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# Nutritional assessment of patients with type 1 Diabetes Mellitus

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# NUTRITIONAL ASSESSMENT OF PATIENTS WITH TYPE 1 DIABETES MELLITUS

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## Statement

I hereby declare that this thesis is my own work. References to the work of others are indicated in the text. This study has not been previously submitted for a degree to any other university or institution.

Farideh Tahbaz

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# Summary

Type 1 diabetes mellitus is a chronic metabolic disorder affecting both young and old. Treatment goals are becoming standardised, but the degree to which patients with type 1 diabetes meet their goals in practice is uncertain.

The aim of this study was to evaluate the nutrition-related outcomes of patients with type 1 diabetes diagnosed since 1984 in the Illawarra and at least one year post-diagnosis. Subjects were recruited from the list of patients registered at the Diabetes Education Unit (DEU) of the Illawarra Area Health Service.

All patients were aged between 18-40 years. For the purpose of comparison a similar nondiabetic group who were either relatives/friends of patients or volunteers was recruited. In these two groups, anthropometric measurements, blood pressure, glycosylated hemoglobin (HbA1c%), lipids, urine albumin: creatinine ratio, dietary intake as well as quality of life were assessed.

Dietary intake of subjects was assessed by a meal-based diet history (Burke method), and food pattern and food preparation questionnaires (based on validated DCCT questionnaires). The results were analysed using Diet 1 (V4), which uses the Australian NUTTAB 1995 food composition database. The findings were compared to the Australian and American dietary goals and recommendations.

In total 55 patients (18 female and 37 male) and 47 controls (17 female and 30 males) were seen. The means of body measurements including body mass index (BMI), waist to hip ratio (WHR) and body fat%, blood pressure and blood lipids and lipoproteins were within normal range and not significantly different between the two groups. However blood pressure was higher in overweight subjects in both groups. Also obesity or overweight was associated with an adverse lipid and lipoprotein pattern. Plasma fibrinogen concentration in people with type 1 diabetes was significantly higher than in control subjects. Higher plasma fibrinogen levels were associated with higher BMI and elevated plasma triglyceride and decreased HDL cholesterol concentrations.

The albumin: creatinine ratios in spot and early morning urine samples of patients with type 1 diabetes were significantly higher than these ratios in the control group. The negative relationship of early morning urine sample with HDL cholesterol was noted.

In this study the degree of glycemic control was evaluated by HbA1c%. Mean values were 8.52% (SD 2.21) and 8.56% (SD 1.91) for females and males respectively, higher than in the DCCT intensively treated patients and lower than in

the DCCT conventionally treated patients. A number of variables including anthropometric measurements, blood pressure, diabetes history, blood and urine results were evaluated as independent variables in several models with HbA1c% in the diabetic group. No statistically significant associations were found. It was shown however that those with the least education had the poorest diabetic control but this also was not found to be significant.

The dietary intake results indicated that only starch intake (in males and females) and dietary fibre intake (in males) were higher among patients than in controls when the macronutrient and alcohol intakes were compared in two groups. None of the same sex comparisons in diabetic and control subjects showed a significant difference in regard to the energy intake and the contribution of macronutrients. No associations were found between dietary intakes and glycemic control in diabetic subjects.

The findings were compared to the recommended dietary intakes (RDI). Significantly more subjects with diabetes than controls reached the guidelines for fat consumption, however the majority of those with diabetes consumed saturated fatty acids at a level greater than the recommendations. Protein intake of patients with type 1 diabetes in terms of grams per body weight and its contribution to total energy intake was higher than recommended. The average carbohydrate (energy%) was similar in both groups and it was lower than the recommendations. Their micronutrient intake for both diabetic and control subjects were higher than the recommendations. Sodium intake in diabetic subjects was higher than in controls.

The overall diet quality of the subjects was scored on the basis of several dietary recommendations. It was found that the higher the diet score (ie the more closely the diet adhered to the recommendations) the better the glycemic control but this association was not significant. Educated people in both groups had higher diet scores. Also in regard to consumption of basic food groups, it was found that diabetic subjects had more fruit than controls did. Both groups did not have legumes and nuts (low glycemic index foods) in their diets regularly.

Not all the patients complied with their dietary prescriptions. Those with better dietary adherence had better glycemic control, although the correlation did not reach significance level.

Quality of life of patients and controls was evaluated with the Diabetes Quality of Life Measure (applied in the DCCT) and SF-36 (a tool which measures non-disease-specific aspects of quality of life). Our results were comparable with those from the DCCT, showing neither group of patients had severe complications. Diabetes worry subscale on the Diabetes Quality of Life Measure was associated

negatively with the duration of diabetes and HbA1c%. The result of SF-36 subscales were similar to controls, in both groups these subscales were inversely related to either BMI and/or WHR.

The results of this study highlights the possibility of improving diabetic control and as a result preventing the related complications in this group of patients by nutritional management. However caution should be taken in generalizing from these results, given the relatively small sample size. An up to date frequency counting of this disease is recommended. Then larger scale studies that involve the majority of the patients in this area and/or nutrition intervention programs (like nutrition education) are needed to confirm the generalisability of this study.

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## Abbreviations

| | |
|---|---|
| BMI | Body Mass Index |
| CHO | Carbohydrate |
| CVD | Cardiovascular disease |
| DCCT | Diabetes Control and Complications Trial |
| DKA | Diabetic ketoacidosis |
| DQOL | Diabetes Quality of Life Measure |
| dBP | Diastolic blood pressure |
| HbA1c% | Glycosylated hemoglobin |
| IDDM | Insulin-dependent diabetes mellitus |
| MUFA | Monounsaturated fatty acids |
| M/S | Monounsaturated to saturated fatty acids |
| NIDDM | Noninsulin Dependent Diabetes Mellitus |
| PUFA | Polyunsaturated fatty acids |
| P/S | Polyunsaturated to saturated fatty acids |
| RDI | Recommended Dietary Intake |
| SAFA | Saturated fatty acids |
| sBP | Systolic blood pressure |
| WHR | Waist to hip ratio |
| WHO | World Health Organization |

# Chapter 1

# Introduction

This thesis is about some aspects of the nutritional management of type 1 diabetes mellitus. Type 1 diabetes also known as insulin dependent diabetes mellitus (IDDM) is one of the most important and serious diseases involving young people. It is a chronic disease primarily involving glucose metabolism. The disease markedly increases the risk of morbidity and mortality from various micro- and macrovascular complications.

The Australian incidence rate is found to be between the rates from England and New Zealand. The people in these countries have similar ethnic and genetic backgrounds. But compared with most countries in the world (the highest rate found in Finland and the lowest rate in Japan), Australia has very high rates of childhood type 1 diabetes (incidence rate of 14.5 per 100,000 person per year with prevalence of 361/100,000 (1990-1991) in NSW reported by Verge et al. 1994a). Type 1 diabetes accounts for approximately 10% to 20% of all diabetes mellitus in the western world (Ludwig-Beymer et al. 1996, Karam et al. 1991).

Multiple factors contribute to the genesis of type 1 diabetes. It has become clear that it is caused by an autoimmune process which destroys pancreatic beta-cells resulting in the loss of insulin production (Green et al. 1992, Karvonen et al. 1993, Watkins et al. 1996). The potential causes of β cell destruction include a genetic predisposition to the disease, and exposure to environmental triggers that may activate mechanisms leading to a progressive loss of beta cells and insulin production (Walker and Cudworth 1980, Barnett et al. 1981, Dosch et al. 1992, Copstead 1995, Watkins et al. 1996). Between 5-8% of individuals with newly diagnosed type 1 diabetes have a first degree relative

(parent or sibling) with type 1 diabetes (Atkinson and MacLaren 1994, Copstead 1995). The risk to children whose father has type 1 diabetes is double that if the mother has type 1 diabetes (El-Hashimy et al. 1995).

Environmental factors may include viral infection and dietary factors (Porth 1994). A number of common human viruses including mumps virus, coxsackie B3 and B4 viruses and reovirus type 3 may induce diabetes by different mechanisms (Yoon 1990, Watkins et al. 1996). Early exposure to cow's milk protein may be an important factor in initiation of the β cell destruction (Daneman et al. 1987, Scott et al. 1988, Scott 1990), while breast-feeding may be protective, (Metcalfe and Baume 1992, Norris Kostraba et al. 1992) but these effects have not been clearly indicated.

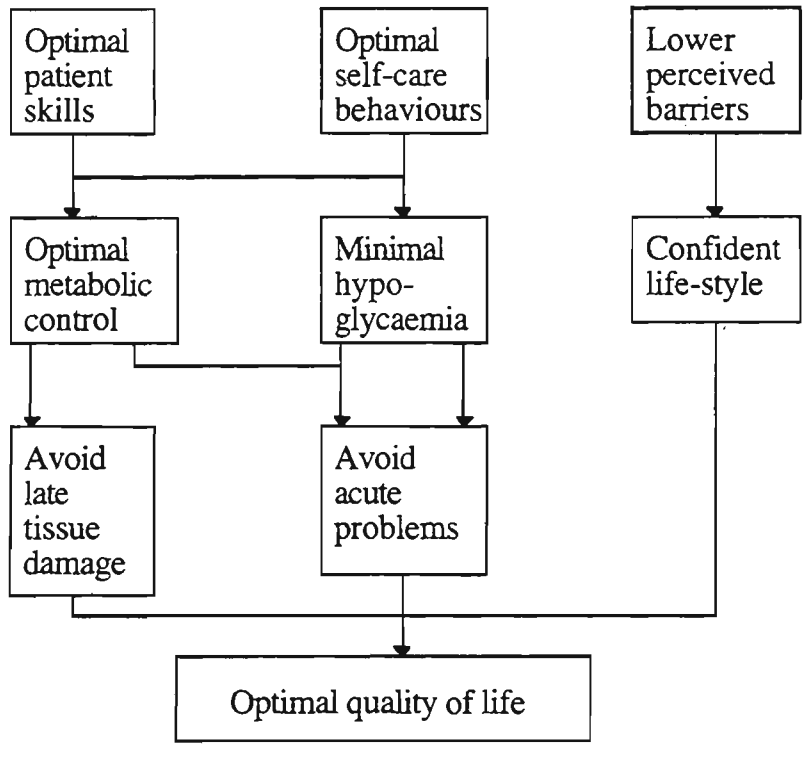
Current research indicates that poor metabolic control of type 1 diabetes is eventually associated with serious complications. Recently the Diabetes Control and Complications Trial (DCCT) conclusively demonstrated that intensive therapy reduced the risk of progression and developing diabetes complications in 1441 patients with type 1 diabetes (age 13-39 years). The results show that intensive therapy compared to conventional therapy could reduce the risk of retinopathy, nephropathy and neuropathy by 50-75%. In this trial there was about a threefold increase in the frequency of severe hypoglycemia and an average weight gain of 4.6 kg in the group receiving intensive therapy compared to the group receiving conventional therapy (DCCT Research Group 1993a). In the group receiving intensive therapy macrovascular events was 42% less, the number of events was small and the difference was not statistically significant.

The Pittsburgh Epidemiology of Diabetes Complication Study also showed that in well controlled patients with type 1 diabetes who had been diagnosed for more than 25 years there is an association between glycemic control and avoidance of major complications (Orchard et al. 1990, Lloyd et al. 1996a). They also showed that those with higher education and income tend to have fewer complications.

In other studies like the EURODIAB IDDM Complications Study it was shown that duration of diabetes and poor glycemic control are established risk factors for diabetes complications (EURODIAB IDDM Complications Study Group 1994). In the same group of patients high fibrinogen together with raised blood pressure in subjects with retinopathy and microalbuminuria may have a pathogenic role in arterial disease (Greaves et al. 1997).

The glycemic control of patients with diabetes requires long-term managed treatment. The objectives of diabetes management are described in figure 1 (European IDDM Policy Group 1993) and indicate the interactions between components of care, monitoring of glycemic status, dietary modification and exercise regulation.

**Figure 1- Objectives of diabetes management**




Those who fund our health care system are increasingly concerned to monitor the quality and effectiveness of health care interventions and match them to treatment goals. It is recognized that care must not only be effective but also efficient and any program must be adaptable so that it suits for individual patients

with individual needs and behaviours. One of the goals of WHO Multinational Project for Childhood Diabetes is to collect information on evaluation of the efficiency and effectiveness of health care of patients with type 1 diabetes (WHO DIAMOND Project Group 1990).

