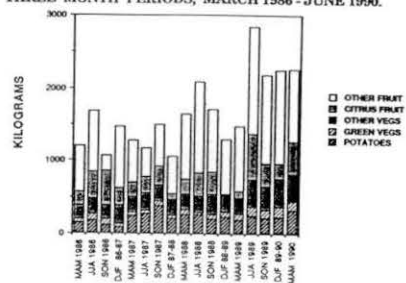


This folic acid comes from fresh food.

Page 7

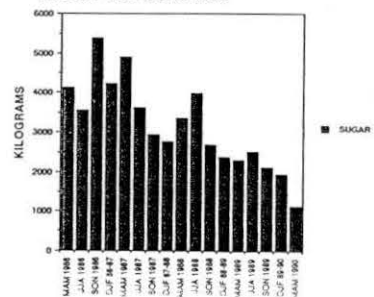
Now the store sells lots of fresh fruit and vegetables.

THREE MONTH PERIODS, MARCH 1986 - JUNE 1990.



But the store sells less sugar too.

MARCH 1986 - JUNE 1990.



Page 8

(Continued over..)

This research has shown that people can fight together against sicknesses like diabetes, weak blood and heart trouble.



Minjilang has shown the way for other communities to keep their people healthy and strong.



A3.2. Aboriginal Research Assistant (Nutrition and Public Health): Duty guidelines and outline of training program

Designation	Research Assistant: Nutrition and Public Health
Level	NTPS A3 (depending on experience)
Responsible to	Director of Menzies School of Health Research and his nominee
Hours	8.00 am to 4.21 pm
Period	One year

a: Duty Guidelines

1. To provide a cultural perspective on health and nutritional status of Aboriginal groups
2. To assist with field work and data collection in Aboriginal communities
 - 2.1 Explanation of research priorities
 - 2.2 Obtaining of informed consent
 - 2.3 Data collecting- interviewing
 - 2.4 Data collection- food and nutrient intake/anthropometric measurement
 - 2.5 Feedback of results in a meaningful way
3. To assist with entering data onto computer
4. To assist with preparation and development of audio visual materials
5. To assist with design of a community-based nutrition intervention program
6. To assist with the evaluation of this program
7. To assist with general clerical duties

b: Training components

1. Food and Nutrition
 - 1.1 Basic principles of nutrition and food composition
 - 1.2 Basic principles of hygiene
 - 1.3 Basic cooking skills
 - 1.4 Menu planning/budgeting skills for low income groups
 - 1.5 Applied nutrition: nutritional needs of special groups
 - 1.6 Applied nutrition: nutrition and growth
 - 1.7 Applied nutrition: non-communicable diseases
 - 1.8 Principles of anthropometric measurement
 - 1.9 Measurement of dietary intake
 - 1.10 Measurement of store-turnover
 - 1.11 Principles of nutrition education / adult education
 - 1.12 Community development and nutrition
2. Audio visual skills
 - 2.1 Use of equipment
 - 2.2 Making nutrition teaching aids
 - 2.3 Public speaking
3. Clerical skills
 - 3.1 Filing, photocopying
 - 3.2 Library systems and reference searches
4. Computing skills
 - 4.1 IBM compatible software: word processing, spreadsheets, dietary analysis
 - 4.2 Apple MacIntosh software: wordprocessing, graphical packages
5. Personal development
 - 5.1 Literacy and numeracy skills
 - 5.2 Co-operation in research team endeavours
 - 5.3 Technical skills as outlined above

A3.3. ALPA Nutrition Policy

In establishing a written policy we recognise the need for it to be dynamic and evolutionary. Some aspects of the policy will be greatly enhanced through proposed 'Nutrition Education'

ALPA will seek to present a balanced range of products to its customers giving increased emphasis to the nutritional worth of our product selection (see Healthy Store Food Guidelines produced by MSHR)

Under some circumstances we will only carry the product which proves to be the most beneficial for health. Examples being CANOLA OIL and POLYUNSATURATED MARGARINE.

We will have at all times appealing and plentiful supplies of fresh fruit and vegetables, diet soft drinks, 100% fruit juices and some wholemeal bread.

The need of diabetics should be acknowledged and we will carry artificial sweeteners and suitable dry biscuits in range. For special requirements we will assist where possible.

The take-away section in all stores will emphasise this nutritional bias to the best of our ability without restricting choice. All stores will introduce sandwiches for their nutritional value over pies and pasties.

Co-operation with government health workers on a nutrition program will be implicit. In conjunction with the proposed 'Nutrition Education' shelf talkers for health heart food will be displayed.

A3.4. Summary of 12 month Minjilang Project Budget

Salaries	\$ 48000
Equipment	\$ 4500
Maintenance	
Infrastructure support (MSHR)	\$ 3000
Materials and stationery	\$ 1000
Analysis of samples	\$ 6200
Travel and subsistence	\$ 2700
<hr/>	
TOTAL	\$ 65400

Addendum 4. Examples of traditional stories considering over-consumption of sugar and fat.

In specific regions, traditional stories may be used in culturally-relevant nutrition intervention projects.

- A4.1. *"This magic man, Nagamara, travelled right through Cape York Peninsula, right through to western Arnhem Land.. right up to Coburg Peninsula.. That's where he travelled. He had met a lot of people. They came to the place where he was camped. This was near Port Essington, or somewhere there in Yiwadja country. One day these people went out hunting, and there was with them a young man. He was very strong, very active. He got a lot of 'sugarbag', wild honey. And he ate and he ate till he was really full. So he said, "I'll cross this creek." He walked over with a lot of his friends. Everybody crossed the creek and he came after, and as he crossed the creek he got this pain. "What's happened?" they asked. 'I think, well I must have - I've eaten too much wild honey.' But he kept on walking till they came to a camp. They all settled down, but he was sick that night. He was in pain. They said to him, 'We can't do anything.' They tried their best to make him better and make him strong. But they couldn't do anything. He got worse and worse, until he died."* (Lamilami, 1974:17).
- A4.2. *Another of Lamilami's stories concerns a 'stranger' from Oenpelli, who, on a 'holiday' at North Goulburn Island, wanted to eat a lot of turtle gravy and meat. "So we said to this man, 'We're just warning you because we come from the saltwater side. We are coastal people, we know everything about turtles. We know you will be sick if you eat too much.' Anyway, he was annoyed and he wouldn't take any notice. So, he ate just the same as the others and that night he was very sick. He got a bit better but he was still sick.."* (Lamilami, 1974:221).
- A4.3. *A Gunwinggu myth relates that "Aidjilad was killed by a huge wild Dog, Mulwar by name, that died shortly after eating her because its belly was distended with fat and 'fat oozed from its nostrils'" (Berndt and Berndt, 1970:45).*
- A4.4. *"Two Yiwadja brothers who caught and cooked a large fish, found 'it had too much fat, it made their hearts bad', so they threw most of the fat away uneaten" (Berndt and Berndt, 1970:45).*
- A4.4. *One belief pertaining to over-consumption in desert regions is that "When a person eats too much meat, breath is blocked, meat is blocked in the intestines" (Wiminydji and Peile, 1978).*

BIBLIOGRAPHY

- Abbie, A.A. (1960), Physical changes in Australian Aborigines consequent upon European contact, *Oceania* 31:140-144.
- Abbie, A.A. (1966), The health of the Australian Aborigines, *Organorama* 3:13-16.
- Abbie, A.A. (1968), *Studies in physical anthropology, Volume 1*, AIAS, Canberra.
- Abbie, A.A. (1974), Physical Standards of nomadic Aboriginal children, *Medical Journal of Australia* 1:470-471.
- Abbie, A.A. (1975), *Studies in physical anthropology, Volume 2*, AIAS, Canberra.
- Abbie, A.A. and Schroder, J. (1960), Blood Pressures in Arnhem Land Aborigines, *Medical Journal of Australia* 2:493-496.
- Abramson, J.H., Slome, C. and Kosovsky, C. (1963), Food frequency interview as an epidemiological tool, *American Journal of Public Health* 53:1093-1101.
- Acheson, E.D. and Doll, R. (1964), Dietary factors in carcinoma of the stomach: a study of 100 cases and 200 controls, *Gut* 5:126-131.
- Acheson, E.D., Campbell, I.T., Edholm, O.G., Miller, D.S. and Stock, M.J. (1980), The measurement of food and energy intake in man- an evaluation of some techniques, *American Journal of Clinical Nutrition* 33:1147-1154.
- Akerman, K. (1975), Altered food taboos: Effects on the geriatric population of south-west Kimberley, *Oceania* 46:122-125.
- Albrecht, P.G. (1974). The social psychological reasons for the alcohol problems among Aborigines. In: Hetzel, B.S. et al (eds), *Better health for Aborigines?*, pp 36-41, UQP, St. Lucia.
- Alexander, K. (1985), *A study of kava in Arnhem Land Aboriginal communities*, ANU NARU, Monograph, Darwin.
- Allen, J.R., Heywood, P.F., Mensch, M.G., Saxelby, C., Ward, J.A. and Zed, C.A. (1977), Dietary methods used in an ascorbic acid supplementation trial in Australian Aboriginal and Caucasian school children, *Food and Nutrition Notes and Reviews* 34:12-17.
- Altman, J.C. (1980), The Aboriginal economy. In: *Northern Australia: Options and Implications*, Jones, R. (ed), pp 87-108, Research School of Pacific Studies, ANU, Canberra.
- Altman, J.C. (1982), *Hunter-gatherers and the State: the economic anthropology of the Gunwinggu of North Australia*, Unpublished PhD thesis, ANU, Canberra.
- Altman, J.C. (1987), *Hunter-gatherers today: an Aboriginal economy in north Australia*, AIAS, Canberra.
- Anderson, P.J. (1976), *Report on research into Aboriginal income distribution and expenditure patterns at Yuendumu, NT*, (Typescript), AIAS, Canberra.
- Australia, (1979), *Aboriginal Health*, House of Representatives Standing Committee on Aboriginal Affairs, AGPS, Canberra.
- Australia, (1988), *Health for All Australians*, Report of the Health Targets and Implementation (Health For All), Committee to the Australian Health Ministers Advisory Council and the Australian Health Ministers' Conference, AGPS, Canberra.
- Australia, (1989), *Composition of foods Australia*, AGPS, Canberra.
- ABS, (1979), *Apparent consumption of foodstuffs and nutrients, Australia 1975-76 and 1976-77*, ABS, Canberra.
- ABS, (1984), *Apparent consumption of foodstuffs and nutrients, Australia 1982-83*, ABS, Canberra.
- ABS, (1986a), *A guide to the consumer price index*, AGPS, Canberra.
- ABS, (1986b), *Census of population and housing, Northern Territory (Census 1986)*, AGPS, Canberra.
- ABS, (1987a), *Apparent Consumption of Food and Nutrients, Australia, 1985-86*, AGPS, Canberra.
- ABS, (1987b), *Age and sex of persons in statistical local areas and statistical divisions, Northern Territory (Census 1986)*, AGPS, Canberra.
- ABS, (1990), *Aboriginal people in the Northern Territory*, Government Printer of the NT, Darwin.
- Baghurst, K.I. and Baghurst, P.A. (1981), The measurement of usual dietary intake in individuals and groups, *Transactions of the Menzies Foundation*, 3:139-160, The Menzies Foundation, Melbourne.
- Baghurst, K.I. and Baghurst, P.A. (1989), The uses of food composition data in nutritional epidemiology, *Food Technology in Australia (Special Supplement)* 42:S11-S14.
- Baghurst, K.I. and Record, S.J. (1984), A computerised dietary analysis system for use with diaries or food frequency questionnaires, *Community Health Studies* 8:11-18.
- Bailey, L.B. and Cerda, J.J. (1988), Iron and folate nutriture during the life cycle, *World Review of Nutrition and Dietetics*, 56:65-92.
- Bain, M. S. (1974). Alcohol use and traditional social control in Aboriginal societies. In: Hetzel, B.S. et al (eds), *Better health for Aborigines?*, pp 42-52, UQP, St. Lucia.
- Baird, P.C. and Schutz, H.G. (1980), Lifestyle correlates of dietary and biochemical measures of nutrition, *Journal of the American Dietetic Association*, 76:228-235.
- Barnes, J.A. (1963), Social organisation: limits of contemporary studies, In: Sheils, H. (ed), *Australian Aboriginal studies: A symposium of papers presented at the 1961 research conference*, AIAS, Oxford University Press, Melbourne.
- Barr, A. (1989), Senior Pharmacist, NTDHCS, (Personal communication).

- Barrett, M.J. and Brown, T. (1971), Increase in average height of Australian Aborigines, *Medical Journal of Australia*, 2:1169-1172.
- Bartko, J.J. (1966), Interclass correlation coefficient as a measure of reliability, *Psychological Reports*, 19:3-11.
- Barwick, D. (1971), Changes in the Aboriginal population of Victoria 1863-1966, In: Mulvaney, D.J. and Golson, J. (eds), *Aboriginal man and environment in Australia*, pp. 288-315, ANU Press, Canberra.
- Basedow, H. (1904), Anthropological notes made on the South Australian government north-west prospecting expedition, 1903, *Transactions of the Royal Society of South Australia* 28:12-51.
- Basedow, H. (1913), Notes on the natives of Bathurst Island, North Australia, *Journal of the Royal Anthropological Institute* 43:291-323.
- Basedow, H. (1925), *The Australian Aboriginal*, F.W. Preece and Sons, Adelaide.
- Basedow, H. (1932), Diseases of the Australian Aborigines, *Journal of Tropical Medicine and Hygiene* 35:177-185.
- Bastian, P. (1979), Coronary heart disease in tribal Aborigines - The west Kimberley Survey, *Australian and New Zealand Journal of Medicine*, 9:284-292.
- Bauer, F.H. (1964), *Historical geography of White settlement in part of Northern Australia; Part 2, The Katherine-Darwin region*, CSIRO, Division of Land Research and Regional Survey, Divisional Report No. 64.1, AGPS, Canberra.
- Bazzare, T.L. and Myers, M.P. (1980), The collection of food intake data in cancer epidemiological studies, *Nutrition and Cancer* 1:22-45.
- Beaton, G.H., Milner, J., Corey, P., McGuire, V., Cousins, M., Stewart, E., de Ramos, M., Hewitt, D., Grambsch, P.V., Kassim, N. and Little, J.A. (1979), Sources of variance in 24-hour dietary recall data: implications for nutrition study design and interpretation, *American Journal of Clinical Nutrition* 32:2546-2559.
- Beaton, G.H., Milner, J., McGuire, V., Feather, T.E. and Little, J.A. (1983), Sources of variance in 24-hour dietary recall data: implications for nutrition study design and interpretation, Carbohydrate sources, vitamins and minerals, *American Journal of Clinical Nutrition* 37:986-995.
- Bell, D. (1983), *Daughters of the Dreaming*, McPhee Gribble, George Allen and Unwin.
- Berndt, R.M. and Berndt, C.H. (1942), Acculturation and native policy, *Oceania* 13: 52-70.
- Berndt, R.M. and Berndt, C.H. (1970), *Man, Land and Myth in North Australia- the Gunwinggu people*, Ure Smith, Sydney.
- Berndt, R.M. and Berndt, C.H. (1977), *The World of the first Australians, Aboriginal traditional life: Past and present*, (2nd edn), Ure Smith, Sydney.
- Beynen, A.C. (1982), Biochemical indicators which reflect the intake of (the type of), fats, In: Hautvast, G.A.J. and Klaver, W. (eds), *A concerted action project on nutrition in the European community: The diet factor in epidemiological research*, EURONUT Report 1, pp. 136-139, Wageningen.
- Beynen, A.C., Hermus, R.J. and Hautvast, J.G.A.J. (1980), A mathematical relationship between the fatty acid composition of the diet and that of the adipose tissue in man, *American Journal of Clinical Nutrition* 33:81-85.
- Billington, B.P. (1960), The health and nutrition status of the Aborigines, In: Mountford, C.P. (ed), *Records of the American-Australian Scientific Expedition to Arnhem Land, Volume 2, Anthropology and Nutrition*, pp. 27-54, MUP, Melbourne.
- Bingham, S. (1982), Recent developments in dietary methodology, In: Hautvast, G.A.J. and Klaver, W. (eds), *A concerted action project on nutrition in the European community: The diet factor in epidemiological research*, EURONUT Report 1, pp 106-22, Wageningen.
- Bingham, S. (1983), Biochemical markers of consumption, In: *The dietary assessment of populations*, Scientific Report No.4, pp 26-31, MRC, Environmental Epidemiology Unit, Southampton.
- Bingham, S. (1987), The dietary assessment of individuals; methods, accuracy, new techniques and recommendations, *Nutrition Abstracts and Reviews* 57:705-742.
- Birdsell, J. B. (1953), Some environmental and cultural factors influencing the structuring of Australian Aboriginal populations, *American Naturalist*, 87:171-207.
- Black, A. E. (1981), Pitfalls in dietary assessments. In: Howard, A.N. and McLean Baird, I. (eds), *Recent Advances in Clinical nutrition*, John Libbey, London.
- Black, E.C. and Cleland, J.B. (1938), Pathological lesions in Australian Aborigines, Central Australia (Granites), and the Flinders Ranges. *Journal of Tropical Medicine and Hygiene* 41:69-83.
- Blainey, G. (1982), *Triumph of the Nomads: A History of Ancient Australia*, Revised Edition, Sun Books, Macmillan, South Melbourne.
- Bland, J.M. and Altman, D.G. (1986), Statistical methods for assessing agreement between two methods of clinical measurement, *Lancet*, February 8:307-310.
- Blank, D.W., Hoeg, J.M., Kroll, M.H. and Ruddel, M.E. (1986), The method of determination must be considered in interpreting blood cholesterol levels, *Journal of the American Medical Association* 256:2867-2870.
- Bleakley, W. (1961), *Aborigines of Australia*, Jacaranda Press, Brisbane.
- Block, G. (1982), A review of validations of dietary assessment methods, *American Journal of Epidemiology* 115:492-505.