To facilitate and potentially improve the effectiveness of diabetes care, the current study was undertaken. This study was carried out on patients with type 1 diabetes (18-40 years) living in the Illawarra area, who have been diagnosed since 1984.

The aims of the study were as follows:

- to examine current management and achievement of management goals for type 1 diabetes by people with type 1 diabetes
- in particular, to measure the achievement of dietary and nutritional objectives by people with type 1 diabetes
- to gain a measure of their quality of life.

To address these aims the study proposes to compare a group of people who have lived with type 1 diabetes for some years with a reference population. These aims were translated into the following research questions:

- What is the glycemic control of these patients with diabetes as measured by HbA1c%?
- Is glycemic control associated with likely determinants such as age, sex, duration of diabetes, socio-economic status?
- Are the diabetic subjects able to maintain optimal levels of known determinants of some of the possible complications, determinants such as weight and measures of fatness, blood pressure, plasma lipid levels, and indicators in blood and urine samples?
- Is there any association between the level of glycemic control and these indicators of potential complications?
- How does dietary intake of diabetic subjects compare to those in

controls? Is there any relation between dietary intake and glycemic control in diabetes?

- What is the dietary practice of diabetic and control subjects when compared to the recommended dietary intake and food group consumption? Is there any relation between dietary adherence and glycemic control in people with diabetes?
- What is the quality of life of patients with type 1 diabetes and how does it compare to a reference group?

It is expected that this study provides useful information for future research in individuals with type 1 diabetes in this area.

# Chapter 2

# Literature Review

## 2.1- Type 1 Diabetes

Diabetes mellitus is not a single disease but a number of disorders with glucose intolerance in common. Historically, diabetes was described as a disease distinguished by weight loss and excessive urination, thirst, and hunger. It was observed in at least two forms, one affecting primarily the obese and another more prevalent among younger, thin individuals.

The term diabetes mellitus now describes a syndrome characterized by chronic hyperglycemia and other disturbances of carbohydrate, fat, and protein metabolism (Bennett 1994, Ludwig-Beymer et al. 1996). The World Health Organization (WHO 1985) has identified four major classes of diabetes mellitus: insulin-dependent diabetes mellitus (IDDM or type 1 diabetes mellitus), non-insulin-dependent diabetes mellitus (NIDDM), malnutrition-related diabetes mellitus (MRDM), and others (Bennett 1994).

Type 1 diabetes also known as juvenile-onset diabetes, ketosis-prone diabetes, or insulin dependent diabetes mellitus, most commonly appears in youth and young adults but can occur at any age (Laakso and Pyorala 1985). The classical symptoms such as thirst, polyuria, wasting, and/or ketoacidosis appear abruptly and insulin treatment is required not only to control the hyperglycemia and symptoms but to prevent the spontaneous occurrence of ketoacidosis (Bennett 1994, Copstead 1995).

## Prevalence and incidence of type 1 diabetes

It has been reported that type 1 diabetes accounts for approximately 10% to 20% of all diabetes mellitus in the western world (Ludwig-Beymer et al. 1996, Karam et al. 1991). Type 1 diabetes occurs most frequently in persons of northern European descent. Among other racial groups, such as blacks, Native Americans and Asians, the disease is less common (Diabetes Epidemiology Research International Mortality Study Group 1991).

The WHO DIAMOND Project Group have reviewed the variation in incidence between continents and showed that the lowest incidences were found in Asia, followed by Oceania, South and North America, and the highest were reported in Europe (Karvonen et al. 1993). It was shown that the incidence ranges from a low of 1 to 2 per 100,000 per year in Japan to a high of more than 40 per 100,000 per year in parts of Finland (Diabetes Epidemiology Research International Group 1988, Green et al. 1992). It has been also reported that the worldwide variation in incidence reflects the distribution of ethnic populations and demonstrates the importance of the differential genetic susceptibility between populations (Karvonen et al. 1993).

### *Prevalence and incidence of type 1 diabetes in Oceania*

In New Zealand the incidence of type 1 diabetes among children appears to be lower in Auckland than in Canterbury. The incidence rate of diabetes in Auckland in Caucasoid children was about 12/100,000 per year (Brown 1993). An earlier study had estimated the average incidence of type 1 diabetes at 11.7/100,000 per annum (1982-1986) among those diagnosed under 20 years (Scott and Brown 1991, Mason et al. 1987). Later it was found that the incidence of type 1 diabetes in subjects less than 20 years of age in Canterbury was higher (19.5/100,000, 1990-1992) (Scott et al. 1992). This high incidence was associated

with a high frequency of the HLA-DQβ non-Asp 57 allele in the Canterbury population (Brown 1993); and the incidence therefore could be attributed to a gene-environment interaction (Forbes et al. 1993).

In Western Australia from 1985 to 1989, the mean age adjusted (developed-world population) annual incidence of type 1 diabetes in children aged 0-14 was 13.2 per 100,000 person-year. There was no evidence of an increasing incidence over the 5 years of the study (Kelly and Byrne 1992).

In New South Wales, over a 2-year period (1990-1991) it has been shown that the age standardized incidence rate of type 1 diabetes in 0-14 year boys and girls was 14.5/100,000 persons per year; this rate is in the middle of the worldwide range. When CIs (95% confidence interval 13.0-16.0) were considered, this was similar to rates found previously in Australia. No significant differences were found when comparing the first and second years of the register, boys and girls, geographical areas, or Aboriginal and non-Aboriginal children (Verge et al. 1994a). Earlier in Sydney, it was shown that the annual incidence of type 1 diabetes per 100,000 population aged 0 to 19 years, rose from 10.3 cases in 1984 to 14.8 cases in 1987 (the increase was not significant) (Sutton et al. 1989).

In the Illawarra area the prevalence of diabetes in 0-19 year patients was 69/100,000, with an incidence of 13/100,000 in 1984 (Moses and Mathews 1986).

## 2.2- Etiology of Type 1 Diabetes

It has been suggested that type 1 diabetes results from either a genetic predisposition (diabetogenic genes), or a hypothetical triggering event that involves an environmental agent that serves to stimulate an immune response, or immunologically mediated β cell destruction (Green et al. 1992, Karvonen et al. 1993, Watkins et al. 1996). It was concluded by others that autoimmune destruction of pancreatic β cells is the most common cause of type 1 diabetes (Kahn and Weir 1994).

The highly significant genetic component is illustrated by the 30-50 per cent concordance rate in identical twins (Walker and Cudworth 1980, Barnett et al. 1981, Dosch et al. 1992, Copstead 1995, Watkins et al. 1996) and the 5-8 per cent prevalence in first degree relatives especially if the affected parent is a father (Atkinson and MacLaren 1994, Copstead 1995, El-Hashimy et al. 1995). It has been shown that the main gene associated with a predisposition to type 1 diabetes is in the major histocompatibility complex (MHC) on chromosome 6, in the region associated with the genes for the highly polymorphic immune-system-recognition molecules known as HLA (Human Leukocyte Antigen). This is where the presence of amino acid aspartic acid at position 57 of the β chain of the DQ molecules appears to carry resistance to type 1 diabetes (Dorman et al. 1990, Atkinson and MacLaren et al. 1994). Susceptibility or resistance to type 1 diabetes is associated with different HLA-DR and DQ genotypes (Todd and Bain 1994). There is a strong association between certain HLA antigens (DR3 and DR4) in Caucasians coded by these immune response genes and type 1 diabetes. Thus, it appears that what is inherited as part of the HLA genotype in type 1 diabetes is a susceptibility to an abnormal immune response that affects β cells (Porth 1994, MacLaren 1988).

The incidence of type 1 diabetes in a transmigratory population (Asians migrated from an area of low incidence of childhood diabetes to England where there was a higher incidence) has been studied. The incidence of type 1 diabetes increased in offspring of that transmigratory population, showing that environmental factors (probably viral infections and possibly climatic condition) are important in the etiology of type 1 diabetes (Bodansky et al. 1992).

Environmental agents that have been associated with altered pancreatic β cell function include viral and dietary triggers (Porth 1994). A number of common human viruses including mumps virus, coxsackie B3 virus, coxsackie B4 virus and reovirus type3 could infect human beta-cells and may trigger diabetes (Yoon 1990,

Watkins et al. 1996). These conditions are supported by several experimental models in which insulinopenic diabetes has been triggered by viruses in animals.

Earlier epidemiological studies have shown that the prevalence of type 1 diabetes among patients with congenital rubella syndrome was significantly higher in both the United States and Australia (about 20%) (Menser et al. 1978), although studies in the United Kingdom have failed to confirm this association (Smithsells et al. 1978).

Evidence for a diabetogenic effect of a milk component has accumulated rapidly including confirmatory observations in animal experiments (Daneman et al. 1987, Scott et al. 1988, Scott 1990). There has been also a long-standing debate about a protective effect of breast-feeding against the initiation of the β cell destructive process in some individuals (Glatthaar et al. 1988, Mayer et al. 1988, Savilhati and Akerblom 1990, Vritranen et al. 1991, Metcalfe and Baum 1992, Norris Kostraba et al. 1992). A number of studies have shown that infants who were fed with breast-milk substitutes before 3 or 4 months of age faced an increased risk for type 1 diabetes (Virtanen et al. 1991, Norris Kostraba et al. 1992, Virtanen 1992, Verge et al. 1994b). Others were not confident whether the cow's milk protein in commercially available infant formulas was associated with the process of destruction of the β cells (Work Group on Cow's Milk Protein and Diabetes Mellitus 1994). The possibility that infants fed breast milk substitutes may be more susceptible to infections which trigger type 1 diabetes does not seem to have been explored in any study.

Recently a meta-analysis was performed on the early infant diet and type 1 diabetes risk (Norris and Scott 1996). It was suggested that the increased risk of type 1 diabetes associated with any of the infant diets was small. In addition experimental data on the diabetogenic effect of milk containing diets in the BB rat and NOD mouse did not support the postulated effect of milk.

## 2.3- Complications of Type 1 Diabetes

The complications of type 1 diabetes may be classified as either acute or chronic (Zeman 1991, Laycock and Wise 1996).

### *Acute complications*

Acute complications can be reversed quickly with the adjustment of blood glucose level. The most important acute complications of diabetes mellitus are hypoglycemia, diabetic ketoacidosis and hyperosmolar nonacidotic diabetes. In addition other difficulties in control such as the Somogyi effect and dawn phenomenon may be observed (Zeman 1991, Ludwig-Beymer et al. 1996).

In patients with type 1 diabetes, hypoglycemia is known as an insulin reaction or insulin shock (Cryer 1996). The treatment of this condition must be prompt. Repeated severe reactions may cause brain damage (Zeman 1991, Widom and Simonson 1994).

Hypoglycemia can be a major problem in the management of patients with type 1 diabetes (Cryer et al. 1994), although in the Diabetes Control and Complications Trial (DCCT) Research Group (1991) only a minority of intensively treated patients showed episodes of severe hypoglycemia. Nevertheless in the DCCT there was a two- to threefold increase in the incidence of hypoglycemia in the patients receiving intensive insulin treatment (DCCT Research Group 1987).

Diabetic ketoacidosis (DKA) usually results from lack of insulin. This leads to a decreased uptake and utilization of glucose by the tissues, increased glucose synthesis from protein, and the breakdown of fat in the liver and in adipose tissue. At the same time there are some major changes such as increased secretion of glucagon, cortisol, catecholamines and growth hormone (counter-regulatory hormones). There is a rise in blood glucose, non-esterified fatty acids

and ketone bodies. The clinical features consist of dehydration, lethargy, and vomiting, followed by drowsiness progressing if untreated to coma and occasionally death.

***Chronic complications***

These complications are the result of hyperglycemia (Nathan 1996) perhaps in combination with a genetic predisposition (Watkins et al. 1996). The chronic complications of diabetes are largely the consequence of microvascular lesions. These lesions may be triggered by sorbitol accumulation, glycoprotein or mucopolysaccharide formation in blood vessels. The lesions most frequently occur in the walls of blood vessels of the nerves, kidneys, and eyes of patients with diabetes (Zeman 1991).

Follow up of 307 patients with type 1 diabetes for 40 years showed that the occurrence of visual impairment, blindness, renal failure, stroke and amputation were 14%, 16%, 22%, 10%, 12% and 21% respectively (Deckert et al. 1978). In the DCCT it was shown that producing near-normal blood glucose levels by intensive therapy reduces the risk of retinopathy, nephropathy and neuropathy by 50-75% compared with conventional treatment (Genuth 1996).

Angiopathy, which may be classified into macroangiopathy and microangiopathy accounts for about 80% of diabetic deaths (Laycock and Wise 1996).

Macroangiopathy leads to major macrovascular outcomes such as death from cardiovascular disease, myocardial infarction, and peripheral vascular events. One consequence of macroangiopathy is vascular occlusion in the legs of patients with diabetes, leading to intermittent claudication (Zeman and Hanson 1991).

Microangiopathy affects the retina of the eye (retinopathy), the glomeruli of the kidney (nephropathy), and some parts of the nervous system (neuropathy) (Ludwig-Beymer 1994, Nathan DM 1993). In this condition, the basement

membranes around capillaries thicken, contributing to the loss of circulation in the limbs and increasing susceptibility to infection.

Retinopathy occurs in all forms of diabetes (Nathan 1993). As with all diabetes-specific complications, the development of retinopathy depends on the duration of the disease (Krolewski et al. 1986, Pirart 1978). After seven years, approximately 50 per cent of patients with type 1 diabetes have some degree of retinopathy detectable by stereoscopic fundus photography (Palmberg et al. 1981) and the incidence rises rapidly after 15 years from diagnosis (Palmberg et al. 1981, Santiago 1993).

Retinopathy starts with microaneurysms arising from the terminal capillaries of the retina. Dot and blot hemorrhages appear; this is termed nonproliferative retinopathy. This usually does not lead to the loss of vision. With increasingly severe retinopathy, the abnormal vessels can become occluded, leading to retinal ischemia with infarctions in the nerve layer of retina. If vitreous hemorrhages occur vision will be obscured (Nathan 1993).

Deckert et al. (1978) and Andersen et al. (1983) showed that nephropathy developed in 35 to 45 percent of patients with type 1 diabetes. Diabetic nephropathy evolves through several interconnected phases: an early phase of physiologic abnormalities of renal function, a phase of microalbuminuria, and a clinical phase with persistent clinical proteinuria progressing to end-stage renal failure (Trevisan and Viberti 1996).

Microalbuminuria may occur five years after the onset of diabetes (Viberti et al. 1982, Viberti and Keen 1984, Mathiesen et al. 1984, Mogensen and Christensen 1984, Selby et al. 1990). Some studies have reported a significantly higher HbA1c% in microalbuminuric individuals (Mathiesen et al. 1984, Wiseman et al. 1984, Klein et al. 1992). In a study of 1888 patients with type 1 diabetes who were aged less than 40 years and with disease for less than 35 years, the prevalence of microalbuminuria was about 3% and it was related to elevated blood

pressure and longer duration of the disease (The Microalbuminuria Collaborative Study Group 1992).

Patients developing clinical nephropathy have an increased incidence of coronary heart disease compared with patients not developing nephropathy (Dorman et al. 1984, Jensen et al. 1987, Trevisan and Viberti 1996). These patients have higher blood pressure and serum cholesterol (Jensen et al. 1987, Barnes 1993).

Initially in type 1 diabetes there is renal hypertrophy, with expansion of the glomeruli, including the mesangium and glomerular basement membrane. Glomerular composition changes slowly, leading to characteristic mesangial expansion, thickening of the glomerular basement membrane, and afferent and efferent arteriosclerosis. With more advanced nephropathy, glomerular closure occurs. There is compensatory hypertrophy of the functioning glomeruli during this stage. End-stage renal disease is characterized by small, atrophic kidneys with diffuse glomeroulosclerosis (Mauer et al. 1984, Osterby 1987, Bilous et al. 1989, Feldt-Rasmussen et al. 1991).

It has been found that a peripheral, symmetric sensorimotor neuropathy is the most common form of diabetic neuropathy, whose other forms include cranial and peripheral motor neuropathies and autonomic neuropathy. Autonomic neuropathy can affect gastric or intestinal motility, erectile function, bladder function, cardiac function, and vascular tone. Gastroparesis may not only cause symptoms but also alter the absorption of meals and affect glycemic control (Nathan 1993). The DCCT Research Group (1988a) in their electrophysiologic studies demonstrated subclinical abnormalities, including slowed motor- and sensory-nerve conduction in most patients after 5 to 10 years of diabetes.

## 2.4- Glycemic Control and the Onset of Complications

The effect of good diabetic control in prevention or delaying the onset of complications has been discussed since the first clinical use of insulin in 1922 (Page and Tattersall 1994). For instance Johnsson (1960) and Pirart (1978) showed that poor glycemic control is associated with an increased risk of microvascular complications.

This relationship was confirmed in several other studies (Doft et al. 1984, Wiseman et al. 1984, Weber et al. 1986, Bangstad et al. 1989, Mortensen 1990, Klein et al. 1993). Later a relationship between HbA1c level and development of retinopathy and microalbuminuria in young patients with type 1 diabetes was reported (Joner et al. 1992).

The Diabetes Control and Complications Trial (DCCT) was designed to compare the effects of intensive to conventional treatment on the development and progression of early vascular and neurologic complications in 1441 patients with type 1 diabetes (age 13-39 years patients with 1-5 years of diabetes) for a mean follow-up of 7 years (DCCT Research Group 1986, 1987, 1993). In this trial intensive therapy could delay the onset and slow the rate of progression of retinopathy, nephropathy, and neuropathy by a range of 35 to more than 70 percent. Also this kind of treatment reduced the risk of subsequent albuminuria and microalbuminuria by 54% and 39% respectively. On the other hand it was found that the risk of hypoglycemia in intensively treated patients was two to six times higher than that observed with conventional treatment (DCCT Research Group 1993a). Thus the risk of hypoglycemia must be taken into account when assessing the benefits of intensive treatment in this trial (DCCT Research Group 1991).

The DCCT Research Group concluded that the onset and progression of diabetic retinopathy and nephropathy in adolescent subjects would be delayed and

slowed by intensive therapy, although the risk of hypoglycemia is increased (DCCT Research Group 1994).

As part of the Pittsburgh Epidemiology of Diabetes Complication Study, it was shown that good glycemic control in patients who had been diagnosed with type 1 diabetes for more than 25 years was associated with avoidance of major complications (Orchard et al. 1990). Others followed up the effects of glycemic control on the subjects of Pittsburgh Epidemiology of Diabetes Complications (EDC) who were diagnosed from 1986 to 1988 (Lloyd et al. 1996a). It was found that during the first 4 years of follow-up, subjects who were in "poor" control at baseline were significantly more likely to develop microalbuminuria, proliferative retinopathy, and distal symmetrical polyneuropathy, compared with subjects who were in "fair" control. This study showed weaker associations for the later stages of renal disease, and little relation was noticed between glycemic control and coronary disease. Those with higher education and income had fewer complications.

An outcome study of type 1 diabetes patients in Soweto, South Africa showed that after a 10 year follow up retinopathy affected 52%, peripheral neuropathy 42% and nephropathy 28% of the patients (all significantly increased from the first assessment). A significant proportion of deaths was due to renal failure (50%). Although diabetic complications frequently occur in these patients, most of them reported a satisfactory quality of life (Gill et al. 1995).

Recently a prospective epidemiological study was carried out to describe the relation between glycated hemoglobin and the incidence or progression, or both, of diabetic microvascular complications in patients with type 1 diabetes (Klein et al. 1996). The results showed that there was a strong relation between glycemic control and the incidence or progression of diabetic retinopathy, the incidence of gross proteinuria, and the incidence of loss of tactile sensation or temperature sensitivity in type 1 diabetes.

A study was conducted to compare the psychological characteristics of adults (age 21-40 year) with type 1 diabetes who had poor glycemic control with similar patients in good glycemic control (Jacobson et al. 1990). They found that patients with chronic poor control felt physically best at a higher blood glucose level than patients with good diabetic control. There were no differences in their level of diabetes knowledge, self-esteem, or psychiatric symptomatology.

In the Oslo Study, the effect of long-term strict glycemic control on peripheral and autonomic nerve function in adult patients with type 1 diabetes was investigated (age 18-42 year). It was observed that lowering blood glucose during 8 years retarded the deterioration in nerve conduction velocity in the diabetic nerve (Amthor et al. 1994).

### *Cardiovascular disease (CVD) and its risk factors in type 1 diabetes*

The increased risk of mortality in patients with type 1 diabetes is because of cardiovascular disease (Kannel and McGee 1979). A number of risk factors for cardiovascular disease in type 1 diabetes have been described.

#### *Overweight and obesity*

Weight gain was a problem for some individuals with type 1 diabetes under strict glycemic control (Wing et al. 1990, DCCT 1988b). This weight gain may result in increased risk of lipid disturbances and higher blood pressure, which would counteract the benefits of improved glycemic control (Lasker 1993, DCCT 1993a, Kannel and McGee 1979).

In the DCCT although there was an increasing risk for weight gain, intensive therapy still had a beneficial effect on macrovascular disease (DCCT 1995a). In the EURODIAB Complications Study overt cardiovascular disease was found in 9% of men and 10% of women, increasing with age. Cardiovascular

disease was associated with higher waist to hip ratio, BMI, triglyceride and hypertension, and with the duration of diabetes (Koivisto et al. 1996).

The findings on mortality risks associated with body weight in people with type 1 diabetes are conflicting. Goodkin (1975) reported that obesity may be protective, while others reported that obesity among women with diabetes increased the risk of death (Chazan 1970). In Denmark no association could be found between obesity and survival (Borch-Johnsen et al. 1987).

More recently WHO carried out a cohort study and examined morbidity and mortality risks associated with body weight in individuals with type 1 diabetes (Chaturvedi et al. 1995). A large number of subjects were recruited from nine centres worldwide. Body weight was positively associated with blood pressure and cholesterol in men. Fasting blood glucose increased significantly with body weight in women. Mortality was lowest in people with body mass index of 20 to <24 $kg/m^2$.

*Biochemicalfactors*

Earlier studies had shown that the prevalence of dyslipidemia was higher in individuals with type 1 diabetes than non-diabetic controls (Mann JI et al. 1978, Court et al. 1978, Sosenko et al. 1980, Lopes-Virella et al. 1981, Al Muhtaseb et al. 1992). Other researches found no difference between blood cholesterol in diabetic and non-diabetic subjects (Chase and Glasgow 1976, Ewald et al. 1984, Strobl et al. 1985, Kobbah et al. 1988, Salzer et al. 1993). Plasma cholesterol, triglycerides, and lipoproteins were in the normal range when diabetic control was good in type 1 diabetes (Sosenko et al. 1980, Briones et al. 1984, Gonen et al. 1985, Nikkila et al. 1985).

Hypertriglyceridaemia is closely associated with coronary heart disease (Jarrett 1978, Beach et al. 1979). HDL cholesterol was found to be high rather than low in this group of patients on insulin (Kennedy et al. 1978, Nikkila and

Hormila 1978, Mattock et al.1979, Durrington 1980). LDL cholesterol is normal or low (Schernthaner et al. 1983, Gonen et al. 1985, Laakso et al. 1985). There were no significant differences in serum triglyceride, serum cholesterol, LDL cholesterol, and HDL cholesterol between men with type 1 diabetes and non-diabetic men (Winocour et al. 1986).

As part of the Diabetes Control and Complications Trial, lipid and lipoprotein levels in patients with type 1 diabetes (age 13 to 40 years) for 1-15 years were compared with the values in healthy non-diabetic controls (DCCT Research Group 1992). The fasting blood samples were analysed for cholesterol, triglyceride, low-density lipoprotein cholesterol and high-density lipoprotein cholesterol. Results in males and older females with type 1 diabetes were similar to the values found in controls. Total cholesterol, LDL cholesterol and triglycerides were all directly related with HbA1c and fasting blood glucose. HDL cholesterol decreased when insulin dose, body weight, serum creatinine, urinary albumin, and systolic and diastolic blood pressure were increased. There was a weak correlation between blood lipids and dietary variables.

Others found that the prevalence of dyslipidaemia among 51 children and adolescents with type 1 diabetes was higher than in a control population of schoolchildren (39% versus 17%) (Azad et al. 1994). Fasting serum cholesterol alone was raised in 25% of children and adolescents with type 1 diabetes, and in a further 14% cholesterol and triglyceride were raised. These values for non-diabetic subjects were lower, namely 16% and 4% respectively. Serum total cholesterol, low density lipoprotein cholesterol, non-esterified fatty acids and triglyceride were higher in diabetic subjects. With worsening diabetic control, serum total cholesterol, triglyceride, and apolipoprotein (apo)B concentration increased, while serum high density lipoprotein cholesterol and apoA-I levels were unchanged. A positive correlation was found between glycated haemoglobin and total cholesterol, triglycerides, and apoB in diabetic children. Therefore it was concluded that raised blood lipids in young patients with type 1 diabetes was

related to poorer glycemic control. Also it was shown that lipid levels in patients with type 1 diabetes with good diabetic controls were similar with those in non-diabetic subjects.