- Bonjour, J.P. (1980), Vitamins and alcoholism, II Folate and vitamin B₁₂, *International Journal of Vitamin and Nutrition Research* 50:96.
- Bourne, G.H. (1953), Foods of the Australian Aboriginal, *Proceedings of the Nutrition Society of Australia* 12:58-61.
- Boyden, A.N. and Agar, N.S. (1981), Letter: Plasma cholesterol levels in Australian Aborigines, *Medical Journal of Australia* 1:196.
- Boyes, J., Dixon, B., Lion, R., Clarke, D., Rae, C., and Puruntatameri, A. (1991). Diabetes story project, Paper presented at the 23rd. Annual Conference of the Public Health Association of Australia *The health of indigenous peoples*, Alice Springs.
- Brady, M. (1986), Leaving the spinifex: The impact of rations, missions and the atomic tests on the southern Pitjantjatjara, *Records of the South Australian Museum* 20:35-45.
- Brady, M. and Morice, R. (1982), Defiance or despair? Petrol sniffing in an Aboriginal Community. In: Reid, J. (ed), *Body, land and spirit: Health and healing in traditional Aboriginal society*, pp 72-88, UQP, St.Lucia.
- Brady, M. and Palmer, K. (1984). *Alcohol in the outback: two studies of drinking*. ANU. NARU, Darwin.
- Brand, J.C., Miller, J.J., Refshauge, A.J., Razzaby, L. and Kalokerinos, A. (1978), Improvement in growth rates of Sydney Aboriginal children, *Proceedings of the Nutrition Society of Australia* 3:92.
- Brand, J.C., Chirikoff, V., Lee, A. and McDonnell, J. (1982a), Nutrients in important bushfoods, *Proceedings of the Nutrition Society of Australia* 7:50-54.
- Brand, J.C., Chirikoff, V., Lee, A. and Truswell, A.S. (1982b), An outstanding food source of vitamin C, *Lancet* 2:873-875.
- Brand, J.C., Rae, C., McDonnell, J., Lee, A., Chirikoff, V. and Truswell, A.S. (1983), The nutritional composition of Australian Aboriginal bushfoods, *Food Technology in Australia* 37:275-279.
- Brand, J.C. and Chirikoff, V. (1985), Australian Aboriginal bushfoods: the nutritional composition of plants in the desert region, *Australian Aboriginal Studies* 2:38-46.
- Brand, J.C., Chirikoff, V. and Truswell, A.S. (1985), The nutritional composition of Australian Aboriginal bushfoods, 3. Seeds and nuts, *Food Technology in Australia* 35:293-98.
- Breinl, A. (1912), Report on health and disease in the Northern Territory, *Bulletin of the NT of Australia* 1:32-55.
- Breinl, A. and Holmes, M.J. (1915), Medical report on the data collected during a journey through some districts of the Northern Territory, *Bulletin of the NT of Australia* No.15.
- Brin, M. (1976), Recent information on thiamine nutritional status in selected countries, In: Gubler, C., Fujiwara, M., Dreyfus, P.M. (eds), *Thiamine*, Chapter 10, John Wiley and Sons, New York.
- British Medical Journal, (1981), Another look at zinc, *British Medical Journal* 282:1098-1099.
- Brock, J. (1988), *Top End Native Plants*, J.Brock, Darwin.
- Brokensha, P. (1975), *The Pitjantjatjara and their crafts*, Aboriginal Arts Board, Australia Council, Sydney.
- Brokensha, P. and McGuigan, C. (1977), Listen to the Dreaming: the Aboriginal homelands movement, *Australian Natural History* 19:118-123.
- Brown, A.J., Chirikoff, V. and Roberts, D.C.K. (1987), Fatty acid composition of seeds from the Australian Acacia species, *Lipids* 22:490-494.
- Brown, K.H., Black, R.E. and Becker, S. (1982), Seasonal changes in nutritional status and the prevalence of malnutrition in a longitudinal study of children in rural Bangladesh, *American Journal of Clinical Nutrition* 36:303-313.
- Brown, T. and Barrett, M.J. (1972), Growth in Central Australian Aborigines: weight, *Medical Journal of Australia* 2:999-1002.
- Brown, T. and Barrett, M.J. (1973), Increase in average weight of Australian Aborigines, *Medical Journal of Australia* 2:25-27.
- Brubacher, G. (1982), Biochemical indicators which reflect the intake of vitamins. In: Hautvast, G.A.J. and Klaver, W. (eds), *The diet factor in epidemiological research*, EURONUT Report 1, pp 125-135. Wageningen.
- Bryce, S. (1986), *Woman's gathering and hunting in the Pitjantjatjara homelands*, IAD, Alice Springs.
- Burke, B.S. (1947), The diet history as a tool in research, *Journal of the American Dietetic Association* 23:1041-1046.
- Butler, W.J., Ostrander, L.D., Carman, W.J. et al (1985), Mortality from coronary heart disease in the Tecumseh Study: Long term effects of diabetes mellitus, glucose tolerance and other risk factors, *American Journal of Epidemiology* 121(4): 541-547.
- Calaby, J.H. (1971), Man, fauna and climate in Aboriginal Australia. In: Mulvaney, D.J. and Golson, J. (eds), *Aboriginal man and environment in Australia*, pp. 80-93, ANU Press, Canberra.
- Calvert, D. (1981), Laboratory assessment of plasma lipids and lipoproteins, *Transactions of the Menzies Foundation* 3:95-111, The Menzies Foundation, Melbourne.
- Cameron, W.I., Moffitt, P.S., and Williams, D.D.R. (1986), Diabetes mellitus in Australian Aborigines of Bourke, New South Wales, *Diabetic Research and Clinical Practice*, 2:307-314.
- Campbell, A.H. (1965), Elementary food production by the Australian Aborigines, *Mankind* 6:206-11, 6:288.
- Campbell, T.D. (1939a), Food, food values and food habits of the Australian Aborigines in relation to their

- dental conditions: Introduction, *Australian Journal of Dentistry* 43:1-15.
- Campbell, T.D. (1939b), Food, food values and food habits of the Australian Aborigines in relation to their dental conditions: Habits of food preparation and consumption, *Australian Journal of Dentistry* 43:45-55.
- Campbell, T.D. (1939c), Food, food values and food habits of the Australian Aborigines in relation to their dental conditions: Essential food factors, *Australian Journal of Dentistry* 43:73-87.
- Campbell, T.D. (1939d), Food, food values and food habits of the Australian Aborigines in relation to their dental conditions: Jaws and dentition, *Australian Journal of Dentistry* 43:141-156.
- Campbell, T.D. (1939e), Food, food values and food habits of the Australian Aborigines in relation to their dental conditions: Dental caries, *Australian Journal of Dentistry* 43:177-198.
- Campbell, T.D. (1939f), Dental observations of Aborigines compared with civilised groups, *Mankind* 2:228-229.
- Campbell, T.D. and Barrett, M.J. (1953), Dental observations on Australian Aborigines: a changing environment and food pattern, *Australian Journal of Dentistry* 57:1-6.
- Campbell, T.D. and Lewis, A.J. (1926), The Aborigines of South Australia: dental observations recorded at Ooldea, *Australian Journal of Dentistry* 30:371-376.
- Cane, S. and Stanley, O. (1985), *Land use and resources in desert homelands*, NARU, ANU, Canberra.
- Carnegie, D.W. (1898), *Spinifex and Sand*, C. Arthur Pearson, London.
- Carrol, P.J. (1979), *Aboriginal diet- Alligator Rivers region*, Unpublished mimeograph, Office of the Supervising Scientist, Ranger Uranium Mine.
- Cashell, K. (1981), Apparent consumption of foodstuffs and nutrients in Australia, *Transactions of the Menzies Foundation* 3:203-220, The Menzies Foundation, Melbourne.
- Casley-Smith, J.R. (1958), The haematology of the central Australian Aborigines, Part 1, *Australian Journal of Experimental Biology and Medical Science* 36:23-38.
- Casley-Smith, J.R. (1959a), The haematology of the central Australian Aborigines, Part 2, *Australian Journal of Experimental Biology and Medical Science* 37:481-488.
- Casley-Smith, J.R. (1959b), Blood pressures in Australian Aborigines, *Medical Journal of Australia* 1:627-633.
- Casley-Smith, J.R. (1960), The haematology of the central Australian Aborigines, Part 4: Haemoglobin, erythrocyte counts, constants, fragilities and sedimentation rates; Effects of urbanisation, *Australian Journal of Experimental Biology and Medical Science* 38:37-46.
- Castelli, W.P. (1984), Epidemiology of coronary heart disease: The Framingham Study, *American Journal of Medicine* 76: 4-12.
- Castelli, W.P., Garrison, R.J., Wilson, P.F.W., Abbott, R.D., Kalousdian, S. and Kannel, W.B. (1986), Incidence of Coronary Heart Disease and Lipoprotein Cholesterol Levels; The Framingham Study, *Journal of the American Medical Association* 256: 2835-2838.
- Cawte, J. (1973), Social medicine in central Australia- The opportunities of Pitjantjara Aborigines, *Medical Journal of Australia* 1:221-233.
- Cawte, J. and Mununggurr (1977), Aboriginal counsellors talk on alcohol, *Medical Journal of Australia* 2: 723-725.
- CDH, (1982), *Dietary guidelines for Australians*, AGPS, Canberra.
- CDCSH, (1986), *National dietary survey of adults, 1983, No.1: Foods consumed*, AGPS, Canberra.
- CDCSH, (1987a), *National dietary survey of adults, 1983. No. 2: Nutrient Intakes*, AGPS, Canberra.
- CDCSH, (1987b), *Towards better nutrition for Australians*, Report of the Nutrition Task Force of the Better Health Commission, AGPS, Canberra.
- CDCSH, (1988), *Health for All Australians*, Report to the Australian Health Ministers Advisory Council and the Australian Health Ministers' Conference, by the Health Targets and Implementation (Health For All), Committee, AGPS, Canberra.
- Chandra, R.K. (1980), *Immunology of nutritional disorders*, Edward Arnold, London.
- Chandra, R.K. (1983), Nutrition, immunity and infection: present knowledge and future directions, *Lancet* March 26: 688-691.
- Charnock, J.S., Casley-Smith, J., and Schwartz, C.J. (1959), Serum magnesium-cholesterol relationships in the central Australian Aborigine and in Europeans with and without ischaemic heart disease, *Australian Journal of Experimental Biology and Medical Science* 37:509-516.
- Chaseling, W.S. (1957), *Yulengor: nomads of Arnhem Land*, Epworth Press, London.
- Cheek, D.B., Spargo, R.M. and Holt, A.B. (1981), Evidence for zinc deficiency in Aboriginal settlements in North Western Australia, *Medical Journal of Australia (Special Supplement)* 1:4-5.
- Cheek, D.B., Spargo, R.M. and Holt, A.B. (1982), Hair zinc and copper and retarded growth in Aboriginal children of North Western Australia, *Australian Paediatric Journal* 18:139-140.
- Cheek, D.B., McIntosh, G.H., O'Brian, V., Ness, D. and Green, R.C. (1989), Malnutrition in Aboriginal children at Yalata, South Australia, *European Journal of Clinical Nutrition* 43:161-168.
- Chen, T.S., Lui, C.K.F., and Smith, C.H. (1983), Folic acid content of tea, *Journal of the American Dietetic Association* 82:627-632.
- Cherikoff, V., Brand, J.C. and Truswell, A.S. (1985), The nutritional composition of Australian Aboriginal bushfoods, 2: Animal foods, *Food Technology in Australia* 37:208-11.
- Chippendale, G.M. (1963), Pasture degeneration in Central Australia, *Australian Journal of Agricultural*

- Cleland, J.B. (1928), Disease amongst the Australian Aborigines, *Journal of Tropical Medicine and Hygiene* 31:53-59, 65-70, 141-145, 157-160, 173-177, 196-198, 202-206, 216-220, 232-235, 262-266, 281-282, 290-294, 307-313, 326-330.
- Cleland, J.B. (1929), Scurvy. *Medical Journal of Australia* 2:867.
- Cleland, J.B. (1949), The Australian Aboriginal: The significance of his past, the present, his future, *Reports of the ANZAAS* 28:66-78.
- Cleland, J.B. (1957), Our natives and the vegetation of southern Australia, *Mankind* 5:149-162.
- Cleland, J.B. (1962), Disease in the Australian native, *Journal of Tropical Medicine and Hygiene* 65:95-105.
- Cleland, J.B. (1966), The ecology of the Aboriginal in South and Central Australia, In: Cotton, B.C. (ed), *Aboriginal man in South and Central Australia, Part I: Handbook of the Flora and Fauna of South Australia*, Adelaide.
- Cleland, J.B. and Fry, H.K. (1930), An outbreak of scurvy with joint lesions in Australian aborigines in Central Australia, *Medical Journal of Australia* 1:410-412.
- Cleland, J.B. and Johnston, T.H. (1933), The ecology of the Aborigines of central Australia: Botanical notes, *Transactions of the Royal Society of South Australia* 57:113-124.
- Cleland, J.B. and Johnston, T.H. (1937), Notes on native names and use of plants in the Musgrave Ranges, *Oceania* 8:208-215.
- Cleland, J.B. and Johnston, T.H. (1938), Notes on native names and use of plants in the Musgrave Ranges, *Oceania* 8:329-342.
- Clements, F.W. (1974), Aboriginal health and nutritional status, *Australian and New Zealand Journal of Medicine* 4:198-199.
- Cockington, R.A. (1980), Growth of Australian Aboriginal children related to social circumstances, *Australian and New Zealand Journal of Medicine* 10:322-325.
- Cole, K.E. (1980), *Arnhem Land: Places and people*, Rigby Publishers Ltd., Sydney.
- Coles-Rutishauser, I.H.E. (1979), Growing up in Western Australia: if you are Aboriginal, *Proceedings of the Nutrition Society of Australia* 4:27-32.
- Coles-Rutishauser, I.H.E. (1985), Food intake studies in Australian Aborigines: some methodological considerations, *Proceedings of the 13th International Congress of Nutrition*, pp 706-710. John Libbey, London.
- Coles-Rutishauser, I.H.E. (1987), Body mass and body composition in Australian Aboriginal women, In: *Proceedings of the Menzies Symposium, Nutrition and Health in the Tropics*, 57th ANZAAS Congress, Townsville, pp 226-232, MSHR, Darwin.
- Collier, G., McClean, A. and O'Dea, K. (1984), Effect of co-ingestion of fat on the metabolic responses to slow and rapidly absorbed carbohydrates, *Diabetologia* 26:50-54.
- Collmann, J. (1979). Social order and the exchange of liquor: a theory of drinking among Australian Aborigines, *Journal of Anthropological Research* 35:208-224.
- Connors, C. (1989). Medical officer, Womens Health Program, NTDHCS. (Personal communication).
- Conservation Commission of the Northern Territory (1987), *Gurig National Park plan of management*, Conservation Commission of the Northern Territory, NT, Darwin.
- Cook, C.E.A. (1966), Medicine and the Australian Aboriginal: A Century of contact in the Northern Territory, *Medical Journal of Australia* 1:559-565.
- Coolican, R.E. (1974), The role of the general practitioner in a rural part-Aboriginal community. In: Hetzel, B.S. et al (eds), *Better health for Aborigines?*, UQP, St. Lucia.
- Coombs, H.C. (1974), Decentralisation trends among Aboriginal communities, *Search* 5:135-43.
- Coombs, H.C. (1978), *Kulinma: listening to Aboriginal Australians*, ANU Press, Canberra.
- Coombs, H.C., Dexter, B.G., and Hiatt, L.R. (1982), The outstation movement in Australia. In: E. Leacock and R. Lee (eds), *Politics and history in band societies*, Cambridge University Press.
- Cordon, M. (1962), Observations on food habits of Europeans and Aborigines in the Northern Territory, *Food and Nutrition Notes and Reviews*, 19:3-8.
- Cowlishaw, G.K. (1980), Family planning: a post contact problem. In: Reid, J. (ed), *Body, land and spirit. Health and healing in traditional Aboriginal society*, pp 31-48, UQP, St. Lucia.
- Cowlishaw, G.K. (1981), The determinants of fertility among Australian Aborigines, *Mankind* 13:37-55.
- Cox, J.W. (1979), Growth characteristics of preschool Aboriginal children *Australian Paediatric Journal* 15:10-15.
- Coyne, T. and Darnton-Hill, I. (1979), Australian Aborigines: nutrition and changing disease patterns, *New Doctor* 12:32-37.
- Craig, G.M., Elliot, C. and Hughes, K.R. (1985), Masked vitamin B₁₂ and folate deficiency in the elderly. *British Journal of Nutrition* 54:613-619.
- Cribb, A.B. and Cribb, J.W. (1976), *Wild food in Australia*, William Collins, Sydney.
- Crotty, J.M. (1958), Anaemia and nutritional disease in Northern Territory native children, *Medical Journal of Australia* 2:322-325.
- Crotty, J.M. and Webb, R.C. (1960), Mortality in Northern Territory Aborigines, *Medical Journal of Australia* 2:489-492.