The relationship of serum lipids, lipoproteins, apolipoproteins and antioxidants with renal dysfunction was examined in 121 type 1 diabetic subjects (O'Brien et al. 1996). The glomerular function was assessed by urinary albumin/creatinine ratio on three early morning spot urine samples. There was a significant correlation between urinary albumin/creatinine and serum total cholesterol, triglycerides, apolipoproteins A-I, A-II and B, and glycated haemoglobin. No significant association was found between glomerular function and HDL cholesterol. In another study it was concluded that in patients with type 1 diabetes, all lipoprotein abnormalities improve with good glycemic control except VLDL which is increased in these patients (Chait and Brunzell 1996).

In New Zealand the current degree of metabolic control in diabetic patients attending a diabetes clinic from 1992 to 1995, has been reported (Dunn 1996). Blood cholesterol, HDL cholesterol and triglyceride were all within normal range and mean total cholesterol was close to the target values set by the National Heart Foundation (Mann et al. 1993).

The risk factors of coronary heart disease were investigated in 90 adults with type 1 diabetes (Winocour et al. 1992). In order to compare the results, 172 non-diabetic normal subjects were recruited. It was found that about 25% of subjects of each group had coronary heart disease. Type 1 diabetic patients with coronary heart disease had significantly higher levels of systolic blood pressure, albumin excretion, serum creatinine, triglycerides, VLDL cholesterol and C-peptide and decreased level of HDL and HDL2 cholesterol compared to patients without coronary heart disease. It was found that coronary heart disease in type 1 diabetes was closely related to age and levels of blood pressure and total serum lipids. In this group of patients apolipoproteins and albuminuria were not considered important predictors of coronary heart disease.

Fibrinogen has been found to be elevated in patients type 1 diabetes and hyperfibrinogenemia is an independent predictor of vascular complications (Ganda and Arkin 1992). The EURODIAB Complications Study Group showed that fibrinogen had a positive relationship to vascular risk factors and a possible role in arterial disease in diabetes (Greaves et al. 1997).

***Type 1 diabetes and the kidney***

Several studies have all concluded that the development of renal complications is associated with poor metabolic control (Wiseman et al. 1984, Kroc Collaborative Study Group 1984, Feldt-Rasmussen et al. 1986, Mathiesen et al. 1990). Those patients who progressed to microalbuminuria during a four year follow up had worse blood glucose control and higher blood pressure (Microalbuminuria Collaborative Study Group 1993).

In another report the prevalence of renal disease in two type 1 diabetes populations in the US (EDC) and in Europe (EURODIAB) were compared (Lloyd 1996b). The results showed that the prevalence of macroalbuminuria was higher in EDC than in EURODIAB. Adjusting for glycemic control, hypertension or smoking did not change the risk of macroalbuminuria.

Patients who took part in the Pittsburgh Epidemiology of Diabetes Complications Study were re-examined after two years (Coonrod et al. 1993). It was found that in these patients who had normal albumin excretion at baseline, glycemic control, age or duration of type 1 diabetes, disturbed lipids, and possibly elevated blood pressure might all contribute to the development of microalbuminuria.

The presence of persistent microalbuminuria can be regarded as a sensitive predictor of development of clinical diabetic nephropathy (Parving et al. 1982, Viberti et al. 1982, Viberti and Keen 1984, Mathiesen et al. 1984, Mogensen and Christensen 1984). In a 10-year perspective study it was shown that after

another 5 to 10 years of diabetes, overt proteinuria develops in those patients destined to develop end-stage renal disease (Mathiesen et al. 1995).

In patients with type 1 diabetes there is an association between nephropathy and cardiovascular disease (CVD) (Jensen et al. 1987, Borch-Johnsen and Kreiner 1987, Winocour et al. 1987, Deckert et al. 1989, Dullaart et al. 1989, Jay et al. 1991, Deckert et al. 1992). Although from epidemiological studies it can be concluded that elevation of blood pressure, cholesterol and fibrinogen in albuminuric patients with type 1 diabetes are potential cardiovascular risk factors, the exact mechanism for this effect is not clear (Jensen et al. 1988, Deckert et al. 1992).

Microalbuminuria is found to have a strong predictive power in renal disease in diabetic patients (Mogensen 1997). This has been shown in several other studies (Mogensen et al. 1984, Viberti et al. 1982, Mogensen et al. 1986). Measurement of albumin can be performed either on a timed urine sample for a more accurate result or on a spot urine sample for initial screening (Jerums et al. 1994). Microalbuminuria is diagnosed when the urinary albumin excretion rate (UAER) is >20 μg/min but <200 μg/min (Deckert et al. 1992). Because there is little information on various cut-off points of albumin concentrations, using the albumin: creatinine ratio has been suggested on random (Gatling et al. 1988, Watts et al. 1986) and early morning (Gatling et al. 1988, Cohen et al. 1987, Hutchison et al. 1988, Gatling et al. 1985, Marshall and Alberti 1986) urine samples. The results of these studies show that the measurement of the albumin: creatinine ratio in an early morning urine sample appears to be the most reliable method of screening for microalbuminuria, with sensitivity of 88 to 100% and specificity of 81 to 100% depending on the cut-off ratio chosen (Marshall 1991).

## 2.5- Dietary Management in Type 1 Diabetes

Dietary management has long been regarded as the cornerstone in the treatment of all types of diabetes mellitus, although various kinds of diets have been prescribed by different people (Birkbeck et al. 1976). The best diet for patients with type 1 diabetes has still not been validated and more studies about high fat versus high-carbohydrate diet for long-term have been recommended (Grundy 1991).

Table 1 shows the distribution of macronutrients (energy%) recommended to diabetic patients in different periods (American Diabetes Association 1998). It is claimed that today there is no one "diabetic" or "ADA" diet. The recommended diet should only be prescribed on the basis of dietary assessments, treatment goals and desired outcomes.

**Table 1-Historical perspective of nutrition recommendations**

| | Distribution of calories(%) | | |
|---|---|---|---|
| Year | Carbohydrate | Protein | Fat |
| Before 1921 | | Starvation diets | |
| 1921 | 20 | 10 | 70 |
| 1950 | 40 | 20 | 40 |
| 1971 | 45 | 20 | 35 |
| 1986 | Up to 60 | 12-20 | <30 |
| 1994 | * | 10-20 | *, ≈ |

* Based on nutritional assessment and treatment goals. ≈ Less than 10% of calories from saturated fats.

***Dietary intake***

Widdowson (1947) showed that the diets of 13 diabetic children (5 to 17 years) were lower in energy and carbohydrate but higher in fat and protein than the diets of non-diabetic people.

Others have assessed the current dietary practice and the insulin regimen of diabetic children (Hackett et al. 1986). The children and their parents were asked

to record all food intake during three consecutive days, with the time of consumption and all the foods in household measures. HbA1 and a random C peptide were measured on their blood samples. It was found that intakes of energy, fibre and compliance with prescriptions for carbohydrate were the only factors related to HbA1. Also it was shown that their energy intakes were similar with expected values for non-diabetic subjects. Similar results was found in another study in patients with type 1 diabetes (Kinmonth and Baum 1984).

It was reported that the daily dietary variation in the energy and carbohydrate intake of above mentioned population (Hackett et al. 1986) was less than that of non-diabetic children. Dietary compliance changed during the day. A qualitative approach to prescription diets for individuals with diabetes was suggested (Hackett et al. 1988).

Dietary intakes of diabetic children (age 5-10 year) were compared with those of non-diabetic children of similar age living in Sydney (Ballinger et al. 1994). Dietary data were collected by the three day food record. They found that the intake of most nutrients in diabetic children was higher than non-diabetic children and their intakes met the Australian recommended dietary intakes. The need for the development of guidelines specific for diabetic children was emphasized.

The nutritional intake of 2868 patients with type 1 diabetes (15 to 60 years) from 30 centres in Europe was assessed by a validated 3-day food record (Toeller et al. 1996). It was found that the recommendations from the Diabetes and Nutrition Study Group of the EASD were achieved for total fat, saturated fatty acids and carbohydrate by only 14%, 14% and 15% of patients respectively.

In a study in England the nutritional intake of people with type 1 diabetes was assessed. It was indicated that the majority of patients met the recommendations when they were only advised on a low fat, high carbohydrate diet without any education about carbohydrate exchanges (Pearson et al. 1996).

In the DCCT dietary data were collected to show the influence of dietary factors on the outcomes of the trial. The DCCT (1995) reports that the diets of patients at the baseline was different from the ADA guidelines, with 38% of calories derived from fat and 45% from carbohydrates. Two groups of intensive and conventionally treated patients in this trial had similar calorie intake, nutrients, alcohol, fibre and caffeine. The patients (age 13-39 years) in both treatment groups were instructed about meal plans and timing of food consumption before the trial and also energy and nutrient intakes (DCCT Research Group 1988c).

*Dietary assessment methods*

In Sydney, a one week adolescent diabetes food frequency questionnaire (ADFFQ) and a four day food record were applied in a group of patients with type 1 diabetes (age 12-18 year) (Garnett et al. 1995). For both dietary methods estimated energy intake/estimated basal metabolic rate (EIBMR) was calculated and the results were compared with the levels, which were suggested by Goldberg et al. (1991). It was found that results of ADFFQ were similar to the four day food record regarding the average intakes of nutrient and ranking individuals according to the intakes of protein, fat, carbohydrate, fibre and vitamin C. The mean EIBMR for individuals' ADFFQs and food records represented habitual intake, but there was underreporting in girls. The authors suggested that the ADFFQ is a useful tool in dietary intake studies (although the ADFFQ was completed after the dietary record which gave experience of recording the food intake).

In the DCCT the usual dietary intakes, particularly of energy, macronutrients, dietary fibre, cholesterol, and fatty acids were assessed by the use of standardized food models and a validated set of two-dimensional shapes. In this study dietary data were collected by the meal-based diet history method originated by Burke (1947). Each patient completed a Food Preparation

Questionnaire and a Food Pattern Questionnaire prior to the diet interview. Diet history was performed by trained interviewers in order to record the data in a standardized form. The diet histories completed at the end of years 1 and 2 of the study showed the reproducibility of this method in the DCCT (Schmidt et al. 1994).

In Tasmania, the application of a self-administered semiquantitative food frequency questionnaire (FFQ) for measurement of energy and macronutrient intakes of adults with type 1 diabetes was studied (Riley and Blizzard 1995). The questionnaire was adapted from a FFQ designed for use in Australia (Baghurst and Record 1984). The results were compared with 2 days of weighed dietary records. It was found that the estimated energy and nutrient intakes were similar except dietary protein that was 10% higher by FFQ. It was concluded that FFQ method in this study performed similarly to the method used in other populations. In this group of patients with type 1 diabetes, readministration of the same method gave a record of decreased energy and macronutrient intake, a tendency, which needs to be confirmed in other studies.

***Dietary adherence***

Most patients with type 1 diabetes are reported to have poor adherence to diet plans. The knowledge and compliance to a controlled carbohydrate diet in a group of patients with type 1 diabetes were studied (age 14-85 years) (McCulloch et al. 1983). It was found that 44% had forgotten their dietary prescription and 35% were not able to estimate carbohydrate. The scores of a quiz regarding the carbohydrate content of foods found to be related significantly to HbA1 levels. It was concluded that better understanding and adherence to a controlled carbohydrate diet leads to a better glycemic control.

In a study dietary adherence was assessed by counting the number of deviations from the planned number of feedings, exchanges, and caloric content. It

was found that two thirds of subjects adhered to the number of planned feeding, while only 10% of them adhered to prescribed exchanges 90% of the time. It was also shown that younger age group (0-11 years) tended to report the best adherence. Those patients with good dietary adherence showed better glycemic control (Christensen et al. 1983).

In Sydney an educational diet program was conducted for patients with type 1 diabetes (age 18 to 75 years). Patients who complied with the recommendations had significantly better glycemic control (Webb et al. 1984).