- Cummings, J.H. (1981), Dietary fibre and large bowel cancer, *Proceedings of the Nutrition Society of Australia* 40:7-14.
- Curnow, D.H. (1957), The serum proteins of Aborigines in the Warburton Ranges area, *Medical Journal of Australia* 2:608-609.
- Curr, E.M. (1886), *The Australian race, Volumes 1 and 2*, Trubner and Co, London.
- Curr, E.M. (1887), *The Australian race, Volumes 3 and 4*, Trubner and Co, London.
- Cutter, T. (1975), *Report to Central Australian Aboriginal Congress*, (unpublished), CAAC, Alice Springs.
- Cutter, T. (1978), Nutrition and food habits of the central Australian Aboriginal, In: Hetzel, B.S. and Frith, H.J. (eds), *Symposium on the nutrition of Aborigines in relation to the ecosystem of Central Australia*, pp. 63-72, CSIRO, Melbourne.
- Dadswell, I.W. (1934), The chemical composition of some plants used by Australian Aborigines as food, *Australian Journal of Experimental Biology and Medical Science* 12:13-18.
- Daley, C. (1931), Food of the Australian Aborigines, *Victorian Naturalist* 48: 23-31.
- Davidson, W.S. (1957), Health and nutrition of Warburton Range natives of Central Australia, *Medical Journal of Australia* 2:21-22.
- Davis, R.E., Kelly, A. and Byrne, G. (1965), Serum folate levels in the Australian Aboriginal, *Medical Journal of Australia* 2:21-22.
- Davis, R.E. and Pitney, W.R. (1957), Some haematological observations on Aborigines in the Warburton Ranges area, *Medical Journal of Australia* 2:605-608.
- Davis, R.E., Smith, B.K., and Curnow, D.H. (1975), Pyridoxal, folate and vitamin B₁₂ concentrations in Western Australian Aborigines, *Australian Journal of Experimental Biology and Medical Science*, 53:93-105.
- Davis, R.E. (1990). Senior haematologist, Royal Perth Hospital. (Personal communication).
- Davis, S. (1980), *Diet, disease and demography: hunter-gatherers in Arnhem Land*, Unpublished BA(Hons) Thesis, University of New England.
- Dearden, C., Harman, P. and Morley, D. (1980), Eating more fats and oils in a step towards over-coming malnutrition, *Tropical doctor* 10:137-142.
- DeFonzo, R.A. (1981), The effect of insulin on renal sodium retention: a review with clinical implications, *Diabetologia* 21:165-171.
- Department of Community Development (1989), *Aboriginal communities infrastructure assessment, Minjilang community*, DCD, Darwin.
- Department of Lands and Housing (1989), *Minjilang, Report on the availability of serviced land*, NT Government, Darwin.
- Department of Lands and Housing (1989a), *Warruwi, Report on the availability of serviced land*, NT Government, Darwin.
- Devanesen, D. (1983), Traditional art in the health worker training program, *The Aboriginal Health Worker* 7:4-8.
- Devanesen, D. (1986), *Traditional aboriginal medicine and the bicultural approach to health care in Australia's Northern Territory*, Unpublished paper, NTHD, Darwin.
- Devanesen, D. and Briscoe, J. (1980), *The health worker training program in Central Australia*, The Lambie-Dew oration 1980, Medical Society, University of Sydney, Sydney.
- Devanesen, D. and Henshall, T.S. (1982), A study of plant medicines in Central Australia, *Transactions of the Menzies Foundation* 4:161-166, The Menzies Foundation, Melbourne.
- Devanesen, D., Furber, M., Hampton, D., Morteza, H., Kinmonth, N. and Peach, H.G. (1986), *Health Indicators in the Northern Territory*, NTDH, Darwin.
- Devitt, J. (1988), *Contemporary Aboriginal women and subsistence in remote, arid Australia*, Unpublished PhD Thesis, Department of Anthropology and Sociology, University of Queensland.
- Diel, H. and Mannerberg, D. (1981), Hypertension, hyperlipidaemia, angina and coronary heart disease, In: Trowell, H.C. and Burkitt, D.P. (eds), *Western diseases: their emergence and prevention*, pp.372-392, Edward Arnold, London.
- Dinareello, C.A. (1985), Interleukin 1 and the pathogenesis of the acute phase response, *New England Journal of Medicine* 311:1413-1418.
- Djagamara (1980), Sugar abuse, *The Aboriginal Health Worker* 4:46-49.
- Dobbin, M.P.H. (1977), *The health and nutritional status of Victorian Aboriginal children*, Unpublished PhD thesis, Monash University.
- Doherty, R.L. (1973), The health status of Aboriginal communities in Cape York, In: R.L.Kirk, (ed), *The human biology of Aborigines in Cape York* pp 37-48, AIAS, Canberra.
- Doherty, R.L. (1974), A microbiologist looks at Aboriginal health, *Medical Journal of Australia* 2:286-289.
- Donald, Ngoi Ngoi (1980). Senior Aboriginal Health Worker, Areyonga, NT, (Personal communication).
- Doolan, J.K. (1977), Walk off (and later return), of various Aboriginal groups from cattle stations: Victoria River District, Northern Territory, In: Berndt, R.M. (ed), *Aborigines and change: Australia in the '70s*, pp 159-70, AIAS, Canberra.
- Douglas, R.A. (1957), Sprue, *Medical Journal of Australia* 2:525-526.
- Downing, J.H. (1974), Health Education in Alice Springs, In: B.S.Hetzel et al (eds), *Better health for*

- Aborigines?*, pp.191-200, UQP, St.Lucia.
- Duffy, P., Morris, H. and Neilson, G. (1981), Diabetes mellitus in the Torres Strait region, *Medical Journal of Australia (Special Supplement)* 1:8-11.
- Dugdale, A.E. (1980), Infant feeding, growth and mortality: A 20-year study of an Australian Aboriginal community, *Medical Journal of Australia* 2:380-385.
- Dugdale, A.E. (1986), *Nutrition of Aboriginal children and adults; The need for a long term perspective*, Discussion paper circulated at the NHMRC and Menzies Foundation Workshop: Research Priorities to Improve Aboriginal Health, 26th-28th November 1986, Araluen Arts Centre, Alice Springs, NT.
- Dugdale, A.E. and Lovell, S. (1981), The effect of early growth on distribution of subcutaneous fat in Aboriginal children, *Ecology of Food and Nutrition* 11:53-55.
- Dunn, F.L. (1968), Epidemiological factors: health and disease in hunter-gatherers, In: R. B. Lee and R. Devore (ed), *Man the Hunter*, pp. 221-228, Aldine, Chicago.
- Ealey, E.H.M. (1967), Ecology of the Euro *Macropus robustus* (Gould), in North-Western Australia, 1: The environment and changes in sheep and euro populations, *CSIRO Wildlife Research*, 12:9-24.
- Eastwell, H.D. (1981), Low zinc levels in Aborigines, *Medical Journal of Australia* 2:201.
- Eaton, S.B. and Konner, M. (1985), Paleolithic nutrition, A consideration of its nature and current implications, *New England Journal of Medicine* 312:283-289.
- Edwards, L.D. (1970), Malnutrition and disease in pre-school Aboriginal children in the Walgett area of NSW, *Medical Journal of Australia* 2:1007-1012.
- Edwards, F.M., Wise, P.H., Thomas, D.W., Murchland, J.B., and Craig, R.J. (1976), Blood pressure and electrocardiographic findings in the South Australian Aborigines, *Australian and New Zealand Journal of Medicine* 6:197-205.
- Elkin, A.P. (1974), *The Australian Aborigines: how to understand them*, (revised edn), Angus and Robertson, Sydney.
- Elkin, A.P. (1951), Reaction and interaction: a food gathering people and european settlements in Australia, *American Anthropologist* 53:164-186.
- Elliot, B., Maxwell, G.M. and Vawser, N. (1967), Lactose maldigestion in Australian Aboriginal children, *Medical Journal of Australia* 1:46-49.
- El Lozy, M. (1983), Dietary variability and its impact on nutritional epidemiology, *Journal of Chronic Disease* 36:237-249.
- Elphinstone, J.J. (1971), The health of Australian Aborigines with no previous association with Europeans, *Medical Journal of Australia* 2:293-301.
- English, R. (1989), Composition of foods, Australia, *Food Technology in Australia (Special Supplement)* 42:S5-S7.
- English, R.M. (1990), Vitamin B₁₂, In: Truswell, A.S., Dreosti, I.E., English, R.M., Rutishauser, I.H.E. and Palmer, N., *Recommended nutrient intakes, Australian papers*, pp 110-122, Australian Professional Publications, Sydney.
- Epstein, L.M., Reshef, A., Abrahamson, J.H. and Bialik, O. (1970), Validity of a short dietary questionnaire, *Israeli Journal of Medical Science* 6:589-597.
- Ernst, N., Fisher, M., Smith, W., Gordon, T., Rifkind, b.M., Little, J.A., Mishkel, M.A. and Williams, O.D. (1980), The association of plasma high density lipoprotein cholesterol and dietary intake and alcohol consumption, *Circulation* 62:41-52.
- Ernst, N. and Levy, R.I. (1980), Diet, hyperlipidaemia and atherosclerosis, In: Goodhart, R.S. and Sheils, M.E. (eds), *Modern nutrition in health and disease*, 6th edition, Lea and Febiger, Philadelphia, P.A.
- Eyre, E.J. (1845), *Journals of expeditions of discovery into central Australia and overland from Adelaide to King George's sound, in the years 1840-41*, T. and W. Boone, London.
- Fagan, T. (1984), The Aboriginal Medical Service, *New Doctor* 34:19-20.
- FAO/WHO (1979), Protein and energy requirements, *Bulletin of the WHO* 57:65-79.
- Ferguson, J. (1978), Dietitians as behaviour-change agents, *Journal of the American Dietetic Association* 72:231-235.
- Finlay-Jones, R.A. and McCormish, M.J. (1972), Prevalence of diabetes mellitus in Aboriginal lepers, The Derby survey, *Medical Journal of Australia* 2:135-137.
- Finlayson, H. H. (1935), *The red centre, Man and beast in the heart of Australia*, Angus and Robertson, Sydney. (Australiana Facsimile Editions No. 206. 1979).
- Fleming, K. and Devanesen, D. (1985), *Health policies and the development of Aboriginal self management in the Northern Territory*, Unpublished mimeograph, NTDH, Darwin.
- Flood, J. (1974), Pleistocene man at Clogg's Cave: his tool kit and environment, *Mankind* 9, 175-88.
- Foelsche, P (1881), Notes of the Aborigines of northern Australia, *Transactions of the Royal Society of South Australia* 5:1-18.
- Fong, J., Gorring, C. and Quarry, D. (1988a), *Minjilang (Crocker Island)*, NTDHCS, Darwin.
- Fong, J., Gorring, C. and Quarry, D. (1988b), *Warruwi (Goulburn Island)*, NTDHCS, Darwin.
- Fontbonne, A. et al (1989), Hypertriglyceridaemia as a risk factor of coronary heart disease mortality in subjects with impaired glucose tolerance or diabetes, *Diabetologia* 32:300-304.
- Ford, G.W., Belbin, R., Jose, D.G., Vorbach, E.A., and Kirke, D.K. (1976), Growth and immune function in

- Aboriginal children during recovery from malnutrition and infection, *Australian and New Zealand Journal of Medicine* 6:321-328.
- Franklin, M.A. (1982), Delivering health care to Aborigines, *Australian Journal of Social Issues* 17:276-287.
- Freire, P. (1973), *Pedagogy of the oppressed*, Pelican Books, Harmondsworth, Middlesex.
- Frith, N., Hausfeld, R.G., and Moodie, P.M. (1974), *The coast-town project: action research in Aboriginal community health*, AGPS, Canberra.
- Frith, H.J. (1978). Wildlife resources in central Australia, In: Hetzel, B.S. and Frith, H.J. (eds), (1978), *The nutrition of Aborigines in relation to the ecosystem of Central Australia*, pp 87-96, CSIRO, Melbourne.
- Fuller, J.H., Shipley, M.J., Rose, G., Jarrett, R.J. and Keen, H. (1980), Coronary heart disease risk and impaired glucose tolerance - The Whitehall study, *Lancet* 28:1373-1376.
- Fysh, C.F., Hodges, K.J. and Siggins, L.Y. (1960), Analysis of naturally occurring foodstuffs of Arnhem Land, In: Mountford, C.P. (ed), *Records of the American-Australian Scientific Expedition to Arnhem Land, Volume 2: Anthropology and Nutrition*, pp. 136-138, MUP, Melbourne.
- Gadomski, A. and Kjolhede, C. (1988), *Vitamin A deficiency and childhood morbidity and mortality*, School of hygiene and public health, The John Hopkins University, Occasional paper no.4.
- Gale, F. (1983), (ed), *We are bosses ourselves*, AIAS, Canberra.
- Gandevia, B. (1967), The prevalence of signs of chronic respiratory disease in Pintubi and Walbiri Aborigines at Papunya, Central Australia and Warburton, Western Australia, *Medical Journal of Australia* 2:237-242.
- Gandy, G. and Jacobson, W. (1977), Influence of folic acid on birthweight and growth of the erythroblastic infant, 1: Birthweight, *Archives of Diseases of Children* 52:1-6.
- Garcia-Palmieri, M., Sorlie, P., Tillotson, J., Costas, R., Cordero, E. and Rodriguez, M. (1980), Relationship of dietary intake to subsequent coronary disease incidence: the Puerto Rico Heart Health Program, *American Journal of Clinical Nutrition* 33:1818-1827.
- Garrow, J.S. (1974), *Energy balance and obesity in man*, North Holland Publishing Company, Amsterdam and London.
- Gault, A. (1990), *Health Survey, Urapuntja Health Service*, IAD, Alice Springs.
- Gershwin, M.E., Beech, R.S. and Hurley, I.S. (1985), *Nutrition and immunity*, Academic Press, San Diego.
- Gibb Committee (1973), *The situation of Aborigines on pastoral properties in the Northern Territory*, Report of Committee of review, December 1971, Parliamentary Paper No. 62 of 1972, AGPS, Canberra.
- Gibson, M. (1987), *Peoples of the North, Anthropology and tradition: A contemporary Aboriginal viewpoint*, Paper presented at ANZAAS, Townsville.
- Giles, E. (1889), *Australia twice traversed*. Sampson Low, Marston, Searle and Rivington Ltd., London.
- Glathhaar, C., Welbourn, T.A., Stenhouse, N.S. and Garcia-Webb, P. (1985), Diabetes and impaired glucose tolerance - A prevalence estimate based on the Busselton 1981 survey, *Medical Journal of Australia* 143:436-440.
- Goldman, L. and Cook, E.F. (1984), The decline in ischaemic heart disease mortality rates, *Annals of Internal Medicine* 101:825-36.
- Golson, J. (1971), Australian Aboriginal food plants: some ecological and cultural-historical implications. In: Mulvaney, D.J. and Golson, J. (eds), *Aboriginal man and environment in Australia*, pp. 196-238, ANU Press, Canberra.
- Goodall, J. (1987), *The Anthropology of Gender*, Lecture, University College Women's Association 5/2/87, Darwin, N.T.
- Gordon, T., Castelli, W.P., Hjortland, M.C., Kannel, W.B. and Dawber, T.R. (1977), High density lipoprotein as a protective factor against coronary heart disease: the Framingham Study, *American Journal of Medicine* 62:707-714.
- Gordon, T., Kagan, A., Garcia-Palmieri, M.R., Kannel, W.B., Zukel, W.J., Tillotson, J., Sorlie, P. and Hjortland, M. (1981), Diet and its relation to coronary heart disease and death in three populations, *Circulation* 63:500-515.
- Gould, R.A. (1967), Notes on hunting, butchering and sharing of game among the Nganatjara and their neighbours in the Western Australian Desert, *Kroeber Anthropological Society Papers* 36:41-66.
- Gould, R.A. (1969), Subsistence behaviour among the Western Desert Aborigines of Australia, *Oceania* 39:253-74.
- Gould, R.A. (1972), Progress to oblivion, *The Ecologist* 2:17-22.
- Gould, R.A. (1973), *Yiwara: forages of the Australian desert*, Charles Scribner, New York.
- Gould, R.A. (1980), *Living Archaeology*, Cambridge University Press.
- Gracey, M. (1973), Enteric disease in young Australian Aborigines, *Australian and New Zealand Journal of Medicine* 3:576-579.
- Gracey, M. (1976), Undernutrition in the midst of plenty: Nutritional problems of young Australian Aborigines, *Australian Paediatric Journal* 12:180-182.
- Gracey, M. (1977), Nutritional problems of Australian Aborigines, *Proceedings of the Nutrition Society of Australia* 2:11-16.
- Gracey, M. (1979), Nutritional problems of Australian Aboriginal children, *Food and Nutrition Notes and Reviews* 36:165-167.
- Gracey, M. (1985), Aborigines and Health, *Medical Journal of Australia (Special Supplement)* 143: S43-S44.

- Gracey, M. (1986a), The nutrition of Australian Aborigines, In: Wahlqvist, M.L. and Truswell, A.S. (eds), *Recent advances in clinical nutrition* pp. 57-68, John Libbey, London.
- Gracey, M. (1986b), Nutrition of Australian Aborigines, *Transactions of the Menzies Foundation* 2:65-69, The Menzies Foundation, Melbourne.
- Gracey, M., Bourke, V. and Robinson, J. (1980), Patterns of intestinal infection in Australian Aboriginal children, *Medical Journal of Australia* 2:375-380.
- Gracey, M., Murray, H., Hitchcock, N.E., Owles, E.N. and Murphy, B.P. (1983), The nutrition of Australian Aboriginal infants and young children, *Nutrition Research* 3:133-147.
- Gracey, M., Spargo, R.M., Bottrell, C., Hammond, K., Mulholland, K. and Vallentine, J. (1984), Maternal and childhood nutrition among Aborigines of the Kimberley region, *Medical Journal of Australia* 141:506-508.
- Gracey, M., Spargo, R.M. (1987), The state of health of Aborigines in the Kimberley region, *Medical Journal of Australia* 146:200-204.
- Gray, A. (1985), Some myths in the demography of Aboriginal Australians, *Journal of the Australian Population Association* 2:136-149.
- Gray, A. (1990), National estimates of Aboriginal mortality, In: Gray, A (ed), *A matter of life and death*, pp 147-159, Aboriginal Studies Press, Canberra,
- Gray, A.N. and Smith, L.R. (1983), The size of the Aboriginal population, *Australian Aboriginal Studies* 1:2-9.
- Gray, W. J. (1977), Decentralisation trends in Arnhem Land, In: Berndt, R.M. (ed), *Aborigines and change: Australia in the '70s*, pp 114-135, AIAS, Canberra.
- Grey, G. (1841), *Journal of two expeditions of discovery in North West and Western Australia*, T. and W. Boone, London.
- Grundy, S.M. (1986), Cholesterol and coronary heart disease: a new era, *Journal of the American Medical Association* 256:2849-2858.
- Grunseit, F. (1984), Aboriginal children (Comment), *Medical Journal of Australia* 140:697-698.
- Haffner, S.M., Stern, M.P., Hazuda, H.P., Rosenthal, M. and Knapp, J.A. (1986), The role of behavioural variables and fat patterning in explaining ethnic differences in serum lipids and lipoproteins, *American Journal of Epidemiology* 123:831-839.
- Hagen, R and Rowell, M. (1979), *The Anmatjira and Alyawarra Land Claim to Utopia pastoral lease*, Submission prepared for Central Land council, Alice Springs.
- Halsted, C.H. (1980), Folate deficiency in alcoholism, *American Journal of Clinical Nutrition* 33:2736.
- Hambidge, K.M. (1982), Hair analyses: worthless for vitamins, limited for minerals, *American Journal of Clinical Nutrition* 36:943-949.
- Hamilton, A. (1971), *Socio-cultural factors in health among the Pitjantjatjara: a preliminary report*, Unpublished mimeograph, Department of Anthropology, Macquarie University, Sydney.
- Hamilton, A. (1972), Blacks and whites: the relationship of change, *Arena* 30:34-48.
- Hamilton, A. (1974), The traditionally orientated community. In: B.S.Hetzel *et al* (eds), *Better health for Aborigines?*, pp. 14-29, UQP, St.Lucia.
- Hamilton, A. (1975), Aboriginal women, their means of production, In: *The other half: Women in Australian Society* Penguin.
- Hamilton, A. (1980), Dual social systems: technology, labour and women's secret rites in the eastern Western desert of Australia, *Oceania* 1:4-19.
- Hamilton, A. (1981), *Nature and nurture*, AIAS, Canberra.
- Hamilton, A. (1982), Child health and child care in a desert community. In: Reid, J. (ed), *Body, land and spirit: Health and healing in Aboriginal society*, pp. 49-71, UQP, St.Lucia.
- Hankin, J.H., Messinger, H.B. and Stallones, R.A. (1970), A short dietary method for epidemiologic studies, IV: Evaluation of questionnaire, *American Journal of Epidemiology* 91:562-567.
- Hankin, J.H., Rhoads, G.G. and Glober, G.A. (1975), A dietary method for the epidemiology of gastrointestinal cancer, *American Journal of Clinical Nutrition* 28:1055-1060.
- Hargreaves, J.C. and Jones, E.R. (1980), *Leprosy in tropical Australia: A short guide for field staff in the diagnosis, treatment and management of leprosy*, NTDH, Darwin.
- Harris, M.F., Nolan, B. and Davidson, A. (1984), Early childhood pneumonia in Aborigines of Bourke, New South Wales, *Medical Journal of Australia* 1:705-707.
- Harris, S. (1977a), Yolngu rules of interpersonal communication, *Developing Education* 4:(5).
- Harris, S. G. (1977b), *Milingimbi Aboriginal learning contexts*, Unpublished PhD thesis, University of New Mexico, Albuquerque.
- Harrison, L. (1986), *Diet and nutrition in a Tiwi community: a study of factors affecting the health status of under threes at Milikapiti, North Australia*, Unpublished PhD thesis, ANU, Canberra.
- Harrison, L. (1991), Food nutrition and growth in Aboriginal communities. In: Reid, J. and Trompf, P (eds), *The health of Aboriginal Australia*, pp. 123-173.
- Haskell, W.L., Taylor, H.L., Wood, P.D., Schrott, H. and Heiss, G. (1980), Strenuous physical activity, treadmill exercise, test performance and plasma high density lipoprotein cholesterol, *Circulation* 62:53-61.
- Hautvast, G.A.J. and Klaver, W. (1982), *A concerted action project on nutrition in the European community:*

The diet factor in epidemiological research, EURONUT Report 1, Wageningen.

- Heady, J.A. (1961), Diets of bank clerks: development of a method of classifying the diets of individuals for use in epidemiologic studies, *Journal of the Royal Statistical Society*, 124:336-361.
- Hegsted, D.M., McGrandy, R.B., Myers, M.L. and Stare, F.J. (1965), Quantitative effects of dietary fat on serum cholesterol in man, *American Journal of Clinical Nutrition* 17:281-295.
- Heiss, G., Johnson, N.J., Reiland, S., Davis, C.E. and Tyroler, H.A. (1980), The epidemiology of plasma high density lipoprotein cholesterol levels: The Lipid Research Clinics Program Prevalence study Summary, *Circulation* 62:116-136.
- Heller, R.F. (1986), The rise and fall of cardiovascular disease, *Medical Journal of Australia* 144: 686-688.
- Herbert, V. (1987a), Recommended dietary intakes (RDI) of folate in humans, *American Journal of Clinical Nutrition* 45:661-670.
- Herbert, V. (1987b), Nutrition science as a continually unfolding story: the folate and vitamin B₁₂ paradigm, *American Journal of Clinical Nutrition* 46:387-402.
- Hetzl, B.S. and Frith, H.J. (eds), (1978), *The nutrition of Aborigines in relation to the ecosystem of Central Australia*, CSIRO, Melbourne.
- Heywood, P.F. and Zed, C.A. (1977), Dietary and anthropometric assessment of the nutritional status of Aboriginal and white children in Walgett, NSW, *Proceedings of the Nutrition Society of Australia* 2:21-27.
- Hiatt (nee Meehan), B. (1967), The food quest and economy of the Tasmanian Aborigines, *Oceania*, 38:99-133.
- Hiatt, L.R. (1968), Ownership and land use amongst the Australian Aborigines, In: Lee, R.B. and Devore, I. (eds), *Man the hunter*, pp.99-102, Aldine, Chicago.
- Hiddens, L. (1985), Personal communication.
- Hilliard, W.M. (1968), *The people in between: The Pitjantjatjara people of Ernabella*, Hodder and Stoughton, London.
- Hitchcock, N.E. and Gracey, M. (1975a), Dietary patterns in a rural Aboriginal community in South-West Australia, *Medical Journal of Australia (Special Supplement)* 2:12-16.
- Hitchcock, N.E. and Gracey, M. (1975b), The setting for unsatisfactory health and nutritional standards in Australian Aborigines, *Medical Journal of Australia (Special Supplement)* 2:20-23.
- Hitchcock, N.E. and Gracey, M. (1978), Nutrient consumption patterns of families in Busselton, Western Australia, *Medical Journal of Australia* 1:359-362.
- Hitchcock, N.E., Gracey, M., Maller, R.A. and Spargo, R.M. (1987), The physical size of 1887 Aboriginal schoolchildren in the Kimberley region, *Medical Journal of Australia* 146:415-419.
- Hodges, K.J. (1960), Report of the biochemical assessments of nutritional status, In: Mountford, C.P. (ed), *Records of the American-Australian Scientific Expedition to Arnhem Land, Volume 2: Anthropology and Nutrition*, pp. 72-89, MUP, Melbourne.
- Hodges, R.E., Baker, E.M., Hood, J., Sauberlich, H.E., March, S.C. (1969), Experimental scurvy in man, *American Journal of Clinical Nutrition* 22:535-48.
- Hoffbrand, A.V., Newcombe, B.R.A. and Mollin, D.L. (1966), Method of assay of red cell folate activity and the value of the assay as a test for folate deficiency, *Journal of Clinical Pathology* 19:17-28.
- Holmes, M.J. (1913), Health report for the year 1912, *Bulletin of the NT of Australia*, 6.
- Holt, A.B., Spargo, R.M., Iveson, J.B., Faulkner, G.S. and Check, D.B. (1980), Serum and plasma zinc, copper and iron concentrations in Aboriginal communities of North Western Australia, *American Journal of Clinical Nutrition* 33:119-32.
- Hoppner, K. and Lampi, B. (1982), Total folacin activity in breakfast cereals, *Nutrition Reports International* 26:495-500.
- Houston, S. (1985), Aboriginal health, *Medical Journal of Australia (Special Supplement)* 143:S45.
- Hughes, T.A. and Gwynne, J.T., Switzer, B.R., Herbst, C. and White, G. (1984), Effects of caloric restriction and weight loss on glycemic control, insulin release and resistance, and atherosclerotic risk in obese patients with type 2 diabetes mellitus, *American Journal of Medicine* 77:7-17.
- Hunt, H. (1981), Alcoholism among Aboriginal people, *Medical Journal of Australia (Special Supplement)* 1: 1-3.
- Hunter, D. (1990), Biochemical indicators of dietary intake. In: Willett, W.C. *Nutritional epidemiology*, pp. 143-215, Oxford University Press, New York.
- Hyam, G.N. (1939), The Vegetable foods of the Australian Aborigines, *Victorian Naturalist* 56: 95-98, 115-119.
- Hyam, G.N. (1940), The animal food of the Australian Aborigines, *Victorian Naturalist* 57: 136-140.
- Icke, G.C. (1980), *The microbiological assay of thiamine and its clinical significance*, Unpublished MSc Thesis, University of Western Australia.
- Inyegar, L. and Rajalakshmi, K. (1975), Effect of folic acid supplement on birthweight of infants, *American Journal of Obstetrics and Gynaecology* 122:332-337.
- Irvine, F.R. (1957), Wild and emergency foods of Australian and Tasmanian Aborigines, *Oceania* 28:113-142.
- Irvine, F.R. (1970), Evidence of changes in the vegetable diet of Australian Aborigines. In: Pilling, A.R. and Waterman, R.A. (eds), *Diprotodon to Detribalisation: Studies of change among Australian Aborigines*, Michigan State University Press.
- Jacobs, D.S. and Clements, F.W. (1968), *Growth patterns of Australian Aboriginal children in the Northern*

- Territory, Unpublished paper, Australian Medical Congress, 1968.
- Jain, M.G., Harrison, L., Howe, G.R. and Miller, A.B. (1982), Evaluation of a self-administered dietary questionnaire for use in a cohort study, *American Journal of Clinical Nutrition* 36:931-935.
- James, K.W. (1983), Analysis of indigenous Australian foods, *Food Technology in Australia* 35:342-343.
- James, K.W., Hiddens, L.J. and Forbes-Ewan, C.H. (1982), Composition of Australian indigenous foods, *Proceedings of the Nutrition Society of Australia* 7:159.
- James, K.W., Tattersall, P.J., Forbes-Ewan, C.H. and Hancock, A.T. (1984), Composition of Australian indigenous foods, *Proceedings of the Nutrition Society of Australia* 9:173.
- James, K.W., Lichon, M.J., Tattersall, P.J. and Walker, G.J. (1986), Nutrient analysis of Australian indigenous foods, *Proceedings of the Nutrition Society of Australia* 11:102.
- James, W.P.T., Bingham, S.A. and Cole, T.J. (1981), Epidemiological assessment of dietary intake, *Nutrition and Cancer* 2:203-211.
- Jelliffe, D.B. (1966), *The assessment of the nutritional status of the community*, WHO Monogram Series No. 53, Geneva.
- Jenkins, D.J.A., Wong, G.S., Patten, R., Bird, J. et al (1983), Leguminous seeds in the dietary management of hyperlipidaemia, *American Journal of Clinical Nutrition* 38:567-573.
- Jensen, O.M., Wahrendorf, J., Rosenquist, A. and Geser, A. (1984), The reliability of questionnaire-derived historic dietary information and temporal stability of food habits in individuals, *American Journal of Epidemiology* 120:281-290.
- Johnston, T.H. and Cleland, J.B. (1934), The history of the Aboriginal narcotic, pituri, *Oceania* 4:201-223.
- Jones, P.N. and Mills, E.H. (1955), Serum vitamin B₁₂ concentrations in liver disease, *Journal of Laboratory and Clinical Medicine* 46:927-931.
- Jones, R. (1969), Fire-stick farming, *Australian Natural History* 16:224-228.
- Jones, R. (1971), The demography of hunters and farmers in Tasmania. In: Mulvaney, D.J. and Golson, J. (eds), *Aboriginal man and environment in Australia*, pp. 271-287, ANU Press, Canberra.
- Jones, R. (1977), Man as an element of a continental fauna: the case of sundering of the Bassian bridge, In: J. Allen, J. Golson, and R. Jones (ed), *Sunda and Sahul*, pp. 317-86, Academic Press, London.
- Jones, R. (1980), Hunters in the Australian coastal savanna, In: Harris, D. (ed), *Human Ecology in savanna environments*, pp 107-147, Academic Press.
- Jones, R. and Bowler, J. (1980), Struggle for the savanna: northern Australia in ecological and prehistoric perspective. In: Jones, R. (ed), *Northern Australia: Options and Implications*, pp. 3-33, Research School of Pacific Studies, ANU, Canberra.
- Jones, R.J. and Hegarty, M.P. (1981), Screening pasture plants for possible toxic effects on livestock. In: Wheeler, J.L. and Mochrie, R.D. (eds), *Forage evaluation: Concepts and Techniques*, pp. 237-241, CSIRO, Melbourne.
- Jose, D.G., Self, M.H.R., and Stallman, N.D. (1968), A survey of children and adolescents on Queensland Aboriginal settlements, 1967, *Australian Paediatric Journal* 5:71-88.
- Jose, D.G. and Welch, J.S. (1970), Growth retardation, anaemia and infection, with malabsorption and infection of the bowel- The syndrome of protein-calorie malnutrition in Australian Aboriginal children, *Medical Journal of Australia* 1:349-356.
- Jose, D.G., Shelton, M., Tauro, G.P., Belbin, R. and Hosking, C.S. (1975), Deficiency of immunological and phagocytic function in Aboriginal children with protein-calorie malnutrition, *Medical Journal of Australia* 2:699-705.
- Kaberry, P. (1935), *Aboriginal women - sacred and profane*, Routledge and sons, London.
- Kailis, D.G. (1979), Groote Eylandt studies 3: The influence of diet on the prevalence of dental caries in Aboriginal children at Groote Eylandt, NT Australia, 1973, *Proceedings of the Nutrition Society of Australia* 4:118.
- Kailis, D.G. (1982), Partial fluoridation and reduction in dental caries in Groote Eylandt Aboriginal children, 1974-1978, *Aboriginal Health Project Information Bulletin* 2:25-30.
- Kalman, D.A., Goodman, G.E., Osmenn, G.S., Bellamy, G. and Rollins. (1987), Micronutrient assay for cancer prevention trials: serum retinol, retinyl palmitate, α -carotene, and β -carotene with the use of high-performance liquid chromatography, *Journal of the National Cancer Institute* 79:975-982.
- Kalokerinos, A. (1969), Some aspects of Aboriginal infant mortality, *Medical Journal of Australia* 1:185.
- Kalokerinos, A. (1971), Letter, *Medical Journal of Australia* 1:185.
- Kalokerinos, A. (1974), *Every second child*, Nelson, Melbourne.
- Kamien, M. (1975), Aborigines and alcohol: Intake, effects and social implications in a rural community in Western New South Wales, *Medical Journal of Australia* 1:291-298.
- Kamien, M. (1976), The physical health of Aboriginal adults in Bourke, New South Wales, *Medical Journal of Australia (Special Supplement)* 1:38-44.
- Kamien, M. (1978), *The dark people of Bourke: A study of planned social change*, Humanities Press, New Jersey, U.S.A.
- Kamien, M. (1987), The secret bread tests: selective primary health care or experimentation on human beings? *Social Science and Medicine* 24:445-448.
- Kamien, M., Nobile, S., Cameron, P. and Rosevear, P. (1974), Vitamin and nutritional status of a part-

- Aboriginal community, *Australian and New Zealand Journal of Medicine* 4:126-37.
- Karnien, M., Woodhill, J.M., Nobile, S., Rosevear, P., Cameron, P. and Winston, J.M. (1975a), Nutrition in the Australian Aborigine: A dietary study of two Aboriginal families, *Food Technology in Australia* 27:93-103.
- Karnien, M., Woodhill, J.M., Nobile, S., Cameron, P., Rosevear, P. (1975b), Nutrition in the Australian Aborigines- effects of the fortification of white flour, *Australian and New Zealand Journal of Medicine* 5:123-133.
- Karvetti, R. and Knuts, L. (1985), Validity of the 24-hour recall, *Journal of the American Dietetic Association* 85:1437-1442.
- Kerr, G.R., Lee, E.S., Lam, M.M., Lorimor, R.J., Randall, E., Forthofer, R.N., Davis, M.A. and Magnetti, S.M. (1982), Relationships between dietary and biochemical measures of nutritional status in HANES 1 data, *American Journal of Clinical Nutrition* 35:294-308.
- Kettle, E.S. (1966), Weight and height curves for Australian Aboriginal infants and children, *Medical Journal of Australia* 1:972-977.
- Kettle, E.S. (1968), Rural health services in the Northern Territory, *Medical Journal of Australia (Special Supplement)* 2:106.
- Keys, A. (1965), Dietary survey methods in studies on cardiovascular epidemiology, *Voeding* 26:464-483.
- Keys, A. (1970), *Coronary Heart disease in seven countries*, American Heart Association, Monograph No. 29, AHA, New York.
- Keys, A. and Anderson, J.T. (1954), *Symposium on atherosclerosis*, National Academy of Science and National Research Council Publication No. 338.
- Keys, A., Anderson, J.T. and Grande, F. (1957), Prediction of serum-cholesterol responses of man to changes in fats in the diet, *Lancet* 2:959-966.
- Keys, A., Anderson, J.T. and Grande, F. (1965), Serum cholesterol response to changes in the diet. iv. Particular saturated fatty acids in the diet, *Metabolism* 14:776.
- Keys, A., Menotti, A., Karvonen, M.J., Aravanis, C., Fidanza, F., Keys, M.H., et al. (1986), The diet and 15-year death rate in the seven countries study, *American Journal of Epidemiology* 124:903-915.
- Kimber, R.G. (1976), Beginnings of farming? Some man-plant-animal relationships in Central Australia, *Mankind* 10:142-150.
- King, R.A., Smith, R.M., and Spargo, R.M. (1985), Dietary patterns of Aboriginal children in the Kimberley, *Proceedings of the Nutrition Society of Australia* 10:173.
- King, R.A., Smith, R.M., Wilkinson, G.N., Miller, R.B. and Spargo, R.M. (1988), Nutrient intake of Aboriginal children in the Kimberley, *Proceedings of the Nutrition Society of Australia* 13:164.
- Kirk, R.L. (1983), *Aboriginal Man Adapting*, Oxford University Press, Melbourne.
- Kirke, D.K. (1969), Growth rates of Aboriginal children in Central Australia, *Medical Journal of Australia* 2:1005-1009.
- Kirke, D.K. (1971), *Aboriginal infant and toddler mortality and morbidity*, M.D. Thesis, University of Adelaide.
- Kissebah, A.H., Vydellingum, N., Murray, R. et al (1982), Relation of body fat distribution to metabolic complications of obesity, *Journal of Clinical Endocrinology and Metabolism* 54:254-260.
- Klaver, W., Knuiman, J.T., van Staveren, W.A. (1982), Proposed definitions for use in the methodology of food consumption studies, In: Hautvast, G.A.J. and Klaver, W. (eds), *A concerted action project on nutrition in the European community: The diet factor in epidemiological research*, EURONUT Report 1, Wageningen.
- Klevey, L.M., Bistran, B.R., Fleming, C.R. and Neumann, C.G. (1987), Hair analysis in clinical and experimental medicine, *American Journal of Clinical Nutrition* 46:233-236.
- Knight, S. and Rowse, T. (1990), *The Hermannsburg store study: characteristics of shoppers*, Unpublished paper, MSHR, Alice Springs.
- Krantzler, N.J., Mullen, B.J., Comstock, E.M., Holden, C.A. Schultz, H.G., Grivetti, L.E. and Meiselman, H.L. (1982), Methods of food intake assessment- an annotated bibliography, *Journal of Nutrition Education* 14:108-119.
- Krotkiewski, M., Bjorntorp, P., Sjostrom, L. and Smith, U. (1983), Impact of obesity in metabolism in men and women: importance of regional adipose tissue distribution, *Journal of Clinical Investigation* 72:1150-1162.
- Kushi, L.H., Lew, R.A., Stare, F.J., Ellison, C.R., Lozy, M., Bourke, G., Daly, L., Graham, i., Hickey, N., Mulcahy, R. and Kevaney, J. (1985), Diet and 20 year mortality from coronary heart disease: the Ireland-Boston Diet-Heart study, *New England Journal of Medicine* 312:811-818.
- Kyle-Little, S. (1957), *Whispering wind: Adventures in Arnhem Land*, Hutchinson, London.
- Lamilami (1974), *Lamilami Speaks*, Ure Smith, Sydney.
- Larsson, B., Svardsudd, K., Welin, L., Wilhelmsen, L., Bjorntorp, P., and Tibblin, G. (1984), Abdominal adipose tissue distribution, obesity and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913, *British Medical Journal* 288:1401-1404.
- Latz, P.K. (1982), *Bushfires and Bushtucker: Aborigines and plants in Central Australia*, Unpublished MA thesis, University of New England, Armidale.
- Latz, P.K. and Griffin, G.F. (1978), Changes in Aboriginal land management in relation to fire and to food