In a Danish study, daily diets of people with type 1 diabetes were assessed by 7 day food recall interviews and a questionnaire (Palmvig et al. 1987). They found that the average protein intake was 19-20% of total energy. It was also emphasized the evaluation of dietary treatment by assessing the compliance of the patient. In this study it was believed that the measurement of biochemical parameters was not sufficient to assess the patient's glycemic control, but along with these measures, periodical evaluation of the patient's diet by food records or interviews was indicated.

It was reported that women with type 1 diabetes who were participants in the Nurses Health Study consumed less energy from carbohydrates, especially from sucrose, and more energy from protein and fat than did control women. The results that were collected by a semiquantitative food frequency questionnaire showed that these patients did not consume the diet, which is recommended by the American Diabetes Association (Shimakawa et al. 1993).

In Ireland the dietary intake assessment also showed that the patients with type 1 diabetes did not meet the present dietary targets in their diets (Humphreys et al. 1993).

In Germany, it was found that in a group of adults with type 1 diabetes on intensive insulin therapy there was more diet liberalization and therefore the treatment was less burdensome (Muhlhauser et al. 1995). In these patients the duration of diabetes was 15±7 years and they had been on intensified insulin

therapy for six years. Before follow up instructions were provided about diet, insulin injection and measuring blood glucose. The patients were educated on the carbohydrate content of their foods and the estimation of insulin needs for different carbohydrate intakes. Patients did not receive any information on energy, fat, protein and fibre intake. In this study, in which there was no control group, there was no relationship between liberalized diet and HbA1c%, severe hypoglycemia, body mass index or serum cholesterol.

It has been found that in patients with type 1 diabetes reducing protein intake in daily diet can protect some degrees of renal function (Pedersen et al. 1990). It was indicated that short term protein restriction can reduce the early glomerular hyperfiltration (Wiseman et al. 1987, Kupin et al. 1987, Rudberg et al. 1988, Pedersen et al. 1989). Also the results of other studies showed that the protein reduction in the diet could prevent microalbuminuria/proteinuria (Cohen et al. 1987, Ciavarella et al. 1987, Evanoff et al. 1987).

Later about 0.8 g protein per kg body weight (10% of energy from protein) was recommended in the daily diet of patients with type 1 diabetes (Palmvig 1989). Recently the results of five different studies on the restriction of protein intake in type 1 diabetes were analysed (Pedrini et al. 1996). It was concluded that restriction of dietary protein significantly delays the progression of diabetic renal disease (Ciavarella et al. 1987, Barsotti et al. 1988, Walker et al. 1989, Zeller et al. 1991, Dullaart et al. 1993). These studies prescribed a low protein diet which contained from 0.5 g/kg of body weight per day to 0.8 g/kg of body weight daily. After follow-up from 9 to 33 months, they all showed a beneficial effect on the change in albumin level or total protein excretion rate and the reduction in glomerular filtration rate or creatinine clearance.

The effect of salt sensitivity on blood pressure in 30 normotensive subjects with type 1 diabetes (11 with and 19 without microalbuminuria) was studied (Strojek et al. 1995). In this study the diabetic group were compared with non-diabetic controls after an experimental diet which contained 130 kJ/kg/d,

including 20(±1) mmol sodium, 250g complex carbohydrates, 45g fat and 35 mmol potassium daily. It was found that 43% of diabetic subjects (50% patients with microalbuminuria and 37% without microalbuminuria) and 17% non-diabetic subjects were salt sensitive.

In a similar study the effects of moderate sodium restriction on blood pressure were assessed in 16 patients with type 1 diabetes with nephropathy and mildly hypertensive (Muhlhauser et al. 1996). It was found that the reduction of 100mmol/day of sodium intake after 4 weeks led to a slight decrease in diastolic blood pressure.

In a recent study it was concluded that moderate alcohol consumption by healthy, fed subjects does not increase the risk of hypoglycemia (Meeking and Cavan 1997). Others showed that high carbohydrate, high fibre diets which provided 70% of energy as carbohydrate and 70 g dietary fibre of daily diet of 10 subjects with type 1 diabetes (35 to 65 years) decreased their basal insulin requirements and increased peripheral glucose disposal with no change in glycemic control (Anderson et al. 1991).

***Diet and socioeconomic status***

It has been reported that people in higher socioeconomic groups eat more wholemeal and brown bread, more fresh fruit and vegetables and less fatty milk, eggs and meat (Steele et al. 1991, Smith and Baghurst 1992, Shimakawa et al. 1994). Also it was shown that higher socioeconomic status was associated with a lower fat, saturated fat and refined sugar intake, and higher fibre intake (Bolton-Smith et al. 1991, Hulshof et al. 1991, Smith and Baghurst 1992, Shimakawa et al. 1994).

On the other hand in Finland it was shown that higher socioeconomic groups did not follow current national dietary guidelines better than lower socioeconomic groups. Those with higher socioeconomic status consumed more

of the modern recommended foods, such as vegetables and fruits but less traditional recommended foods, such as bread and potatoes (Roos et al. 1996).

***Low glycemic index diets***

The concept of glycemic index allows classification of foods on the basis of the blood glucose responses they produce (Jenkins et al. 1981). The effects of a low glycemic index (GI) diet was studied in a group of patients with type 1 diabetes (Calle-Pascual et al. 1988). During two periods of 4 weeks on diet no significantly differences in insulin dose and glycemic control was observed. In another study it was shown that a moderate switch from high to low glycemic index foods for 3 weeks led to an improvement in metabolic control of subjects with type 1 diabetes (Fontvieille et al. 1988). Low glycemic index starchy foods could reduce serum cholesterol and improved glucose control in diabetic children in a 6 week study period (Collier et al. 1988).

In another study the benefit of a low glycemic index diet was examined (Fontvieille et al. 1992). Two kinds of diets were used by the patients with type 1 diabetes. The high glycemic index diet contained more bread and potato and the low glycemic index diet was enriched with pasta, rice and legumes. The low glycemic diet resulted in an improvement in levels of fructosamine, fasting blood glucose and serum triglycerides, but no significant reduction in body weight, HbA1c%, insulin requirement and other lipids. It was concluded that inclusion of low glycemic index foods in the diet of people with type 1 diabetes might have a favourable effect on carbohydrate and lipid metabolism although this could be achieved with small changes in the dietary habits of individuals.

Later it has been claimed that the low GI diets are "user friendly" and in the majority of studies they have had beneficial effects on glycemic and lipid control of patients with diabetes (Brand Miller 1994). In a report by Wolever and Brand Miller (1995) it was concluded that in patients with type 1 diabetes, the

isoenergetic substitution of starch by sucrose at a moderate amount does not have a significant effect on the blood glucose level (Vaaler et al. 1980, Steel et al. 1983, Hassinger et al. 1983, Forlani et al. 1989, Loghmani et al. 1991). These investigators also reported that the rise of blood glucose after a meal depends on many factors including the source of the sugar, method of preparation and its composition.

## 2.6- Exercise in Type 1 Diabetes

It has been suggested that exercise does not necessarily improve glycemic control in patients with type 1 diabetes. However, it is recommended for these group of patients because of its effect on cardiovascular fitness and psychological well-being and for better social life and recreation (American Diabetes Association Position Statement 1990).

In early studies it was shown that exercise in type 1 diabetes patients lowered blood glucose acutely (Berger et al. 1977, Kemmer 1979). Later it was reported that patients participating in a program with regular exercise had improved blood glucose level (Stratton et al. 1987, Marrero 1988). It was also found that strength-training program for 10 weeks for men with type 1 diabetes was associated with increased strength, reduced blood glucose and HbA1c% with no change in insulin dose and lower cholesterol level (Durak et al. 1990). Others have failed to demonstrate long term beneficial effects of exercise on glycemic control (Zinman et al. 1977, Wallberg-Henriksson et al. 1986).

It has also been shown that supervised regular exercise in type 1 diabetes patients for 12 weeks did not improve their blood glucose and HbA1c% (Zinman et al. 1984). This lack of effect on glycemic control was attributed to the increased intake of energy. As part of the Pittsburg study, it was shown that long term physical exercise in patients with type 1 diabetes was not associated with an adverse effect on health (LaPorte et al. 1986). In another study it was shown that

in subjects with type 1 diabetes, insulin-mediated glucose disposal was positively related to the state of physical fitness and negatively to HbA1c level (Arslanian et al. 1990).

Recently the impact of physical activity on cardiovascular risk factors in well-controlled patients with type 1 diabetes was studied (Lehmann et al. 1997). It was shown that increased physical activity resulted loss of abdominal fat and a decrease in blood pressure and lipid related cardiovascular risk factors.

On the other hand it was reported that exercise could present risk of hypoglycemia for patients with type 1 diabetes. Further when vigorous physical activity is superimposed on the insulin-deficient state, may cause hyperglycemia and later may lead to ketosis (Horton 1996). It has also been shown that vigorous exercise increases proteinuria (Mogensen and Vittinghus 1975, Viberti et al. 1978).

## 2.7- Quality of Life and Well-Being in Type 1 Diabetes

It is now well recognized that the impact of chronic diseases and their treatments on health outcome must be assessed on the quality of life of the patients rather than limiting the assessment on traditional methods such as mortality and morbidity data (Croog et al. 1986, Sansoni 1995). Type 1 diabetes is a chronic disease which affects the quality of life because the treatments are burdensome practically, emotionally and socially, and the complications cause weakness and in some cases they are life threatening (Fisher et al. 1982, Rodin 1983, Jacobson 1994).

It has been reported that patients with type 1 diabetes who had better metabolic control, had less emotional problems than those that were poorly regulated (Simonds 1977, Anderson et al. 1981, Mazze et al. 1984). Others have found that the patients with diabetes have a decrease in their quality of life (Stewart et al. 1989, Mayou et al. 1990, Nerenz et al. 1992 and Lloyd et al. 1992).

The DCCT Research Group (1988c) assessed the quality of life of the two treatment group and compared their results after 10 years of trial. The Diabetes Quality-of-Life (DQOL) measure was designed in a multiple choice form questionnaire. Items of the questionnaire did not identify the kind of the treatments. The questions related to satisfaction, impact and worry, and were responded to on a 5-point Likert scale.

The items of DQOL were derived from other studies (Sullivan 1979, Jacobson and Hauser 1983), experienced health professionals and the patients with type 1 diabetes. The validity of this measure was determined by three assessment instruments including, the Symptom Checklist-90-R (SCL) (Derogatis, 1977, Derogatis et al. 1976), the Bradburn Affect Balance Scale (ABS) (Bradburn 1969, McDowell and Praught 1982), and the Psychosocial Adjustment of Illness Scale (PAIS) (Derogatis 1983, Derogatis 1983). After administering different measures to patients, significant correlations were found between their total score of DQOL, the satisfaction and impact scales, and the SCL, ABS, and PAIS scales. The reliability and validity of DQOL as a screening measure was supported by this study.

In another report it was concluded that both intensively and conventionally treated patients showed a similar levels of well-being and psychological health at the beginningand the end of the trial. Hypoglycemia did not lessen the diabetes-related quality of life (the DCCT Research Group 1996).

The effects of type 1 diabetes on patient perception of their quality of life have been examined in another study (Jacobson et al. 1994). For this purpose two measures of quality of life, consisting of DQOL and the Medical Outcome Study Health Survey 36-Item Short Form (SF-36) (Stewart et al. 1989) were used for this group who were older than 18.

The Medical Outcome Health Survey SF-36 measures six aspects of functional health status including 1) physical functioning, 2) effect of physical illness on role functioning, 3) perception of general health, 4) effect of illness on

social functioning, 5) discomfort because of pain, and 6) mental health (Ware and Sherbourne 1992 and International Resource Centre (IRC) for Health Care Assessment 1991).

The reliability of these two measures were computed and found to be similar to the values which were reported previously (The DCCT Research Group 1988, Stewart et al. 1989, Wu et al. 1991, Ware and Sherbourne 1992, Watchel et al. 1992). SF-36 scales showed a good internal consistency with type I diabetes. This measure was less sensitive to lifestyle issues like the effect of diet or insulin treatment than the DQOL. On the opposite it is more sensitive to changes in the number or severity of complications. Therefore the application of these two measures in assessing quality of life of diabetic patients were suggested.

The results show that the quality of life was not influenced by demographic factors such as sex or education level. Older individuals had worse physical functioning. The patients who were single or married had better quality of life than those who were separated or divorced. With two measures quality of life was shown to be lower in patients with more severe complications.