- plants in central Australia, In: Hetzel, B.S. and Frith, H.J. (eds), *The nutrition of Aborigines in relation to the ecosystem of Central Australia*, CSIRO, Melbourne.
- Lawrence, R. (1969), *Aboriginal habitat and economy*, Occasional Paper No.6., Department of Geography, ANU, Canberra.
- Lawrence, R. (1971), Aboriginal habitat and economy: a historical perspective, In: Mulvaney, D.J. and Golson, J. (eds), *Aboriginal man and environment in Australia*, pp. 249-61, ANU Press, Canberra.
- Lee, A. (1985), Issues in Multicultural Nutrition Education, In: *New Dilemmas and Directions for Dietitians in a Growing Multicultural Society*, Proceedings of Dietitians Association of Australia National Education Workshop, Darwin, August 1985.
- Lee, A. (1987), Apparent consumption of food and nutrients in Coastal and Desert Aboriginal communities: Implications for nutrition education, In: *Proceedings of the Menzies Symposium: Nutrition and Health in the Tropics*, 57th ANZAAS Congress, Townsville, pp 270-288, MSHR, Darwin.
- Lee, A. (1990), Summary of nutrition report, In: Gault, A. *Health Survey, Urapuntja Health Service*, pp. 39-41, IAD, Alice Springs.
- Lee, A. (1991), Strategies to address poor nutritional status and non-communicable disease, In: *Report from the national conference on Aboriginal nutrition in remote and rural communities*, pp 29-39, Menzies School of Health Research, Government Printer of the Northern Territory.
- Leichhardt, L. (1847), *Journal of an overland expedition in Australia from Morton Bay to Port Essington 1844-1845*, London.
- Lester, J. (1974), Communication between Aborigines and Whites, In: Hetzel, B.S. et al (eds), *Better health for Aborigines?*, pp. 187-190, UQP, St. Lucia.
- Levitt, D. (1981), *Plants and people: Aboriginal uses of plants on Groote Eylandt*, AIAS, Canberra.
- Lickiss, J.N. (1970), Health problems of Sydney Aboriginal children, *Medical Journal of Australia* 2:995-1000.
- Lickiss, J.N. (1974), The urban situation, In: Hetzel, B.S. et al (eds), *Better health for Aborigines?*, pp. 29-33, UQP, St. Lucia.
- Lindsay, D. (1884), *Mr. D. Lindsay's explorations through Arnhem's Land*, South Australian Parliamentary Papers No. 239.
- Lindsay, D. (1890), Explorations in the Northern Territory of South Australia, *Papers of the Proceedings of the Royal Society of Tasmania (South Australia Branch)* 2:1-16.
- Lion, R., Rac, C. and O'Dea, K. (in preparation), *Health survey of two Northern Australian communities*, MSHR, Darwin.
- Lion, R., Bonson, A., Puruntatameri, A. and Rac, C. (1991). Good Food People, Paper presented at the 23rd. Annual Conference of the Public Health Association of Australia *The health of indigenous peoples*, Alice Springs.
- Liu, K., Stamler, J., Dyer, A., McKeever, J. and McKeever, P. (1978), Statistical methods to assess and minimise the role of intra-individual variability in obscuring the relationship between dietary lipids and serum cholesterol, *Journal of Chronic Disease* 31:399-418.
- Lockwood, D. (1964), *The lizard eaters*, Cassell, Melbourne.
- Long, J.P.M. (1970), *Aboriginal settlements: a survey of institutional communities in Eastern Australia*, Aborigines in Australian Society No. 3, ANU Press, Canberra.
- Long, J.P.M. (1971), Arid region Aborigines: the Pintubi, In: Mulvaney, D.J. and Golson, J. (eds), *Aboriginal man and the environment in Australia*, pp. 262-270, ANU Press, Canberra.
- Loveday, P. (ed), (1982), *Service delivery to remote communities*, NARU ANU, Darwin.
- LRCP, (1984), The Lipids Research Clinics coronary primary prevention trial results 11. The relationship of reduction in incidence of coronary heart disease to cholesterol lowering, *Journal of the American Medical Association* 251:365-374.
- McArthur, M. (1960a), Introduction to the report of the nutrition unit. In: Mountford, C.P. (ed), *Records of the American-Australian Scientific Expedition to Arnhem Land, Volume 2: Anthropology and Nutrition*, pp. 1-13, MUP, Melbourne.
- McArthur, M. (1960b), Food consumption and dietary levels of the Aborigines at the settlements, In: Mountford, C.P. (ed), *Records of the American-Australian Scientific Expedition to Arnhem Land, Volume 2: Anthropology and Nutrition*, MUP, Melbourne.
- McArthur, M. (1960c), Food consumption and dietary levels of the Aborigines living on naturally occurring foods. In: Mountford, C.P. (ed), *Records of the American-Australian Scientific Expedition to Arnhem Land, Volume 2: Anthropology and Nutrition*, pp. 90-136, MUP, Melbourne.
- McArthur, M. (1960d), Conclusions and recommendations, In: Mountford, C.P. (ed), *Records of the American-Australian Scientific Expedition to Arnhem Land, Volume 2: Anthropology and Nutrition*, pp. 139-143, MUP, Melbourne.
- McCarthy, F.D. (1957), Habitat, economy and equipment of the Australian Aborigines, *Australian Journal of Science* 19:88-97.
- McCarthy, F.D. and McArthur, M. (1960), The food quest and the time factor in Aboriginal economic life. In: Mountford, C.P. (ed), *Records of the American-Australian Scientific Expedition to Arnhem Land, Volume 2: Anthropology and Nutrition*, pp. 145-194, MUP, Melbourne.
- MacDonald, I. (1967), Inter-relationship between influence of dietary carbohydrate and fats on fasting lipids,

American Journal of Clinical Nutrition 20:345-351.

- Macfarlane, W.V. (1978), Aboriginal desert hunter/gatherers in transition, In: Hetzel, B.S. and Frith, H.J. (eds), *Symposium on the nutrition of Aborigines in relation to the ecosystem of Central Australia*, pp. 49-63, CSIRO, Melbourne.
- McGee, D., Reed, D., Stemmerman, G., Rhoads, G., Yano, K. and Feinleib, M. (1985), The relationship of dietary fat and cholesterol to mortality in 10 years: the Honolulu Heart Program, *International Journal of epidemiology* 14:97-105.
- McGrath, M., Collins, V., Zimmet, P. and Dowse, G. (1991), *Lifestyle disorders in Australian Aborigines*, International Diabetes Institute, Broilga Press, ACT.
- McGrath, A. (1983), *We grew up the stations: Europeans, Aborigines and cattle in the Northern Territory*, Unpublished PhD Thesis, Latrobe University.
- McGregor, G.A. (1983), Dietary sodium and potassium intake and blood pressure, *Lancet* 1:750-753.
- MacKenzie, C.J.C. (1980), European incursions and failures in northern Australia, In: Jones, R. (ed), *Northern Australia: Options and Implications*, pp. 43-72, Research School of Pacific Studies, ANU, Canberra.
- MacKnight, C.C. (1976), *The voyage to Marege: Macassan trepangers in North Australia*, MUP, Melbourne.
- MacLennan, R. and Pope, J.H. (1982), Models for health research in the Northern Territory, *Transactions of the Menzies Foundation* 4:93-100, The Menzies Foundation, Melbourne.
- McMillan, S. (1990), Group manager, Arnhem Land Progress Association, Darwin (Personal communication).
- McMillan, S. (1991), Issues affecting supply and demand of food in remote Aboriginal communities: the store perspective, In: *Report from the national conference on Aboriginal nutrition in remote and rural communities*, pp.99-109, Menzies School of Health Research, Government Printer of the Northern Territory.
- McNamara, D.J., Kolb, R., Parker, T.S., Batwin, H., Samuel, P., Brown, C.D. and Ahrens, E.H. Jr. (1987), Heterogeneity of cholesterol homeostasis in man: Response to changes in dietary fat quality and cholesterol quantity, *Journal of Clinical Investigation* 79:1729-1739.
- McNeilly, J., Cicchini, C., Oliver, D. and Gracey, M. (1983), Infectious disease in Aboriginal infants and children in Western Australia, *Medical Journal of Australia* 2:547-551.
- MacPherson, J. (1903), Medicine amongst the Australian Aborigines, *British Medical Journal* 2:96.
- Madden, J.P., Goodman, S.J. and Guthrie, H.A. (1976), Validity of the 24-hour recall, *Journal of the American Dietetic Association* 68:143-147.
- Maddock, K. (1972), *The Australian Aborigines: a portrait of their society*, Penguin, London.
- Maggiore, P. (1976), *A study of the nutritional content of some Aboriginal foods*, Paper presented to the ethnobotany workshop, AIAS, Canberra.
- Maggs, D.H. (1978), The potential for horticulture in Central Australia, In: Hetzel, B.S. and Frith, H.J. (eds), *Symposium on the nutrition of Aborigines in relation to the ecosystem of Central Australia*, pp. 115-127, CSIRO, Melbourne.
- Mahalko, J.R., Johnson, L.K., Gallagher, S.K. and Milne, D.B. (1985), Comparison of dietary histories and seven-day food records in a nutritional assessment of older adults, *American Journal of Clinical Nutrition* 42:542-553.
- Maiden, J.H. (1895), The chemistry of the Australian indigenous vegetation, *Reports of the ANZAAS* 6:25-57.
- Malinowski, B. (1913), *The family among the Australian Aborigines*, University of London Press, London.
- Mann, I. (1957), Report on the Ophthalmic findings in the Warburton Range natives of Central Australia, *Medical Journal of Australia* 2:610-612.
- Marbutt, J.A. (1971), The Australian Arid zone as a prehistoric environment, In: Mulvaney, D.J. and Golson, J. (eds), *Aboriginal Man and environment in Australia*, pp. 66-79, ANU Press, Canberra.
- Margetts, B.M. (1982), Report of a workshop on dietary methods in epidemiological studies in Australia, *Journal of Food and Nutrition* 39:197-202.
- Marmot, M.G. (1980), Affluence, urbanisation and coronary heart disease, In: Clegg, E.J. and Garlick, J.P. (ed), *Disease and urbanisation*, pp. 127-145, Taylor and Francis, London.
- Marr, J.W. (1965), Individual weighed dietary surveys, *Nutrition* 19:18-24.
- Marr, J.W. (1971), Individual dietary surveys: purposes and methods, *World Review of Nutrition and Dietetics* 13:105-164.
- Marr, J.W. (1973), Surveys: aims and methods, *Nutrition* 27:239-244.
- Martin, M.J., Hulley, S.B., Browner, W.S., Kuller, L.H. and Wentworth, D. (1986), Serum cholesterol, blood pressure and mortality: Implications from a cohort of 361,662 men, *Lancet* 2:933-936.
- Mata, L.J., Kromal, R.A., Viratia, J.J. and Garcia, B. (1977), Effect of infection on food intake and nutritional state- perspective as viewed from the village, *American Journal of Clinical Nutrition* 30:1215-1227.
- Mathews, J.D., Riley, M.D., Fejo, L., Munoz, E., Milns, N.R., Gardner, I.D., Powers, J.R., Ganygulpa, E. and Gununuwawuy, B.J. (1988), Effects of the heavy usage of kava on physical health: summary of a pilot survey in an Aboriginal community, *Medical Journal of Australia* 148:548-555.
- Mattingly, H.V. (1915), The teeth of some Australian Aboriginal natives and comparisons with the teeth of prehistoric man, *Commonwealth Dental Review*, 12:337-344, 12:388-394, Sydney.
- Mattson, F.H. and Grundy, S.M. (1985), Comparison of the effects of dietary saturated, mono-unsaturated and polyunsaturated fatty acids on plasma lipids and lipoproteins in man, *Journal of Lipid Research* 26:194-

- Maxwell, G.M. and Elliot, R.B. (1969), Nutritional state of Australian Aboriginal children, *American Journal of Clinical Nutrition* 22:716-24.
- Meehan, B. (1980), Who feeds the multitudes? The contribution women make to the diet of Aborigines in Tropical Australia, In: *Women in Food Chains*, Food Justice Centre of Friends of the Earth, Melbourne.
- Meehan, B. (1982), *Shell bed to shell midden*, AIAS, Canberra.
- Meggitt, M.J. (1957), Notes on the vegetable foods of the Walbiri of Central Australia, *Oceania* 28:143-145.
- Meggitt, M.J. (1962), *Desert people: a study of the Walpiri people of Central Australia*, Angus and Robertson, Sydney.
- Menkes, M.S., Comstock, G.W., Vuilleumier, J.P., Helsing, K.J., Rider, A.A. and Brookmeyer, R. (1986), Serum beta-carotene, vitamins A and E, selenium, and the risk of lung cancer, *New England Journal of Medicine* 315:1250-1254.
- Mensink, R.P. and Katan, M.B. (1989), Effect of a diet enriched with monounsaturated or polyunsaturated fatty acids on levels of low-density and high-density lipoprotein cholesterol in healthy women and men, *New England Journal of Medicine* 321:436-441.
- Menzies School of Health Research (1991), *Report from the national conference on Aboriginal nutrition in remote and rural communities*, Government Printer of the Northern Territory.
- Middleton, M. and Francis, S. (1976), *Yuendumu and its children - life and health on an Aboriginal settlement*, DAA, Canberra.
- Miles, M.R. (1978), Current problems in crop production in central Australian Aboriginal communities, In: Hetzel, B.S. and Frith, H.J. (eds), *Symposium on the nutrition of Aborigines in relation to the ecosystem of Central Australia*, pp. 97-104, CSIRO, Melbourne.
- Miller, G.J. and Miller, N.E. (1975), Plasma high density lipoprotein concentration and development of ischaemic heart disease, *Lancet* 1:16.
- Modan, M., Halkin, H., Almog, S., Lusky, A., Eshkol, A., Shelifi, M., Shitrit, A. and Fuchs, Z. (1985), Hyperinsulinaemia. A link between hypertension, obesity and glucose intolerance, *Journal of Clinical Investigation* 75:809-817.
- Moodie, P.M. (1969), Mortality and morbidity in Australian Aboriginal children, *Australian Paediatric Journal* 1:180-185.
- Moodie, P.M. (1972), The Health Disadvantages of Aborigines. In: Racism: the Australian experience. In: Stevens, F.S. (ed), *Black versus White, Volume 2*, pp. 235-42, ANZ Book Company.
- Moodie, P.M. (1973), *Aboriginal Health*, ANU Press, Canberra.
- Moodie, P.M. (1981), Australian Aborigines. In: Trowell, H.C. and Burkitt, D.P. (eds), *Western diseases: their emergence and prevention*, pp. 154-171, Edward Arnold, London.
- Moodie, P.M. and Pederson, E.B. (1971), *The Health of Australian Aborigines: An Annotated bibliography*, CDH, School of Public Health and Tropical Medicine, AGPS, Canberra.
- Moran, D.J., Waterford, J., Hollows, F., and Jones, D.L. (1979), Ear disease in rural Australia, *Medical Journal of Australia* 2:210-212.
- Morgan, R.W., Jain, M., Miller, A.B., Choi, N.W., Mathews, V., Munan, L., Burch, J.D., Feather, J., Howe, G.R. and Kelly, A. (1978), A comparison of dietary methods in epidemiological studies, *American Journal of Epidemiology* 107:488-498.
- Morice, R.D. (1976), Women dancing dreaming: psychosocial benefits of the Aboriginal outstation movement, *Medical Journal of Australia* 2:939-41.
- Morley, D. (1973), *Paediatric priorities in the developing world*, Butterworths, London.
- Mountford, C.O. (ed), (1960), *Records of the American-Australian Scientific expedition to Arnhem Land, Volume 2: Anthropology and Nutrition*, MUP.
- MRC, (1983), *The dietary assessment of populations*, Scientific Report No.4, Medical Research Council, Environmental Epidemiology Unit, Southampton General Hospital, Southampton.
- MRFIT Research Group, (1982), Multiple Risk Factor Intervention Trial: Risk factor changes and mortality results, *Journal of the American Medical Association* 248:1465-1477.
- MRFIT Research Group, (1986), Is relationship between serum cholesterol and risk of premature death from coronary heart disease continuous and graded?, *Journal of the American Medical Association* 2:2823-2828.
- Mulvaney, D.J. (1975), *The prehistory of Australia*, Penguin, Hammondswoth.
- Mulvaney, D.J. (1976), The chain of connection: the material evidence. In: N. Peterson (ed), *Tribes and Boundaries in Australia*, pp. 72-94, AIAS Canberra.
- Mulvaney, D.J. and Golson, J. (eds), (1971), *Aboriginal man and environment in Australia*, ANU Press, Canberra.
- Musgrave, I.A. (1984), Aboriginal mortality in Queensland, *Medical Journal of Australia* 140:16-19.
- NAHS Working Party (1989), *A national Aboriginal health strategy*, Canberra.
- NCHS, (1977), *Growth curves*, US Department of Health, Education and Welfare, Series 11, Number 165, USA.
- NHF, (1983), *The NHF Risk Factor Prevalence Study No. 2*, NHF, Canberra.

- NHF, (1986), *The NHF Risk Factor Prevalence Study in Darwin NT*, NHF, Darwin.
- NHMRC (1971), *Dietary allowances for use in Australia*, AGPS, Canberra.
- NHMRC (1987), *Recommended dietary intakes for use in Australia*, AGPS, Canberra.
- NHMRC (1989), *Implementing the dietary guidelines for Australians*, Report of the subcommittee on nutrition education, AGPS, Canberra.
- NHMRC (1991), *Guidelines on ethical matters in Aboriginal and Torres Strait Islander health research*, Approved by the 111 th session of the NHMRC, Brisbane, June 1991.
- NRC, (1968), *Recommended dietary allowances*, 7th revised edition, National Academy of Sciences, Washington.
- NRC, (1982), *Diet, nutrition and cancer*, Report of National Academy of Sciences, Assembly of Life Sciences, NRC, National Academy Press, Washington.
- NRC, (1985), Errors in nutrient intake measurement In: *Nutrient adequacy: assessment using food consumption surveys*, pp 48-66. National Academy Press, Washington.
- NTDH, (1977), *Aboriginal Health Worker Training Program in the Northern Territory*, Report of the Education and Training Task Force, NTDH, Darwin.
- NTDH, (1984), *Post basic skills module: Food and Nutrition*, NTDH, Darwin.
- NTDH, (1985), *Annual report, 1984-85*, NTDH, Darwin.
- Nathan, P. and Japanangka, D.L. (1983), *Health Business*, Heinmann Educational, Victoria.
- Naughton, J.M., O'Dea, K. and Sinclair, A.J. (1986), Animal foods in traditional Australian Aboriginal diets: Polyunsaturated and low in fat, *Lipids* 11:684-690.
- Neel, J.V. (1962), Diabetes mellitus, a thrifty genotype rendered detrimental by progress?, *American Journal of Human Genetics* 14:353-358.
- Neilson, G. and Williams, G. (1978), Blood pressure and valvular and congenital heart disease in Torres Strait Islanders, *Medical Journal of Australia (Special Supplement)*, 1:12-16.
- Nelson, M. (1983), Food production and sales, In: *The dietary assessment of populations*, Scientific Report No.4, pp. 16-20, Medical Research Council, Environmental Epidemiology Unit, Southampton.
- Nettleton, P.A., Day, K.C. and Nelson, M. (1980), Dietary survey methods, 2: A comparison of nutrient intakes within families assessed by household measures and the semi-weighed method, *Journal of Human Nutrition* 34:349-354.
- Neumann, C.G. (1980), Maternal nutrition and neonatal immunoglobulins, In: Jelliffe, D.B. and Jelliffe, E.F.B. (eds), *Advances in international maternal and child health, Vol 2*, OUP, Oxford.
- Newsome, A.E. (1971), Competition between wildlife and domestic stock, *Australian Veterinary Journal* 47:577-586.
- Nichols, A.B., Ravenscroft, C., Lamphier, D.E. and Ostrander, L.D. (1976), Daily nutritional intake and serum lipid levels: The Tecumseh Study, *American Journal of Clinical Nutrition* 29:1384-1392.
- Nobile, S. (1974), Blood vitamin levels in Aboriginal children and their mothers in Western NSW, *Medical Journal of Australia* 1:601-607.
- Noetling, F. (1910), The food of the Tasmanian Aborigines, *Papers and Proceedings of the Royal Society of Tasmania*, 279-305.
- Nolan, J. (1990), Haematology registrar, Royal Darwin Hospital (Personal communication).
- Northern Territory Aboriginal Communities, (1988), *Traditional bush medicines: An Aboriginal pharmacopoeia*, Greenhouse Publications, Darwin.
- Nurcombe, B. (1974), Petrol inhalation in Arnhemland, In: Hetzel, B.S. et al (eds), *Better health for Aborigines?*, pp. 66-71, UQP, St. Lucia.
- Nye, L.J.J. (1937), Blood pressure of the Australian Aboriginal, with a consideration of possible aetiological factors in hyperpiesia and its relation to civilisation, *Medical Journal of Australia* 2:1000-1001.
- O'Connell, J.F. (1977), Room to move: contemporary Alyawara settlement patterns and their implications for Aboriginal housing policy, *Mankind* 11:119-131.
- O'Connell, J.F., Latz, P.K. and Barnett, P. (1983), Traditional and modern plant use among the Alyawara of Central Australia, *Economic Botany* 37: 80-109.
- O'Dea, K. (1984), Marked improvement in carbohydrate and lipid metabolism in diabetic Australian Aborigines after temporary reversion to traditional lifestyle, *Diabetes* 33:596-603.
- O'Dea, K. (1986a), Aboriginal health and changes in lifestyle, *Australian Family Physician* 15:875-881.
- O'Dea, K. (1986b), *Summary of research on Aboriginal nutrition*, Paper prepared for NHMRC/Menzies Foundation Workshop on Research Priorities in Aboriginal Health, Alice Springs, 1986.
- O'Dea, K. (1987), Body fat distribution and health outcome in Australian Aborigines, *Proceedings of the Nutrition Society of Australia* 12:56-75.
- O'Dea, K. (1988), The hunter-gatherer lifestyle of Australian Aborigines: implications for health, In: McLean, A.J. and Wahlqvist, M.L. (eds), *Nutrition, pharmacology and toxicology*, pp. 26-36, John Libbey, London.
- O'Dea, K. and Sinclair, A.J. (1982), Increased proportion of arachidonic acid in plasma lipids after two weeks on a diet of tropical seafood, *American Journal of Clinical Nutrition* 36:868-872.
- O'Dea, K. and Sinclair, A.J. (1985), The effects of low fat diets rich in arachidonic acid on the composition of plasma fatty acids and bleeding time in Australian Aborigines, *International Journal of Nutrition*

- O'Dea, K., Spargo, R.M. and Ackerman, K. (1980), The effect of transition from traditional to urban life-style on the insulin secretory response in Australian Aborigines, *Diabetes Care* 3:31-37.
- O'Dea, K. and Spargo, R. (1982), Metabolic adaptation to a low carbohydrate-high protein ('traditional'), diet in Australian Aborigines, *Diabetologia* 23:494-498.
- O'Dea, K., Spargo, R. and Nestel, P.J. (1982), Impact of Westernisation on carbohydrate and lipid metabolism in Australian Aborigines, *Diabetologia* 22:148-153.
- O'Dea, K., Naughton, A.J., Rabuco, L., and Smith, R.M. (1987), Lifestyle change and nutritional status in Kimberley Aborigines, *Australian Aboriginal Studies* 1:46-51.
- O'Dea, K., Traianedes, K., Hopper, J.L. and Larkins, R.G. (1988a), Impaired glucose tolerance, hyperinsulinaemia, and hypertriglyceridaemia in Australian Aborigines from the desert, *Diabetes care* 11:23-29.
- O'Dea, K., White, N.G., and Sinclair, A.J. (1988b), An investigation of nutrition-related risk factors in an isolated Aboriginal community in Northern Australia: advantages of a traditionally orientated lifestyle, *Medical Journal of Australia* 148:177-180.
- O'Dea, K., Lion, R.J., Lee, A.J., Trianedes, K., Hopper, J.L. and Rae, C.J. (1990), Diabetes, hyperinsulinemia and hyperlipidaemia in a small Aboriginal community in Northern Australia, *Diabetes Care* 13:830-834.
- Ohlson, L.O., Larsson, B., Svardsudd, K., Welin, L., Eriksson, H., Wilhelmsen, L., Bjorntorp, P. and Tibalin, G. (1985), The influence of body fat distribution on the incidence of diabetes mellitus- 13.5 years of the follow up of participants in the study of men born in 1913, *Diabetes* 34:1055-1058.
- Packer, A.D. (1961), The health of the Australian Native, *Oceania* 32:60-70.
- Pain, R.W. (1988), Are your laboratory's cholesterol results reliable enough?, *Medical Journal of Australia* 149:113-115.
- Palmer, E. (1883), On plants used by the natives of North Queensland, *Proceedings of the Royal Society of NSW* 17:93-113.
- Parsons, P.A. (1965), Growth in a sample of Australian Aborigines from one to twenty-three months of age, *Growth*, 29:207.
- Patel, M.S., Phillips, C.B. and Cabaron, Y. (1991), Frequent hospital admissions for bacterial infections among Aboriginal people with diabetes in Central Australia, *Medical Journal of Australia* 155:218-222.
- Patel, M.S., O'Dea, K. *et al* (in preparation), Hermannsburg health survey.
- Patterson C. (1987), Problems in the Assessment and Overcoming of Nutritional Deficiencies in Australian Aborigines, *Medical Journal of Australia* 147: 319-320.
- Paul, A.A. and Southgate, D.A.T. (1978), *McCance and Widdowson's the composition of foods*, 4th Edition, Her Majesty's Stationery Office, London.
- Peach, H. (1986), Personal communication.
- Pekkarinen, M. (1970), Methodology in the collection of food consumption data, *World Review of Nutrition and Dietetics* 12:145-171.
- Penny, D.H. (1977), *Social accounting for Aboriginal communities: the cases of Willowra and Papunya*, Unpublished monograph, Research division, DAA, Canberra.
- Penny, D.H. and Moriarty, J. (1978), The Aboriginal economy- then and now, In: Hetzel, B.S. and Frith, H.J. (eds), *Symposium on the nutrition of Aborigines in relation to the ecosystem of Central Australia*, pp. 19-24. CSIRO, Melbourne.
- per Bjorntorp, P. (1985), Regional patterns of fat distribution, *Annals of Internal Medicine* 103 (6;2): 994-995.
- Peterson, N. (1973), Camp site location among Australian hunter-gatherers: archaeological and ethnographic evidence for a key determinant, *Archaeology and Physical Anthropology in Oceania* 8:173-193.
- Peterson, N. (1976), The natural and cultural areas of Aboriginal Australia, In: N. Peterson (ed), *Tribes and Boundaries in Australia*, pp 50-71, AIAS, Canberra.
- Peterson, N. (1977a), Aboriginal use of Australian solanaceae, In: Hawkes, J. (ed), *The biology and taxonomy of the solanaceae*, Academic Press, London.
- Peterson, N. (1977b), Aboriginal involvement with the Australian economy in the central reserve during the winter of 1970, In: Berndt, R.M. (ed), *Aborigines and change: Australia in the '70s*, pp.136-147, AIAS, Canberra.
- Peterson, N. (1978), The traditional pattern of subsistence to 1975, In: Hetzel, B.S. and Frith, H.J. (eds), *Symposium on the nutrition of Aborigines in relation to the ecosystem of Central Australia*, pp. 25-35, CSIRO, Melbourne.
- Phillips, M and Kubisch, D. (1985), Letter: Lifestyle diseases in Aborigines, *Medical Journal of Australia* 143:218.
- Pietenen, P. and Huttunen, J.K. (1987), Dietary fat and blood pressure- a review, *European Heart Journal* 8:9-17.
- Pitney, W.R. (1962), Serum vitamin B12. concentrations in the Western Australian Aborigines, *Australian Journal of Experimental Biology and Medical Science* 40:73-80.
- Plant, A. (1990), What do we know about premature adult mortality of Aboriginal Australians, In: Gray, A (ed), *A matter of life and death*, pp 77-85, Aboriginal Studies Press, Canberra,

- Prentice, A.M., Black, A.E., Coward, W.A., Davies, H.L., Goldberg, G.R., Murgatroyd, P.R., Ashford, J., Sawyer, M. and Whitehead, R.G. (1986), High levels of energy expenditure in obese women, *British Medical Journal* 292:983-987.
- Prokopec, M. (1979), Demographical and morphological aspects of the Roonka population, *Archaeology and Physical Anthropology in Oceania*, 14:11-26.
- Proper, D.N., Edmonds, R., and Parsons, P.A. (1968), Birth weights and growth rates up to one year for full-blood and mixed-blood Australian Aboriginal children, *Australian Paediatric Journal* 4:134-143.
- RACO, (1980), *Report of the National trachoma and eye program*, RACO, Sydney.
- Rae, C.J. (1988), *The present and future role of dietitians in rural community health services*, Unpublished document, Nutrition Department, NTDHCS, Darwin.
- Rae, C.J. (1989), *Maternal nutritional status among Aborigines in the Northern Territory: Impact on birth weight*, Unpublished MPH thesis, University of Sydney.
- Rae, C.J., Lamprell, V.J., Lion, R.J. and Rae, A.M. (1982), The role of bush foods in contemporary Aboriginal diets, *Proceedings of the Nutrition Society of Australia* 7:45-49.
- Rae, C.J., Lion, R.J., Riley, M.D., Dyer, M.E. and Kulbek, M. (1987), Are caucasian standards indicating diabetes risk in Aborigines, In: *Proceedings of the Menzies Symposium: Nutrition and Health in the Tropics*, 57th ANZAAS Congress, Townsville, pp 242-253, MSHR, Darwin.
- Räsänen, L. (1982), Validity and reliability of recall methods, In: Hautvast, G.A.J. and Klaver, W. (eds), *A concerted action project on nutrition in the European community: The diet factor in epidemiological research*, EURONUT Report 1, pp 92-100, Wageningen.
- Rassaby, L. (1978), Malnutrition amongst Aborigines in the inner city of Sydney, *New Doctor* 8:36-38.
- Read, R.S.D. (1990), Vitamin C. In: Truswell, A.S., Dreosti, I.E., English, R.M., Rutishauser, I.H.E. and Palmer, N., *Recommended nutrient intakes, Australian papers*, pp 122-149, Australian Professional Publications, Sydney.
- Reaven, G.M. and Miller, R. (1970), Study of the relationship between glucose and insulin responses to an oral glucose load in man, *Diabetes* 17:560-569.
- Reaven G.M., Bernstein, R., Davis, B. and Olefsky, J.M. (1976), Nonketotic diabetes mellitus: insulin deficiency or insulin resistance?, *American Journal of Medicine* 60:80-88.
- Rebgetz, P. (1985). Delivery of health care to Aboriginal communities in the Northern Territory, In: *Health in black and white*, Report of National Physiotherapy Association Conference, Darwin.
- Renaud, S. (1985), Dietary fatty acids and platelet function, *Proceedings of the Nutrition Society of Australia* 10:1-13.
- Reid, J. (1982), (ed), *Body, land and spirit: Health and healing in Aboriginal society*, UQP, St. Lucia.
- Reid, J.C. (1986), Land of milk and honey: the changing meaning of food to an Australian Aboriginal community, In: Manderson, L. (ed), *Shared wealth and symbol: Food culture and society in South East Asia and the South Pacific*, pp 49-67, Cambridge University Press.
- Reid, J.C. and Mununggurr, D. (1977), We are losing our brothers: Sorcery and alcohol in an Aboriginal community, *Medical Journal of Australia (Special Supplement)* 2:1-5.
- Reid, J.C. and Swain, C. (undated), *Rethinking the status of old people in Australian Aboriginal Society*, Commonwealth Institute of Health, Sydney University.
- Reilly, C. and Harrison, F. (1983), Nutritional implications of levels of zinc, copper, iron and lead in hair of British and Australian children, *Proceedings of the Nutrition Society of Australia* 8:209.
- Reynolds, H. (1982), *The other side of the frontier, Aboriginal resistance to the European invasion of Australia*, Penguin, Melbourne.
- Rifkind, B.M., Goor, R. and Schucker, B. (1983), Compliance and cholesterol-lowering in clinical trials: efficacy of diet, In: Schettler, F.G., Gotto, A.M., Middelhoff, G., Habenecht, A.J.R. and Jurutka, K.R. (eds), *Atherosclerosis vi, Proceedings of the sixth international symposium*, Springer-Verlag, New York.
- Riley, M.D., Davis, R.E., Jago, J.D., Mathews, J.D., and Lee, A.J. (1990), Thiamine status in an isolated Aboriginal community, (In preparation).
- Rivett, D.E., Tucker, D.J. and Jones, G.P. (1983), The chemical composition of seeds from some Australian plants, *Australian Journal of Agricultural Research*, 34:427-32.
- Roberts, D.C.K. (1990), Vitamin E. In: Truswell, A.S., Dreosti, I.E., English, R.M., Rutishauser, I.H.E. and Palmer, N. (1990), *Recommended nutrient intakes, Australian papers*, pp. 159-176, Australian Professional Publications, Sydney.
- Roberts, R.W. and Rutishauser, I.H.E. (1978), Are Aborigines underfed? (Abstract), *Australian Paediatric Journal* 14:218.
- Rose, F.G.G. (1965), *The winds of change in Central Australia*, Akademie-Verlag, Berlin.
- Rose, F.G.G. (1987), *The traditional mode of production of the Australian Aborigines*, Angus and Robertson, London.
- Rose, G. (1984), International trends in cardiovascular disease - Implications for prevention and treatment, *Australian and New Zealand Journal of Medicine* 14:375-380.
- Rose, G. (1985), Sick individuals and sick populations, *International Journal of epidemiology* 14:32-38.
- Roth, W.E. (1897), *Ethnological studies among the North-West-Central Queensland Aborigines*, Government