The quality of life of type 1 diabetic patients with pancreatic/renal transplantation was evaluated by Diabetes Quality of Life Measure (DQOL) and a general quality of life instrument (Nathan et al. 1991). It was found that there were no significant differences between the assessment at baseline and the follow-up in the recipients.

As part of the Pittsburgh Epidemiology of Diabetes Complications Study, the association of diabetic complications with psychological factors in patients with type 1 diabetes was studied (Lloyd et al 1992). The study population consisted of 175 childhood-onset patients with type 1 diabetes who had been diagnosed for more than 25 years. The quality of life of these patients was measured by the quality-of-life questionnaire from the DCCT (the DCCT Research Group 1988c), the Bortner questionnaire (Bortner 1969) and the Beck Depression Inventory (BDI) (Beck and Garbin 1988).

The results showed that the quality of life of patients was significantly related to the presence of certain kind of complications. Those who had macrovascular disease or nephropathy had significantly poorer quality of life compared with those patients with no complications. Patients with peripheral vascular disease (PVD) or cardiovascular disease (CVD) reported poorer quality of life compared with patients free of complications.

Depression as measured by BDI was closely related to the presence and the number of diabetic complications. Women had higher scores than men. Type A behaviour was not correlated with the two other measures. its score did not differ among patients with or without complications.

It was found that DQOL and BDI were correlated with the presence of complications. In this study DQOL score was especially related to the presence of nephropathy, while BDI was closely associated with retinopathy and macrovascular disease.

Another study was conducted to explore the quality of life of 69 young adult patients with type 1 diabetes (Eiser et al. 1991). A knowledge questionnaire (adapted from Meadows et al. 1988) and Diabetes Quality of Life (DQOL) measure (The DCCT Research Group 1988c) were administered. Analysis of data showed that there was a high correlation among three subscales including impact/worry, social relationships and diabetes concerns. No correlation was found between age or knowledge with the quality of life subscales. Social satisfaction correlated with lower insulin dose. The number of injections did not have a significant effect on any of the quality of life subscales. There was a correlation between diabetes satisfaction, and disease variables such as the lower levels of fructosamine, more clinic attendance and longer duration of diabetes. Females reported that diabetes had more of a negative impact on their lives than males, although they obtained higher knowledge scores.

In Sweden the quality of life and metabolic control of 73 subjects with type 1 diabetes who had used multiple pen-injection instead of syringe injections

were examined. Quality of life and metabolic control (HbA1c%) were assessed after 9-13 month follow-up (Wikby et al. 1991). The quality of life of this group of patients was assessed by a quality of life package, which was based on previous studies (Hornquist 1982, Hornquist 1989, Hornquist 1995). It was found that overall quality of life and also metabolic control were not related to either age or duration of diabetes. Quality of life and metabolic control were positively related to cohabitation of the patients. Females showed more satisfaction about their life style than males. They also had better eating, smoking and drinking habits.

A study was conducted to assess the influence of the disease on the daily lives and feelings of well-being of 192 adults with type 1 diabetes (Lundman et al. 1990). The patients were 20 years or above, diagnosed for more than two years and had no severe long-term complications. Twenty three structured questions about experiences of having diabetes were asked. Well-being was measured by eleven semantic differentials, which was designed on the basis of the previous work (Bradley et al. 1984). The analysis of the results showed that no major differences was found between the genders. Many of them were worried about eye problems and monitored blood and urine glucose 1 to 3 times a week. Younger patients had significantly more feelings of anxiety, lack of freedom , insecurity and low self-esteem. Those with shorter duration of disease had greater degree of feelings of insecurity and increased worries about complications.

In Sweden, quality of life of patients with type 1 diabetes who switched from syringe to multiple pen injection treatment was assessed (Hornquist et al. 1995). The results showed satisfactory effects on quality of life in patients while using pen injection treatment.

In France, a questionnaire for subjective quality of life profile was used (Dazord et al. 1994). Seven hundred and forty three patients with type 1 diabetes who were receiving two different forms of insulin injection filled up the

questionnaire. The results show that patients satisfaction was highly related to their ability to move around.

*Conclusion*

Although type 1 diabetes (insulin-dependent diabetes mellitus) constitutes only 10-20% of all diabetic cases its management becomes critical as it affects people early in life and it causes multiple complications. Several studies have shown that good glycemic control will reduce the risk of retinopathy, neuropathy and nephropathy. Also there is an increased risk of cardiovascular disease among these patients.

Management of type 1 diabetes includes insulin replacement, diet and physical exercise. Better understanding and adherence to a prescribed diet is associated with better glycemic control. However it has been shown in different studies that diet is the most problematic part of diabetes management for the dietitian and the patient. The reason may be that the recommended diet for patients with diabetes has changed over time, and at any one time several views on best dietary practice may be found among experts. It may also be complicated by inadequate knowledge of dietary management and inadequate motivation of the patient.

Type 1 diabetes affects the quality of life of patients, because its treatments are burdensome both practically and emotionally. It has been shown that patients with better metabolic control, have less emotional problems than those with poor control. Others have shown that quality of life is lower in patients with more severe complications.

Thus the periodic evaluation of diabetes management including the current insulin and dietary regimen along with other indicators of the risk of complications in patients with type 1 diabetes seems necessary.

# Chapter 3

# Methods and Materials

### 3.1- Study Design

The names and addresses of patients with type 1 diabetes were obtained from the Diabetes Education Unit (DEU). Those who were recorded as having type 1 diabetes in the computerized list of DEU and who were in the age range of 18-40 years and had been diagnosed after 1984 in the Illawarra area were eligible for this study. This study was approved by the Wollongong University Ethics Comittee (Appendix 1).

A letter (Appendix 2) was sent to each patient. After one week all patients were contacted by phone. The patients who had changed their phone number and addresses were followed up using the Illawarra telephone directory, the electoral roll and medical records of the Wollongong Hospital. Some of them were followed up by writing letters to their doctors (Appendix 3)

After agreeing to participate in the study, each subject was asked to provide a non-diabetic friend of the same gender and within a 5% range of age. Not all the subjects could do so. Therefore the rest of the non-diabetic controls with the same age, sex and place of birth and residence were recruited by sending a message (Appendix 5) through University's electronic network, Wollongong Hospital and BHP (the Broken Hill Property Company Limited, the largest local employer). These were used to provide an as-near-as possible control subjects for the diabetic subjects. A letter was sent to each control (Appendix 4).

Each patient/control were given an appointment and asked to refer to either the Medical Research Unit or the outpatient clinic at the Wollongong Hospital. All subjects were nonfasting and were studied between 9:00 am to 5:00 pm. After

reading the information sheet (Appendices 6 and 7), subjects gave informed consent (Appendices 8 and 9).

Each individual was given a booklet. After filling out the demographic (Appendix 10), diabetic medical history (in case of diabetic subjects) (Appendix 11), socio-economic data (Appendix 12), food pattern (Appendix 13) and food preparation questionnaires (Appendix 14), each subject was interviewed about their diet and physical exercise. Dietary adherence, weight control, alcohol intake and physical exercise were recorded separately (Appendices 15-18). Then anthropometric and blood pressure measurements were performed, and blood and urine samples were collected.

### 3.2- Anthropometric Measurements

Weight, height, waist and hip circumferences, and skinfold thicknesses were measured and recorded on a form (Appendix 21).

Weight was measured using a beam scale (Seca 710) while the individual was in light clothing and without shoes, standing on the centre of the platform with the body weight evenly distributed on both feet and without touching anything else. Weight was recorded to the nearest 100 g (Gibson 1990, Fidanza 1991, Mahan and Arlin 1992, Scott et al. 1995).

Height was measured by a wall scale employing a steel tape ruled in centimetres and millimetres. An L-shaped bar was used as a measuring instrument which could make contact with the wall and the top of the subject's head. Height was measured without shoes to the nearest 0.5 cm (Gibson 1990, Fidanza 1991, Mahan and Arlin 1992, Scott et al. 1995).

Waist to hip circumference ratio (WHR) which is also called abdominal/gluteal ratio and distinguishes between android and gynoid obesity, was also determined. Hip circumference was measured at the widest circumference over the buttocks or trochanters. Waist was measured at the minimum

circumference of the mid section of the body at the level where the waist narrows, seen from the front. These circumferences were measured in standing subjects with a plastic tape-measure to the nearest 0.5 cm (Gibson 1990, Fidanza 1991, Mahan and Arlin 1992, Scott et al. 1995).

Subcutaneous fat (skinfold thickness) was measured by skinfold calipers. A Harpenden caliper was obtained from British Indicators Ltd, which has an easily read dial up to 40 mm. The measurements were done to the nearest 2 mm. All the measurements were the average of two readings on each site of the body. Measurements were made on the right side of the body, using biceps, triceps, subscapular and suprailiac skinfolds. The calipers were checked and calibrated periodically (Durnin and Wormersley 1974, Gibson 1990, Fidanza 1991, Mahan and Arlin 1992, Scott et al. 1995).

For the triceps measurement, the mid-point of the back of the upper arm between the tips of the olecranon and acromial processes was determined by measurement with the arm flexed at 90°. With the arm hanging freely at the side, the calipers were applied vertically above the olecranon at the marked level (Gibson 1990, Fidanza 1991, Mahan and Arlin 1992, Scott et al. 1995).

For the biceps measurement, the fold is picked up over the belly of the biceps muscle at the same level as the triceps, with the arm hanging freely and the palm facing outwards (Gibson 1990, Fidanza 1991, Mahan and Arlin 1992, Scott et al. 1995).

For the subscapular measurement, the fold is picked up just below the inferior angle of the scapula at 45° to the vertical along the natural cleavage lines of the skin (Gibson 1990, Fidanza 1991, Mahan and Arlin 1992, Scott et al. 1995).

The suprailiac measurement was taken just above the iliac crest, in the midaxillary line with the arm slightly abducted (Gibson 1990, Fidanza 1991, Mahan and Arlin 1992, Scott et al. 1995).

Body density (BD) of the subjects was calculated by linear equations (Durnin and Wormersley 1974) from sum of the all four skinfolds. Then the Siri Equation was used to calculate body fat per cent (Siri 1956).

Body Density (BD) = C-M x LOG 10 Skinfold sum (All four skinfolds)

C and M are the factors, which can be found from the table for males and females separately.

The Siri Equation:

$$FAT\% = \frac{(4.95)}{(BD)} - 4.5 \times 100$$

### 3.3- Blood Pressure

Blood pressure was measured with a standard mercury sphygmomanometer and the appropriately sized cuff, while the subject was sitting and after a five minute rest. The point at which repetitive, clear tapping sounds first appeared for at least two consecutive beats gave the systolic blood pressure. The diastolic blood pressure was measured at disappearance of the Korotkoff sounds (phase 5). Both measurements were taken to the nearest 2 mmHg (O'Brien and O'Malley 1991) by one observer. The results of blood pressure measurements were recorded on a form (Appendix 21).

### 3.4- Diet History

Diet history was assessed using the method of Burke (1947), which was used by the Diabetes Control and Complications Trial (DCCT) and its long-term reproducibility was demonstrated in patients with type 1 diabetes (Schmidt et al. 1994). This was done with the assistance of food and portion size models

(obtained from the Mentone Educational Centre, Victoria) and measuring cups and spoons. Diet checklist (Appendix 22), food pattern and food preparation questionnaires were used as a complementary devices (the DCCT forms were modified and changed to Australian version). Diet history was taken and recorded on a form (Appendix 23). It was attempted to establish the subjects' usual pattern of eating both at meal times and between meals. All the data were later analysed and nutrient intakes assessed using version 4 of the software program Diet 1, which utilises the Australian NUTTAB 1995 food composition database. Each diet history was assessed for basic food groups consumption and their variety (Chippindall et al. 1992 and Wahlqvist and Kouris-Blazos 1997).

### 3.5- Blood Tests

Subjects were in a non-fasted state for blood collection. Blood was collected from the vein and in tubes containing EDTA. Glucose, HbA1c, lipids, fibrinogen, albumin and creatinine were measured in these samples. Blood tests were partially done at the Medical Research Unit and the rest were performed at the biochemical and haematology laboratories at the Wollongong Hospital.

Plasma glucose was assayed colorimetrically by the glucose oxidase/peroxidase (Trinder 1969, Curme et al. 1978) on a Kodak Ektachem 700xR Analyzer (Kodak, USA).