- Printer, Brisbane, Queensland.
- Roth, W.E. (1901), Food, its search, capture and preparation, *North Queensland Ethnography Bulletins*, Vol 3. Government Printer, Brisbane, Queensland.
- Rountree, P.M. (1969), The nasal flora of Australian Aborigines of the Pitjantjatjara tribe, *Medical Journal of Australia* 2:344.
- Rowley, C.D. (1970), *The destruction of Aboriginal Society*, Aborigines in Australian Society, No. 4, ANU Press, Canberra.
- Rowley, C.D. (1971a), *Outcasts in white Australia - Aboriginal policy and practice*, Volume 2, ANU Press, Canberra.
- Rowley, C.D. (1971b), *The remote Aborigines*, Aborigines in Australian Society, No. 7, ANU Press, Canberra.
- Rowley, C.D. (1978), *A matter of justice*, ANU Press, Canberra.
- Rowley, K. and Rowley, B. (1978), Some practical ideas for teaching nutrition, *The Aboriginal Health Worker* 2:45-47.
- Rowse, L.L. and Beilin, L.J. (1983), Nutrition, blood pressure and hypertension, *Medical Journal of Australia (Special Supplement)*, 2:s19-s23.
- Rowse, T. (1988), *From rations to citizenship*, Unpublished paper prepared for the International Hunters and Gatherers conference, Darwin 1988.
- Rutishauser, I.H.E. (1982), Food models, photographs or household measures? *Proceedings of the Nutrition Society of Australia* 7:144.
- Rutishauser, I.H.E. (1990a), Vitamin A. In: Truswell, A.S., Dreosti, I.E., English, R.M., Rutishauser, I.H.E. and Palmer, N., *Recommended nutrient intakes, Australian papers*, pp. 36-51, Australian Professional Publications, Sydney.
- Rutishauser, I.H.E. (1990b), Riboflavin. In: Truswell, A.S., Dreosti, I.E., English, R.M., Rutishauser, I.H.E. and Palmer, N., *Recommended nutrient intakes, Australian papers*, pp. 64-74, Australian Professional Publications, Sydney.
- Rutishauser, I.H.E. (1990c), Vitamin B₆. In: Truswell, A.S., Dreosti, I.E., English, R.M., Rutishauser, I.H.E. and Palmer, N., *Recommended nutrient intakes, Australian papers*, pp. 84-96, Australian Professional Publications, Sydney.
- Rutishauser, I.H.E. and McKay, H. (1986), Anthropometric status and body composition in Aboriginal women of the Kimberley region, *Medical Journal of Australia (Special Supplement)* 144:s8-s10.
- Rutishauser, I.H.E. and Walqvist, M.L. (1984), Making measurements of food and alcohol intake, *Transactions of the Menzies Foundation* 7:97-110.
- Sandison, A.T. (1973), Disease changes in Aboriginal skeletons, *AIAS Newsletter* 3:20-22.
- Sandison, A.T. (1980), Notes on skeletal changes in pre-European contact Australian Aborigines, *Journal of Human Evolution* 9:45-47.
- Sansom, B. (1980), *The camp at Wallaby Cross, Aboriginal fringe dwellers in Darwin*, AIAS, Canberra.
- Sauberlich, H. E., Dowdy, R.P. and Skala, J.H. (1974), *Laboratory tests for the assessment of nutritional status*, CRC Press, Cleveland.
- Scarlett, N.H. (1976), *Ritjja and Gathul: the role of monsoon and mangrove forests in Yualngu traditional economy*, Unpublished mimeograph, AIAS, Ethnobotany workshop, Canberra.
- Scarlett, N., White, N. and Reid, J. (1982), Bush medicines: the pharmacopoeia of the Yolngu of Arnhem Land. In: Reid, J. (ed), *Body, land and spirit. Health and healing in Aboriginal society*, pp 154-191, UQP, St.Lucia.
- Schonfeld, G., Patsch, W., Rude, L.L., Nelson, C. et al (1982), Effects of dietary cholesterol and fatty acids on plasma lipoproteins, *Journal of Clinical Investigation* 69:1072-1080.
- Schwartz, C.J. (1959), Blood pressure, serum cholesterol and atherosclerosis in Australian Aborigines, *Medical Journal of Australia* 2:695.
- Schwartz, C.J., and Casley-Smith, J.R. (1958a), Serum cholesterol levels in atherosclerotic subjects and in the Australian Aborigines, *Medical Journal of Australia* 2:84-86.
- Schwartz, C.J., and Casley-Smith, J.R. (1958b), Atherosclerosis and the serum mucoprotein levels of the Australian Aborigine, *Australian Journal of Experimental Biology and Medical Science* 36:117-120.
- Schwartz, C.J., Day, A.J., Peters, J.A. and Casley-Smith, J.R. (1957), Serum cholesterol and phospholipid levels of Australian Aborigines, *Australian Journal of Experimental Biology and Medical Science* 35:449-456.
- Scrimgeour, D. (1983), *Growth failure in Aboriginal children*, Unpublished paper prepared for Tropical child health unit, Institute of child health, London.
- Scrimshaw, N.S., Taylor, C.E. and Gordon, J.E. (1968), *Interactions of nutrition and infection*, WHO Monograph Series, No.57, Geneva.
- Sempos, C.T., Johnson, N.E., Smith, E.L. and Gilligon, C. (1985), Effects of intra-individual and inter-individual variation in repeated dietary records, *American Journal of Epidemiology* 121:120-130.
- Senti, F.R. and Pilch, S.M. (1984), In: Senti, F.R. and Pilch, S.M. (eds), *Assessment of the folate nutritional status of the US population based on data collected in the second national health and nutritional status examination survey, 1976-1980*, Life Sciences Research Office, Federation of American Societies for Experimental Biology, Bethesda.

- Seward, J.F. and Stanley, F.J. (1981), Comparison of births to Aboriginal and caucasian mothers in Western Australia, *Medical Journal of Australia* 2:80-84.
- Shekelle, R., Macmillan, A., Paul, O., Lepper, M. et al. (1981), Diet, serum cholesterol, and death from coronary heart disease: The Western Electric study, *New England Journal of Medicine*, 304:65-70.
- Shelley, C.J. (1979), *Food and nutrition*, Aboriginal Health Worker training program, Post basic health course, pp 119, NTHD, Darwin.
- Shelley, C.J. (1982), Nutrition education in the Northern Territory, *Journal of Food and Nutrition* 39:81-86.
- Shelley, C., Lamprell, V. and Lion, R. (1981), Tucker today, *Proceedings of the Nutrition Society of Australia* 6:122.
- Sibthorpe, B. (1989), *All our people are dyin': Food and Health in a Rural Aboriginal Community*, Unpublished PhD thesis, ANU, Canberra.
- Silberbauer, G.B. (1971), Ecology of the Ernabella Aboriginal community, *Anthropological Forum*, 3:21-36.
- Simons, L., Whish, P., Marr, B., Jones, A. and Simon, J. (1981), Coronary risk factors in a rural community which includes Aborigines: Inverell heart disease prevention program, *Australian and New Zealand Journal of Medicine* 11:386-390.
- Simons, L.A. et al (1986), Serum cholesterol and coronary heart disease: Implications of recent intervention studies, *Australian and New Zealand Journal of Medicine* 16:528-535.
- Simopoulos, A.S. (ed), (1982), Assessment of nutritional status, Selected papers: Conference on the assessment of nutritional status, *American Journal of Clinical Nutrition* 35:1089-1325.
- Sinclair, H. (undated), *The problem of changing food habits*, Departmental document, NTDH.
- Sinclair, H. (1968), Nutrition and the Aboriginal, *Health* 18:11-14.
- Sinclair, H. (1977), Factors affecting Aboriginal nutrition, *Medical Journal of Australia (Special Supplement)*, 1:1-4.
- Sladden, T.J. (1987), Cardiovascular disease risk factors in an Aboriginal community, Unpublished MSc thesis, University of Sydney.
- Smith, L.R. (1982), Aboriginal health and Aboriginal health statistics, *Aboriginal Health Information Bulletin* 1:14-24.
- Smith, R.M., Spargo, R.M. and Cheek, D.B. (1982), Zinc status and the growth of Aboriginal children in the North-West of Australia, *Proceedings of the Nutrition Society of Australia* 7:37-44.
- Smith, R.M., King, R.A., Spargo, R.M., Cheek, D.B., Field, J.B. and Veitch, L.G. (1985), Growth-retarded Aboriginal children with low plasma zinc levels do not show a growth response to supplementary zinc, *Lancet* 1:923-924.
- Smithells, R.W., Sheppard, S. and Schorah, C.J. (1976), Vitamin deficiencies and neural tube defects, *Archives of Diseases in Children* 56:911-918.
- Smithells, R.W., Nevin, N.C., Seller, M.J., Sheppard, S. et al. (1983), Further experience of vitamin supplementation for prevention of neural tube defects recurrences, *Lancet* 1:1027-1031.
- Smyth, R.B. (1878), *The Aborigines of Victoria*, 2 Volumes, Government Printer, Melbourne.
- Solomons, N.W. (1980), Zinc and copper in human nutrition, In: Karcioğlu, Z.A. and Sarper, R.M. (eds), *Zinc and copper in medicine*, pp. 224-275, Charles C. Thomas, Springfield, Illinois.
- Sommer, A., Tarwotjo, I., Djunaedi, E., West, K., Loeden, A. and Tilden, R., (1986), Impact of vitamin A supplementation on childhood mortality: a randomised controlled community trial, *Lancet* 1:1169-73.
- Soong, F.S. (1975), Health education as a means of improving Aboriginal health: A study of the nurse's role, *Medical Journal of Australia (Special Supplement)* 1:4-8.
- Soong, F.S. (1976), Health education approaches in Aboriginal Communities in the Northern Territory- What we have learned, *Medical Journal of Australia (Special Supplement)* 2:1-5.
- Soong, F.S. (1979), Developing the role of Primary Health Workers (Aboriginals), in the Northern Territory- A challenge to the Health Profession, *New Doctor* 11:29-31.
- Soong, F.S. (1981), *Cultural brokers in Western Arnhem Land: A study of the role of the Aboriginal Health Worker*, Unpublished Diploma of Anthropology thesis, University of Sydney.
- South Australian Government (1880-1901), *Government Resident's Report*, Government Printers, Adelaide.
- Spargo, R.M. (1975), Aboriginal communities in remote Australia, Health care delivery-A doctor's role, *Medical Journal of Australia (Special Supplement)* 1:1-3.
- Specht, R.L. (1958a), An introduction to the ethnobotany of Arnhem Land, In: Specht, R.L. and Mountford, C.P. (eds), *Records of the American-Australian Scientific Expedition to Arnhem Land, Volume 3: Botany and Plant Ecology*, MUP, Melbourne.
- Specht, R.L. (1958b), The climate, geology, soils and plant ecology of the northern portion of Arnhem Land, In: Specht, R.L. and Mountford, C.P. (eds), *Records of the American-Australian Scientific Expedition to Arnhem Land. Vol. 3. Botany and Plant Ecology*, pp 333-414, MUP, Melbourne.
- Spencer, B. (1928), *Wanderings in wild Australia*, Macmillan, London.
- Spencer, B. and Gillen, F.J. (1899), *The native tribes of Central Australia* Macmillan, London.
- Spencer, B. and Gillen, F.J. (1904), *The Northern tribes of Central Australia*, Macmillan, London.
- Spencer, B. and Gillen, F.J. (1969), *The native tribes of Central Australia*, Anthropological publications, (Facsimile Edition).
- Spencer, W.B. (1914), *Native Tribes of the Northern Territory of Australia*, McMillan, London.

- Sporn, M.B. and Roberts, A.B. (1983), Role of retinoids in differentiation and carcinogenesis, *Cancer Research* 43:3034-3040.
- Stacy, S.J.G. (1975), Nutritional integration of the Aborigine, *Food and Nutrition Notes and Reviews* 32:163-168.
- Stacy, S.J.G. (1977), *Who should learn what? Health education amongst traditionally orientated Australian Aborigines*, Paper presented at 7th International Congress of Dietetics.
- Stamler, J., Wentworth, D. and Neaton, J.D. (1986), Is relationship between serum cholesterol and risk of premature death from coronary heart disease continuous and graded? Findings in 356,222 primary screened of the Multiple Risk Factor Intervention Trial (MRFIT), *Journal of the American Medical Association* 256:2823-2828.
- Stanik, S. and Marcus, R. (1980), Insulin secretion improves following dietary control of plasma glucose in severely hyperglycaemic obese patients, *Metabolism* 29:346-350.
- Stanley, D. (1976), Aboriginal communities on cattle stations in the Northern Territory of Australia, *Australian Economic Papers*.
- Stanner, W.E.H. (1933), The Daly River Tribes: A report of fieldwork in North Australia, *Oceania* 3:377-405.
- Stanner, W.E.H. (1937), Aboriginal modes of address and reference in the North-West of the Northern Territory, *Oceania* 7:300-315.
- Stanner, W.E.H. (1960), Durmugan, A. Nangiomeri. In: Casagrande, J.B. (ed), *In the company of man*, Harper, New York.
- Stanner, W.E.H. (1974), Some Aspects of Aboriginal health, In: Hetzel, B.S. et al. (eds), *Better health for Aborigines?*, pp. 3-14, UQP, St. Lucia.
- Stanton, K.G., McCann, V., Knuiiman, M., Constable, I.J. and Welborn, T. (1985), Diabetes in part-Aborigines of Western Australia, *Diabetologia* 28:16-21.
- Stevens, F. (1974), *Aborigines in the Northern Territory cattle industry*, ANU Press, Canberra.
- Stirling, E.C. (1896), The Horn Scientific expedition, Part IV.
- Stockley, L. (1984), Direct measurement of intake. In: *The dietary assessment of populations*, MRC Scientific Reports, No.4, pp. 1-4, MRC Environmental Epidemiology Unit, Southampton.
- Strehlow, T.G.H. (1947), *Aranda Traditions*, MUP, Melbourne.
- Strehlow, T.G.H. (1965), Culture, social structure, and environment in Aboriginal Australia. In: (Berndt, R.M. and Berndt, C.H. eds), *Aboriginal man in Australia*, pp. 121-45, Angus and Robertson, Sydney.
- Strehlow, T.G.H. (1971), *Songs of Central Australia*, Angus and Robertson.
- Stryker, S., Kaplan, L., Stein, E.A., Stampfer, M.J., Sober, A. and Willett, W.C. (1988), The relation of diet, cigarette smoking, and alcohol consumption to plasma β -carotene and α -tocopherol levels, *American Journal of Epidemiology* 127:283-296.
- Sturt, C. (1969), *Narrative of an expedition into Central Australia during the years 1844*, 2 Volumes, Greenwood Press, New York.
- Sullivan, H, Gracey, M and Hevron, V. (1987), Food costs and nutrition in remote areas of Northern Australia, *Medical Journal of Australia* 147:334-337.
- Sunderman, J. and Dyer, H. (1984), Chronic ear disease in Australian Aborigines, *Medical Journal of Australia* 140:708-711.
- Suskind, R.M. (1980), Immune status of the malnourished host, *Journal of Tropical Paediatrics* 2:1-6.
- Sweeney, G. (1947), Food supplies of a desert tribe, *Oceania* 17:289-99.
- Sykes, B. (1977), An analysis of the difficulties confronting Aboriginal community-controlled nutrition programs, *Proceedings of the Nutrition Society of Australia* 2:32-36.
- Tarwotjo, I., Sommer, A., West, Jr., K.P., Djundaedi, E., Mele, L. and Hawkins, B. (1987), Influence of participation on mortality in a randomised trial of vitamin A prophylaxis, *American Journal of Clinical Nutrition* 45:1466-1471.
- Tatz, C. (1974), Innovation without change, In: Hetzel, B.S. et al (eds), *Better health for Aborigines?*, pp. 107-120, UQP, St. Lucia.
- Taylor, J.C. (1977), A precontact Aboriginal medical system on Cape York Peninsula, *Journal of Human Evolution* 6:419-432.
- Taylor, J.C. (1979), Diet, health and economy: some consequences of planned social change in an Aboriginal community, In: Berndt, R.M. (ed), *Aborigines and Change: Australia in the '70s*, Social Anthropology Series No. 11, AIAS, Humanities Press Inc, New Jersey, USA.
- Thom, T.J., Epstein, F.H., Feldman, J.J. and Leaverton, P.E. (1985), Trends in total mortality and mortality from heart disease in 26 countries from 1950-1978, *International Journal of Epidemiology* 14:510-520.
- Thomas, S. and Cordon, M. (1970), *Tables of composition of Australian foods*, CDH, AGPS, Canberra.
- Thomas, S. and Cordon, M. (1977), *Metric tables of composition of Australian foods*, AGPS, Canberra.
- Thompson, D.F. (1939), The seasonal factor in human culture: illustrated from the life of a contemporary nomadic group, *Proceedings of the Prehistory Society* 5:209-21.
- Thompson, D.F. (1948), Arnhem Land: Explorations amongst an unknown people. Part 1. *Geographical Journal* 112:146-164.
- Thompson, D.F. (1949a), Arnhem Land: Explorations amongst an unknown people. Part 2. *Geographical Journal* 113:1-8.