HbA1c was measured by a high pressure liquid chromatography method (Bio-Rad Haemoglobin A1c Micro-column Test Instruction Manual 1990). The column was a Pharmacia Mono S HR 5/5 column and the mobile phase was buffered sodium malonate and lithium chloride. The detector was an ETP Kortek 65B UV set at 405 nm; 0.01 AUFS. This measurement was performed at the biochemistry laboratory of Wollongong Hospital on whole blood samples.

For the lipid and lipoprotein assessments, tubes containing EDTA (1.6mg/ml, Monovette Sarstedt, Germany) were used. The blood samples were

centrifuged in a swinging bucket centrifuge (Sorvall Instruments, USA) at 1500xg for 20 minutes to yield plasma. Samples were assayed on a fresh sample or kept at -85C° prior to analysis. Total cholesterol was assayed enzymatically (Allain et al. 1974) using reagent kits (Boehringer Mannheim Germany). This was then determined on a COBAS FARA analyzer (Hoffman-La Roche, Switzerland). HDL cholesterol was assayed after the addition of dextran sulphate-Mg chloride solution to the plasma sample, to precipitate apolipoprotein B-containing lipoproteins (Warnick et al. 1982). The supernatant liquid containing the HDL fraction was then quantified using a routine cholesterol analysis. LDL cholesterol was calculated using the modified Friedewald formula (Samman and Truswell 1993). Triglyceride in plasma sample was hydrolyzed enzymatically (Eggstein 1966) by reagent kits (Boehringer Mannheim, Germany) and determined by colorimetry on a COBAS FARA analyzer.

Serum creatinine was determined by an enzymatic method using a Kodak Ektachem Clinical Chemistry Slide (CREA). This slide contains a dry, multilayered analytical element coated on a transparent polyester support. The analysis is performed as a two-point rate measurement. The rate determining step is the hydrolysis of creatinine to creatine by creatinine amidohydrolase. The creatine is then converted to a dye whose rate of formation is proportional to the concentration of creatinine in the specimen (Eastman Kodak Company 1987, Ambrose et al. 1983).

Plasma albumin was measured by a colorimetric method. The Kodak Ektachem Clinical Chemistry Slide (ALB) was used. This slide contains a dry, multilayered analytical element coated on a clear polyester support. The analysis is based on the binding of bromcresol green dye to albumin, resulting in a substantial shift in the wavelength of light absorbed by the free dye. The density of the albumin-bound dye is related to the concentration of albumin in the sample and is measured spectrophotometrically at 630 nm and reported in grams per decilitre (Eastman Kodak Company 1986).

Fibrinogen was measured by the Electra 1000 C Automatic Coagulation Timer (MLA) on plasma samples that has been collected into sodium citrate tubes (i.e. one part citrate + nine parts blood). In the presence of a high concentration of thrombin, the thrombin clotting time is inversely proportional to the fibrinogen concentration of plasma. When the final concentration of fibrinogen is in the range of 1.0-4.0 g/l a linear relationship exists between the elapsed time for clot formation and the concentration of fibrinogen (Corriveau and Fritsma 1988).

### 3.6- Urine Tests

Albumin and creatinine were determined on spot and early morning urine samples at the biochemical laboratory of the Wollongong Hospital. The urine sugar and ketones were tested by reagent strips at the Medical Research Unit.

The urinary albumin excretion rate was assessed on a fresh spot sample on the day of the study. Micro Albumin Reagent, when used in conjunction with Beckman Specific Protein Analyzer and Calibrator 4 is intended for quantitative determination of micro quantities of albumin in urine by rate nephelometry. The method employed in the Beckman MA Test measures the rate of increase in light scattered from particles suspended in solution as a result of complexes formed during an antigen-antibody reaction (Beckman, MA micro Albumin instructions, 1990). Urinary albumin excretion was expressed as a function of urine creatinine.

Urinary creatinine was determined by an enzymatic method using Kodak Ektachem Clinical Chemistry Slide (CREA). The method was similar to the measurement of creatinine in plasma samples.

Urine sugar and ketones (acetoacetic acid) were tested by Ames Multiple Reagent Strips (Bayer Diagnostics). Results were recorded on the fresh spot urine sample and the reading of each reagent was at the time specified on the colour chart. The test was repeated when a positive result was obtained.

The results of biochemical assays and the diet history were sent to all the subjects (and their doctors if they wished) (Appendices 24 - 28).

### 3.7- Quality of Life

Two measures of quality of life were applied in combination to examine quality of life of people with diabetes: Diabetes Quality of Life Measure (DQOL) which was designed for the Diabetes Control and Complications Trial (The DCCT Research Group 1988c) and the Medical Outcome Study Health Survey 36-Item Short Form (SF-36) (Ware and Sherbourne 1992) (Appendices 19 and 20). Control subjects were asked to complete the SF-36 only. The following formula was used to score both measures (Ware et al, 1993, Jacobson and the DCCT Research Group 1995):

$$\text{Transformed Scale} = \left[\frac{(\text{Actual raw score} - \text{lowest possible raw score})}{\text{Possible raw score range}}\right] \text{X } 100$$

Using this method DQOL and SF-36 scores were arithmetically transformed to a 100-point scale (100 reflects the highest possible quality of life score and 0 the lowest possible quality of life).

### 3.8- Statistical Analysis

The results were analyzed with the computer package JMP version 3. Unpaired Student's t-test or Wilcoxon rank sum test where appropriate were applied to determine the significance differences between the means of continuous and discontinuous variables. The chi-squared test was used to compare the proportions and categories. Log-transformation was used for the albumin:

creatinine ratio with skewed distribution. Pearson's correlation coefficient was used to show the association between two variables.

Multiple regression analysis using stepwise regression was performed to identify significant independent predictors of diabetic control. Analysis of covariance was also used to assess the significanceof interaction between diabetic and control groups. In this situation where we have two groups of patients and controls, an explanatory variable can have a different effect on the predicted response Y depending on the value of a second explanatory variable. To model an effect of this type, we create what is known as an interaction term. This is examined by creating a new variable which is their product and adding this to the model (Pagano and Gauvreau 1993, Altman 1995).

Education and income level were entered into the models as a categorical variables (three groups for each) (Altman 1995). Values for $p<0.05$ were considered significant for all results. Internal consistency between subscales of the DCCT quality of life measure and the SF-36 were examined by Cronbach's $\alpha$ (Anastasi and Urbina 1997).

# Chapter 4

# Results

The results of this study will be presented in different sections. Measurements including frequency, mean and standard deviation of the values are given, and any differences between findings in patients with type 1 diabetes and control subjects are examined.

It will begin with the general characteristics of the recruited subjects distinguishing between people with diabetes and controls. Data from subjects with diabetes are then analysed to find out about the significant differences in their glycemic control in regard to the independent variables like age, sex, duration of diabetes, dietary intake and adherence and other predictors of glycemic control. This also includes anthropometric measurements and results from blood and urine analysis described for both diabetic and control subjects.

Dietary intake of people with diabetes and control subjects were compared with the Australian dietary guidelines and recommended dietary intakes and their differences are tested statistically. Diet quality of the diabetic and control subjects on the basis of basic food group consumption and dietary score are shown and the differences found by contingency table analysis and chi square indicated.

Qualities of life of diabetic and control subjects will be presented by two measures. The pairwise correlation of quality of life subscales with one another and with other variables will be revealed separately and the fitted models will be illustrated. Internal consistency between subscales will be shown by Cronbach's alpha. At the end of each section, associations of dependent variables (those which are related to diabetic control) on several independent variables will be tested and presented in regression models.

## 4.1- Population Characteristics

There were 130 patients with type 1 diabetes in the computerized list of the Diabetes Education Unit (DEU) who had been diagnosed since 1984 and prior to the beginning of the study (September 1995). These patients were in the age range of 18 to 40 years at the time of recruiting. A letter (Appendix 2) was sent to all of them and they were contacted by phone (address and phone number were provided by the DEU list). Those who did not answer were sought through the hospital and endocrinologist registration lists, phone directory and electoral roll. Fifty-six individuals with type 1 diabetes participated in the study. This number represents 65.1 percent of the original list who were available.

**Table 2- Non participant subjects**

| Reason for not participating | Number |
|---|---|
| Moved to another place | 11 |
| Not willing to participate | 26 |
| Changed their residences (unable to contact) | 27 |
| Patients with type 2 diabetes | 3 |
| Non-diabetic | 1 |
| Studying for Higher School Certificate | 4 |
| Health problem [a] | 2 |

[a] One individual was quadriplegic and one had a thyroid problem

Each one of the patients was asked to provide a friend or a distant relative with similar sex and age and without diabetes. The patients recruited 16 controls. An additional 32 people took part in the study after the messages (Appendix 5) were sent through the university network, hospital, and BHP.

Table 3 summarizes the demographic characteristics of the patients and controls. It should be mentioned that one individual from the patient group and one from the control group were excluded as they were outliers regarding body

weight, biochemical parameters and underreporting their dietary intake. Also these two people found to have not answered the questionnaires, quality of life and SF-36 forms completely. Therefore results presented are for 55 patients and 47 controls.

There were no significant differences between the two groups in the numbers of females and males. In both groups there were more males than females. The mean of ages in each group differed, although ranges overlapped. The effect of the age was found on each variable by a series of dummy variables in each regression model. In the diabetic group there were more married and fewer singles than in the control group.

The educational level of both groups was compared and no significant differences were found between the groups. The number of subjects in each income level was computed and statistical analysis by contingency table did not show any significant difference between them for the two groups. . Not all of the subjects answered to the questions related to the education and income level.

Most participants were born in Australia. There were no significant differences between the number of Australian and non-Australian parents and grandparents in the groups. Among patients 5 were born in England, 2 in Poland, 1 in New Zealand, 1 in Macedonia and 1 in Uruguay. Four controls were born in England, 1 in New Zealand and 1 in France. All patients and controls had been Australian resident for at least 14 years.

**Table 3- Demographic characteristics of the subjects**

| Variables | Diabetic n=55 | Control n=47 | p value |
|---|---|---|---|
| Sex n (%) | | | 0.71 |
| Female | 18 (32.7) | 17 (36.2) | |
| Male | 37 (67.3) | 30 (63.8) | |
| Age (yr.) | | | <0.01 |
| Mean±SD | 31.24±6.10 | 27.72±5.07 | |
| range(yr) | 18-40 | 18-40 | |
| Marital status n (%) | | | 0.23 |
| single | 15 (27.3) | 20 (42.5) | |
| married | 36 (65.4) | 23 (48.9) | |
| separated | 4 ( 7.3) | 4 ( 8.6) | |
| Education* n (%) | | | 0.26 |
| commenced high school | 4 ( 7.3) | 1 ( 2.1) | |
| finished high school | 15 (27.3) | 12 (25.5) | |
| tertiary schooling started | 14 (25.4) | 8 (17.1) | |
| tertiary schooling finished | 21 (38.2) | 26 (55.3) | |
| no answer | 1 ( 1.8) | 0 ( 0.0) | |
| Income ($/yr.) n (%) | | | 0.64 |
| less than 12000 | 3 ( 5.5) | 2 ( 4.3) | |
| 12000-15000 | 2 ( 3.6) | 4 ( 8.5) | |
| 15001-18000 | 1 ( 1.8) | 2 ( 4.3) | |
| 18001-22000 | 1 ( 1.8) | 2 ( 4.3) | |
| 22001-26000 | 3 ( 5.5) | 3 ( 6.3) | |
| 26001-32000 | 5 ( 9.1) | 2 ( 4.3) | |
| 32001-40000 | 5 ( 9.1) | 4 ( 8.5) | |
| 40001-50000 | 9 (16.4) | 6 (12.7) | |
| 50001 and over | 18 (32.7) | 20 (42.5) | |
| no answer | 8 (14.5) | 2 ( 4.3) | |
| Country of Birth n (%) | | | 0.45 |
| Australia | 45 (81.8) | 41 (87.2) | |
| Not-Australia | 10 (18.2) | 6 (12.8) | |

* There was nobody at the levels of commenced and finished primary school.

The date of diagnosis of diabetes was recorded on the computerized list of DEU and also was asked from the patients with type 1 diabetes. The data on the diabetes history of the patients are summarized in table 4.

The duration of diagnosis ranged from one to twelve years. All of the patients were receiving insulin injections. Two female and 5 male diabetic subjects

were taking oral drugs as well. The number of injections was 2, 3 or more times per day for 49 patients (6 did not answer to this question). Seven females and 11 males had been hospitalized for ketoacidosis. Thirty-three patients wanted their results to be sent to their doctors, 20 of them preferred not to do so and 3 of them did not answer the question.