- Thompson, D.F. (1949b), Arnhem Land: Explorations amongst an unknown people. Part 3. *Geographical Journal* 114:53-67.
- Thompson, D.F. (1949c), *Economic structure and the ceremonial exchange cycle in Arnhem Land*, McMillan, Melbourne.
- Thompson, D.F. (1962), The Bindibu expedition: exploration among the desert Aborigines of Western Australia, *Geographical Journal* 128:1-14, 143-157, 262-278.
- Thompson, D.F. (1975), *Bindibu country*, Thomas Nelson, Melbourne.
- Thompson, J.E. (1984), Aboriginal health (letter), *Medical Journal of Australia* 140:738.
- Thomson, N. (1982), Aboriginal nutrition: the need for a cultural and historical perspective, *Proceedings of the Nutrition Society of Australia* 7:20-29.
- Thomson, N. (1983), Aboriginal infant mortality, 1976-1981, *Australian Aboriginal Studies* 1:10-15.
- Thomson, N. (1984a), Aboriginal health-current status, *Australia and New Zealand Medical Journal*, 14:705-718.
- Thomson, N. (1984b), Australian Aboriginal health and health-care, *Social Science and Medicine* 18:939-948.
- Thomson, N. (1984c), The role of zinc in Aboriginal nutrition, *Aboriginal Health Information Bulletin* 5:10-14.
- Thomson, N. (1985), Review of available Aboriginal mortality data 1980-1982, *Medical Journal of Australia (Special Supplement)*, 143(9):46-49.
- Thomson, N. (1991), Recent trends in Aboriginal mortality, *Medical Journal of Australia* 154:235-239.
- Thorburn, A.W., Brand, J.C. and Truswell, A.S. (1987a), Slowly digested and absorbed carbohydrate in traditional bushfoods: A protective factor against diabetes?, *American Journal of Clinical Nutrition* 45:98-106.
- Thorburn, A.W., Brand, J.C., O'Dea, K., Spargo, R.M. and Truswell, A.S. (1987b), Plasma glucose and insulin responses to starchy foods in Australian Aborigines: A population now at high risk of diabetes, *American Journal of Clinical Nutrition* 46:282-285.
- Till, M.R. (1978). Development and use of limited and saline water supplies, In: Hetzel, B.S. and Frith, H.J. (eds), *Symposium on the nutrition of Aborigines in relation to the ecosystem of Central Australia*, pp. 127-138, CSIRO, Melbourne.
- Tilley, E. (1974). Prevention of undernutrition in Aboriginal children, *Australian Nurses Journal*, 4:37,40.
- Tindale, N. (1925), Natives of Groote Eylandt and of the west coast of the Gulf of Carpentaria, *Records of the South Australian Museum* 3:61-134.
- Tindale, N. (1953), On some Australian cossidæ including the moth of the witjuti (witchetty), grub, *Transactions of the Royal Society of South Australia* 76:56-65.
- Tindale, N. (1972), The Pitjandjara. In: Bicchieri, M.G. (ed), *Hunters and gatherers today*, Holt, Rinehart and Winston.
- Tindale, N.B. (1974), *Aboriginal tribes of Australia*, ANU Press, Canberra.
- Tonkinson, R. (1974), *The Jigalong mob: Aboriginal victors of the desert crusade*, Cummings, Menlo Park, California.
- Trowell, H. (1981), Hypertension, obesity, diabetes mellitus and coronary heart disease, In: Trowell, H.C. and Burkitt, D.P. (eds), *Western diseases: their emergence and prevention*, pp. 3-32, Edward Arnold, London.
- Trowell, H. and Burkitt, D. (1981), Contributor's reports. In: Trowell, H.C. and Burkitt, D.P. (eds), *Western diseases: their emergence and prevention*, pp. 427-436, Edward Arnold, London.
- Trulson, M.F. and McCann, M.B. (1959), Comparison of dietary survey methods, *Journal of the American Dietetic Association* 35:672-676.
- Truswell, A.S. (1976), Diet and nutrition of hunter-gatherers, In: Elliot, K. and Whelan, J. (eds), *Health and Disease in Tribal Societies*, pp. 213-221, Ciba Foundation Symposium No. 49, Elsevier, Amsterdam.
- Truswell, A.S. (1981), Anthropometric assessment with heights and weights, *Transactions of the Menzies Foundation* 3:59-71, The Menzies Foundation, Melbourne.
- Truswell, A.S. (1983), The development of dietary guidelines, *Food Technology in Australia* 35:498-502.
- Truswell, A.S. (1984a), Folate, *Journal of Food and Nutrition* 41:143-54.
- Truswell, A.S. (1984b), End of static decade for coronary disease, (Editorial) *British Medical Journal* 289:509-510.
- Truswell, A.S. (1985), ABC of nutrition: Measuring nutrition, *British Medical Journal* 291:1258-1262.
- Truswell, A.S. (1990), Folate, In: Truswell, A.S., Dreosti, I.E., English, R.M., Rutishauser, I.H.E. and Palmer, N., *Recommended nutrient intakes, Australian papers*, pp. 96-110, Australian Professional Publications, Sydney.
- Truswell, A.S., Dreosti, I.E., English, R.M., Rutishauser, I.H.E. and Palmer, N., *Recommended nutrient intakes, Australian papers*, Australian Professional Publications, Sydney.
- Turner, D.H. (1974), *Tradition and transformation- A study of Aborigines in the Groote Eylandt area*, AIAS, Canberra.
- UPK, (1987), *Report of Uwankara Palyanyku Kanyintjaku An environmental and public health review within the Anangu Pitjantjara Lands*, Nganampa Health Council, South Australian Health Commission, Aboriginal Health Organisation of SA.
- USDHEW, (1972). *Food composition table for use in East Asia*, USDHEW Publication, Washington, D.C.

- USDHHS, (1988), *The Surgeon General's Report on Nutrition and Health*, Public Health Service, US Government Printing Office, Washington.
- Valliappan, V. (1986), Alcohol and pregnancy outcomes; A pilot study in Aboriginal communities, Unpublished MM Thesis, University of Sydney.
- van Buynder, P. (1991), *Epidemiology of renal disease in Australian Aborigines*, Unpublished MPH thesis, University of Sydney.
- Vanderwal, R. (1982), *The Aboriginal photographs of Baldwin Spencer*, John Curry, O'Neil Publishers, South Yarra, Victoria.
- van Dongen, R., Davignons, V. and Abbie, A.A. (1962), Aboriginal blood pressures at Beswick, south-west Arnhem Land, and correlation with physical dimensions, *Medical Journal of Australia* 2:286-289.
- van Staveren, W.W., and Burema, J. (1982), Comments on the use of terms to qualify dietary survey methods, Validity of dietary survey methods: a critical approach, In: Hautvast, G.A.J. and Klaver, W. (eds), *A concerted action project on nutrition in the European community: The diet factor in epidemiological research*, EURONUT Report 1, pp. 86-92, Wageningen.
- Vesper, J. (1987), *The interviewer's story*, Aboriginal Family Demography study, Working paper No. 6, Department of Demography, Research School of Social Sciences, ANU.
- von Sturmer, J. (1981), Talking with Aborigines, *AIAS Newsletter*, 15:13-30.
- WHO, (1954), Expert committee on Health Education of the Public: First Report, *WHO Technical report series* 89:22.
- WHO, (1985), Diabetes Mellitus, *WHO Technical report series*, 727:10-12.
- WHO, (1986a), European collaborative trial of multifactorial prevention of coronary heart disease, Final report on the 6-year results, *Lancet* 1:869-872.
- WHO, (1986b), *Guidelines for training community health workers in nutrition*, Second edition, WHO, Geneva.
- Waddy, J. (1986), Classification of food from a Groote Eylandt Aboriginal point of view, In: Manderson, L. (ed), *Shared wealth and symbol: Food culture and society in South East Asia and the South Pacific*, pp. 144-165, Cambridge University Press.
- Wadsworth, G. (1984), *The diet and health of isolated populations*, CRC Press, Florida.
- Walker, A.C. and Harry, J.G. (1972), A survey of diarrhoeal disease in malnourished Aboriginal children, *Medical Journal of Australia* 1:904-911.
- Walker, A.C., Brocklehurst, J. and Rynn, M. (1982), Aboriginal child health and nutrition, *Transactions of the Menzies Foundation* 4:179-182, The Menzies Foundation, Melbourne.
- Walker, R.B. and Roberts, D.C.K. (1988), Colonial food habits, In: Truswell, A.S. and Wahlqvist, M.L. (eds), *Food habits in Australia*, Proceedings of the first Deakin/Sydney Universities symposium on Australian nutrition, pp. 40-59, Victoria.
- Wardlow, H. and Lawrence, W. (1932), Further observations on the basal metabolism of Australian Aborigines, *Australian Journal of Experimental Biology and Medical Science* 5:263-272.
- Warner, L. (1958), *A black civilisation*, Harper and Bros., New York. (Revised Edition)
- Waterford, J. (1982), A fundamental imbalance: Aboriginal ill-health, In: Reid, J. (ed), *Body, land and spirit: Health and healing in Aboriginal society*, pp 8-31, UQP, St. Lucia.
- Watsford, S.D. (1955), Anaemia amongst natives in the Northern Territory, *Medical Journal of Australia* 2:684.
- Watson, C, Flemming, J. and Alexander, K. (1988), *A survey of drug use pattern in Northern Territory Aboriginal Communities: 1986-87*, NTDHCS, Darwin.
- Watson, D.S. and Tozer, R.A. (1986), Anaemia in Yirrkala, *Medical Journal of Australia (Special Supplement)*, 144:513-15.
- Webb, L.J. (1969), The use of plant medicines and poisons by Australian Aborigines, *Mankind* 7:137-146.
- Webb, L.J. (1973), "Eat, die, and learn"- the botany of the Australian Aborigines, *Australian Natural History* 17:290-295.
- Webb, T.T. (1933), Aboriginal medical practice in East Arnhem Land, *Oceania* 4:91-98.
- Welborn, T.A., Cumow, D.H., Wearne, J.T., Cullen, K.J., McCall, M.G. and Stenhouse, N.S. (1966), Diabetes detected by blood sugar measurement after a glucose load: report from the Busselton survey, 1966, *Medical Journal of Australia* 2:778-783.
- Welborn, T.A., Cumpston, G.N., Cullen, K.J., Cumow, D.H., McCall, M.G. and Stenhouse, N.S. (1969), The prevalence of coronary heart disease and associated factors in an Australian rural community, *American Journal of Epidemiology* 89:521-536.
- Werner, D.B. (1977), The village health worker: Lackey or Liberator, In: *Proceedings of the international Hospital Federation Congress*, Tokyo, Japan, Hesperian Foundation, Palo Alto, U.S.A.
- White, C. (1969), *Report on field trip to Caledon Bay, Northern Territory December and January 1968-9*, Document No. 69/816, AIAS, Canberra.
- White, I.M. (1975), Sexual conquest and submission in Aboriginal myths. In: L.Hiatt (ed), *Australian Aboriginal Mythology*, AIAS, Canberra.
- White, I.M. (1977), Pitfalls to avoid: the Australian experience, In: Elliot, K. and Whelan, J. (eds), *Health and Disease in Tribal Societies*, pp. 269-292, Ciba Foundation Symposium No. 49, Elsevier, Amsterdam.
- White, I.M., and O'Connell, J.F. (1979), Australian prehistory: new aspects of antiquity, *Science* 203:21.
- White, K., Gracey, M., Schumacher, L., Spargo, R. and Kretchmer, N. (1990), Hyperinsulinaemia and impaired

- glucose tolerance in young Australian Aborigines *Lancet* 335:735.
- White, N.G. (1978), A human ecology research project in the Arnhem Land region: an outline, *ALAS Newsletter* 9:39-52.
- White, N.G. (1985), Sex differences in Australian Aboriginal subsistence: possible implications for the biology of hunter-gatherers, In: Newcombe, F., Ghesquierre, J., and Martin, R. (eds), *Human sexual dimorphism* pp. 323-361, Taylor and Francis, London.
- White, S.A. (1915), Aborigines of the Everard Range, *Transactions of the Royal Society of South Australia* 39:725-731.
- Whitfield, J.B. (1981), Alcohol related biochemical changes in heavy drinkers, *Australian and New Zealand Journal of Medicine* 11:132-139.
- Widdowson, E.M. (1936), A study of English diets by the individual method, 1: Men, *Journal of Hygiene* 36:269-292.
- Widdowson, E.M. (1947), A study of individual children's diets, *MRC Special Report Series*, No. 257, HMSO, London.
- Widdowson, E.M. and McCance, R.A. (1936), A study of English diets by the individual method, 2: Women, *Journal of Hygiene* 36:263-309.
- Widdowson, E.M. and McCance, R.A. (1943), Food tables: their scope and limitations, *Lancet* i:230-232.
- Wiehl, D.G. and Reed, R. (1960), Development of new or improved dietary methods for epidemiological investigations, *American Journal of Public Health* 50:824-828.
- Wilhelmsen, L. (1984), Risk factors for coronary heart disease in perspective *American Journal of Medicine* 76:37-40.
- Wilkinson, G.K., Day, A.J., Peters, J.A. and Casley-Smith, J.R. (1958), Serum proteins of some Central and South Australian Aborigines, *Medical Journal of Australia* 2:158-160.
- Willett, W.C. (1990), *Nutritional epidemiology*, Oxford University Press, New York.
- Willett, W.C., Stampfer, M.J., Underwood, B.A., Taylor, J.O. and Hennekens, C.H. (1983), Vitamins A, E, and carotene: effects of supplementation on their plasma levels, *American Journal of Clinical Nutrition* 38:631-639.
- Willett, W.C., Polk, B.F., Underwood, B.A., Stampfer, M.J., Pressel, S., Rosner, B., Taylor, J.O., Schneider, K. and Hames, C.G. (1984), Relation of serum vitamins A and E and carotenoids to the risk of cancer, *New England Journal of Medicine* 310:430-434.
- Willett, W.C., Sampson, L., Stampfer, M.J. *et al* (1985), Reproducibility and validity of a semiquantitative food frequency questionnaire, *American Journal of Epidemiology* 122:51-65.
- Willett, W.C., Reynolds, R.D., Cottrell-Hoehner, S., Sampson, L. and Brown, M.L. (1987), Validation of a semiquantitative food frequency questionnaire: comparison with a one-year diet record, *Journal of the American Dietetic Association* 87:43-47.
- William, K.W., Lorr, N.A. and young, C.S. (1984), Simultaneous determination of plasma retinol, a-tocopherol, lycopene, α -carotene and β -carotene by high performance liquid chromatography, *Annals of Chemistry* 138:340-345.
- Williams, D.R.R., Moffitt, P.S., Fisher, J.S. and Bashir, H.V. (1987), Diabetes and glucose tolerance in New South Wales coastal Aborigines: possible effects of non-Aboriginal genetic admixture, *Diabetologia* 30:72-77.
- Williams, L.R., Casali, J. and Horne, V. (1981), Evaluation of some Australian plant species for potential as hydrocarbon-producing crops, *Chemistry in Australia* 48:344-345.
- Wilson, P.W., McGee, D.L. and Kannel, W.B. (1981), Obesity, very low density lipoproteins and glucose tolerance over 14 years: the Framingham study, *American Journal of Epidemiology* 114:697-704.
- Wilson, W.E. (1953), A dietary survey of Aborigines in the Northern Territory, *Australian Medical Journal* 1:536-537.
- Wilson, W.E. (1954), The food consumption of Australian Aborigines: natural versus civilised diets, *Australian Medical Journal* 2:599-605.
- Wiminydji and Peile, A.R. (1978), A desert Aboriginal view of health and nutrition, *Journal Anthropological Research* 34:497-523.
- Winterbottom, H.J. (1961), Diabetes mellitus at Mabuag Island, Torres Strait, 1960, *Medical Journal of Australia* 1:780-781.
- Wise, P.H., Edwards, F.M., Thomas, D.W., Elliot, R.B., Hatcher, L. and Craig, R.J. (1970), Hyperglycaemia in the urbanised Aboriginal - The Davenport survey, *Medical Journal of Australia* 2:1001-1006.
- Wise, P.H., Edwards, F.M., Craig, R.J., Evans, B., Murchland, J.B., Sutherland, B. and Thomas, D.W. (1976), Diabetes and associated variables in the South Australian Aboriginal, *Australian and New Zealand Journal of Medicine* 6:191-196.
- Wood, B. (1985), Thiamine status in Australia, *World Review of Nutrition and Dietetics* 46:148-218.
- Wood, B. (1990), Thiamine, In: Truswell, A.S., Dreosti, I.E., English, R.M., Rutishauser, I.H.E. and Palmer, N. (1990), *Recommended nutrient intakes*, Australian papers, pp. 51-64, Australian Professional Publications, Sydney.
- Wood, B. and Breen, K.J. (1980), Clinical thiamine deficiency in Australia: the size of the problem and approaches to prevention, *Medical Journal of Australia* 1:461.

- Woodburn, J. (1980), Hunters and gatherers today and reconstruction of the past, In: E.Geller (ed), *Soviet and Western Anthropology*, Duckworth, London.
- Woodhill, J.M. and Nobile, S. (1971), Vitamin C (l-ascorbic acid and dehydro-l-ascorbic acid), *Medical Journal of Australia* 1:1009.
- Woods, J.D. (1966), The electrocardiogram of the Australian Aborigines, *Medical Journal of Australia* 1:438-441.
- Woodward, D, MacPhail, M. and King, H. (1986), *Diet and Health of the Tasmanian Aborigines- The perspective of a thousand generations*, Unpublished manuscript.
- Woolf, B.C. (1954), Statistical aspects of dietary surveys, *Proceedings of the Nutrition Society of Australia* 12:82-94.
- Worsley, A. (1981), Psychometric aspects of language development techniques in dietary assessment, *Transactions of the Menzies Foundation* 3:161-194, The Menzies Foundation, Melbourne.
- Worsley, A., Baghurst, K.I. and Leitch, D.R. (1984), Social desirability, response bias and dietary inventory responses, *Human Nutrition, Applied Nutrition* 38:29-35.
- Worsley, P.M. (1961), The utilisation of natural food resources by an Australian Aboriginal tribe, *Acta ethnographica* 10:1-2, 158-189.
- Wynder, E.L. (1976), Nutrition and cancer, *Federation Proceedings* 35:1309-1315.
- Yengoyan, A.A. (1976), *Structure, event and ecology in Aboriginal Australia*, In: N. Peterson (ed), *Tribes and Boundaries in Australia*, pp. 121-32, AIAS, Canberra.
- Youmans, J.B., Patton, E.W. and Kern, R. (1942), Surveys of the nutrition of populations, *American Journal of Public Health* 32:1371-1379.
- Young, C.M. and Trulson, M.F. (1960), Methodology for dietary studies in epidemiological surveys, 2: Strengths and weaknesses of existing methods, *American Journal of Public Health* 50:803-814.
- Young, C.M., Hagan, G.C., Tucker, R.E. and Foster, W.D. (1952), A comparison of dietary study methods, 2: Dietary history vs seven-day record vs 24-hour recall, *Journal of the American Dietetic Association* 28:218-221.
- Young, E. (1981), *Tribal communities in rural areas*, Development Studies Centre, ANU, Canberra.
- Young, E. (1984), *Outback stores: retail services in North Australian Aboriginal communities*, ANU, NARU, Darwin.
- Zador, D.A., Rassaby, L. and Truswell, A.S. (1990), Nutritional status of institutionalised elderly people: A study in the Blue Mountains, NSW, *Australian Journal of Nutrition and Dietetics* 47:20-27.
- Zimmet, P. (1986), The diagnosis of diabetes mellitus, *Medical Journal of Australia* 145: 88.
- Zimmet, P.Z., King, H.O.M. and per Bjorntorp, S. A. (1986), Obesity, hypertension, carbohydrate disorder and the risk of chronic diseases. *Medical Journal of Australia* 145: 256-262.

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