**Table 4- Type 1 diabetes history**

| | female n=18 | male n=37 | total n=55 |
|---|---|---|---|
| Duration of diabetes (yr.) n (%) | | | |
| 1-3 | 2 | 11 | 13 ( 23.6) |
| 4-6 | 7 | 9 | 16 ( 29.1) |
| 7-9 | 4 | 7 | 11 ( 20.0) |
| 10-12 | 5 | 10 | 15 ( 27.3) |
| Total | 18 | 37 | 55 (100.0) |
| mean±SD | 7.27±2.82 | 5.97±3.93 | 6.40±3.35 |
| Oral drugs taken n (%) | 2 | 4 | 6 (10.9) |
| Taking insulin n (%) | 18 | 37 | 55 (100) |
| Number of insulin injection n (%) | | | |
| two | 8 | 22 | 30 (54.5) |
| three or more | 9 | 11 | 20 (36.4) |
| no answer | 2 | 3 | 5 ( 9.1) |
| Insulin dose (units/day) mean±SD | 50.80±18.33 | 51.66±24.28 | 51.06±22.51 |
| Insulin dose (units/kg body weight) mean±SD | 0.74±0.28 | 0.66±0.29 | 0.68±0.29 |
| Hospitalized for ketoacidosis n (%) | 7 | 11 | 18 (32.7) |
| Diabetes in the family n (%) | 8 | 21 | 29 (51.8) |

Questions related to the several possible complications were asked in the questionnaire (Appendix 11). Not many patients had complications related to the eye, kidney and cardiovascular system. The results are tabulated in table 5.

**Table 5- Complications survey**

| | no | yes | unknown | not answered |
|---|---|---|---|---|
| **Eye problems** | 51 | 2 | 1 | 1 |
| **Kidney problems** | 53 | 1 | 0 | 1 |
| protein/albuminuria | 45 | 3 | 4 | 3 |
| kidney transplant | 52 | 0 | 0 | 3 |
| kidney dialysis | 52 | 0 | 0 | 3 |
| **Cardiovascular problems** | 51 | 2 | 1 | 1 |
| Abnormal ECG | 51 | 0 | 3 | 4 |
| heart pain or angina | 49 | 2 | 1 | 3 |
| heart attack | 52 | 0 | 0 | 3 |
| bypass surgery | 52 | 0 | 0 | 3 |
| stroke | 53 | 0 | 0 | 2 |
| blood pressure(BP) | 47 | 5 | 2 | 2 |
| BP treatment | 52 | 1 | 0 | 2 |
| circulation in legs | 53 | 1 | 0 | 1 |
| foot ulcers | 51 | 2 | 0 | 2 |
| gangrene | 52 | 0 | 1 | 2 |
| amputation | 54 | 0 | 0 | 1 |
| **Other diseases** | 46 | 6 | 0 | 3 |

### 4.2-Anthropometric Measurements and Blood Pressure

The results of anthropometric measurements and systolic and diastolic blood pressure measured in sitting position of subjects are shown in Table 6 (1 patients refused to have the measurements). There were no significant differences in blood pressure between diabetic subjects and controls (same sex comparisons).

As revealed in table 6 body mass index (BMI) ($kg/m^2$) calculated from the weights and heights of the subjects were not significantly different in same sex comparisons of diabetic and non-diabetic subjects.

The observed frequencies for categories of BMI (underweight, normal, overweight and obese) in diabetics and controls are shown in Table 7. There were no significant differences between frequencies in diabetic and control subjects (same sex comparisons).

Body fat percent was calculated from skinfold thickness measurement at four sites. In both groups females had a significantly higher body fat percent than males ($p<0.0001$ females vs. males in both groups). There were no significant differences between body fat percent in diabetic and control subjects (same sex comparisons) ($p=0.17$ and $p=0.67$ for females and males respectively).

Males in both groups had higher WHR (waist to hip ratio) than females ($p<0.0001$). No significant difference was found when females and males were compared in two groups ($p=0.33$ and $p=0.12$ for females and males respectively).

As expected, males in both groups had significantly higher systolic blood pressure compared to females ($p=0.01$ in diabetic subjects and $p=0.04$ in control subjects). There was no significant difference between similar sexes in two groups ($p=0.09$ and $p=0.83$ for females and males respectively).

Diastolic and systolic blood pressure was significantly higher in males than in females in both groups ($p=0.02$ in diabetic subjects and $p=0.05$ in controls). No significant difference was found between females and males from two groups ($p=0.41$ for females and $p=0.37$ for males).

In the diabetic group 11.1% (one female and 5 males) had a diastolic blood pressure of 95 mmHg or more. This figure was 2.2% in controls (one male). Two diabetic males (3.7%) and 1 control male (2.1%) had a systolic blood pressure higher than 160 mmHg.

**Table 6- Anthropometric measurements and blood Pressure in diabetic and control subjects**

| | Diabetic n=54 | | Control n=47 | |
|---|---|---|---|---|
| | Mean±SD | 95% CI | Mean±SD | 95% CI |
| BMI(kg/m$^2$) | | | | |
| female | 26.01±3.47 | 24.23-27.80 | 23.86±4.25 | 21.67-26.04 |
| male | 25.70±3.97 | 24.38-27.03 | 26.11±3.70 | 24.73-27.79 |
| Body fat% | | | | |
| female | 29.9±2.45 | 28.55-31.26 | 27.15±5.90 | 24.11-30.18 |
| male | 18.83±5.14 | 17.12-20.55 | 18.32±4.52 | 16.64-20.01 |
| WHR[a] | | | | |
| female | 0.76±0.07 | 0.72-0.80 | 0.74±0.04 | 0.72-0.76 |
| male | 0.88±0.06 | 0.86-0.90 | 0.90±0.07 | 0.88-0.93 |
| sBP[b] (mmHg) | | | | |
| female | 113.94±9.73 | 108.94-118.94 | 117.23±7.41 | 113.42-121.05 |
| male | 125.43±16.35 | 119.98-130.88 | 124.66±12.99 | 119.81-129.52 |
| dBP[c](mmHg) | | | | |
| female | 67.94±12.38 | 61.57-74.31 | 69.70±8.92 | 65.12-74.29 |
| male | 76.97±13.74 | 72.39-81.55 | 74.50±7.23 | 71.79-77.20 |

[a] Waist hip ratio. [b] systolic blood pressure. [c] diastolic blood pressure.

**Table 7- Observed frequencies of BMI (kg/m$^2$) in diabetic and control subjects**

| | Diabetic n (%) | | | Control n(%) | | |
|---|---|---|---|---|---|---|
| | total n=54 | female n=17 | male n=37 | total n=47 | female n=17 | male n=30 |
| <20 [a] | 0 | 0 | 0 | 4 ( 8.5) | 3 (17.6) | 1 ( 3.3) |
| 20-25 [b] | 27 (50.0) | 8 (47.0) | 19 (51.4) | 18 (38.4) | 6 (35.3) | 12 (40.0) |
| 25-30 [c] | 23 (42.6) | 7 (41.2) | 16 (43.2) | 19 (40.4) | 7 (41.2) | 12 (40.0) |
| >30 [d] | 4 ( 7.4) | 2 (11.8) | 2 ( 5.4) | 6 (12.7) | 1 ( 5.9) | 5 (16.7) |

[a] underweight. [b] acceptable weight. [c] overweight. [d] obese.

Allowing for interaction between two groups and adjusting for age and sex, stepwise regression analysis of systolic blood pressure on anthropometric measurements showed that BMI was the only variable that entered into the model. A similar regression model was constructed to indicate the association of anthropometric measurements and diastolic blood pressure in two groups. The

result showed that WHR was the only independent variable among anthropometric measurements, which had significant effect on diastolic blood pressure (table 8). Regression analysis did not show any effect of diabetes data (duration of diabetes, number of insulin injections and insulin dose) on blood pressure in diabetic subjects.

**Table 8- Results from regression analysis of systolic and diastolic blood pressure on anthropometric measurements in diabetic and control subjects**

| | sBP | | dBP | |
|---|---|---|---|---|
| | model 1 | model 2 | model 1 | model 2 |
| Intercept | 125.29** | 105.07** | 77.35** | 44.71** |
| group | 0.00 | -8.40 | -0.83 | 22.60 |
| age | -0.17 | -0.16 | -0.17 | -0.12 |
| sex | 4.77** | 4.36 | 3.50** | 0.83 |
| sexxgroup | - | (•) | - | (•) |
| agexgroup | - | 0.29 | - | (•) |
| BMI | - | 0.80* | - | (•) |
| BMIxgroup | - | (•) | - | (•) |
| body fat% | - | (•) | - | (•) |
| body fat%xgroup | - | (•) | - | (•) |
| WHR | - | (•) | - | 38.34* |
| WHRxgroup | - | (•) | - | -28.09* |
| N | 101 | 101 | 101 | 101 |
| $R^2$ | 0.11 | 0.17 | 0.09 | 0.17 |
| Adjusted $R^2$ | 0.08 | 0.13 | 0.06 | 0.13 |
| p value of whole model | <0.01 | <0.01 | <0.05 | <0.01 |

The figures in front of each variable are the estimates.- not available in this model. (•) available but not statistically significantly contributing to the model.
** $p<0.01$. * $p<0.05$.

### 4.3-Biochemical Results of Blood Samples

The levels of HbA1c%, glucose, lipids and lipoproteins, creatinine, albumin and fibrinogen that have been determined in non-fasting blood samples (4 patients did not give blood) are shown in table 9. These are the results of blood samples of 51 patients (35 males and 16 females) and 47 control subjects (30 males and 17 females).

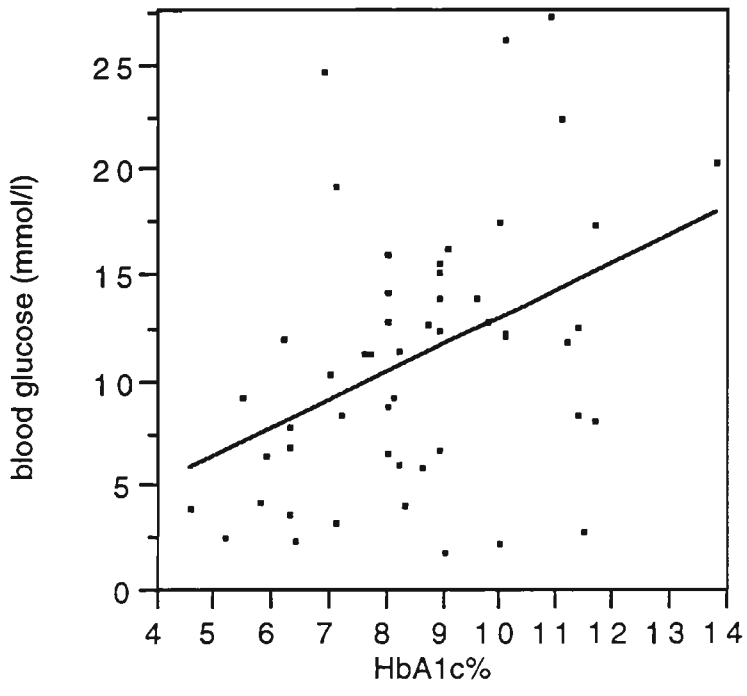
**Table 9-Blood results in diabetic and control subjects**

| | Diabetic n=51 | | Control n=47 | |
|---|---|---|---|---|
| | mean±SD | 95% CI | mean±SD | 95% CI |
| HbA1c% | | | | |
| female | 8.52±2.21** | 7.33-9.69 | 5.22±0.56 | 4.93-5.51 |
| male | 8.56±1.91** | 7.91-9.22 | 5.39±0.55 | 5.19-5.60 |
| Glucose (mmol/l) | | | | |
| female | 9.52±6.33* | 5.85-13.19 | 4.99±0.63 | 4.66-5.31 |
| male | 11.84±6.00** | 9.78-13.90 | 5.13±0.72 | 4.86-5.40 |
| Cholesterol (mmol/l) | | | | |
| female | 4.47±0.68 | 4.11-4.83 | 4.67±1.03 | 4.15-5.20 |
| male | 4.78±1.21 | 4.36-5.19 | 5.17±1.11 | 4.75-5.58 |
| HDL cholesterol (mmol/l) | | | | |
| female | 1.65±0.55 | 1.36-1.94 | 1.57±0.29 | 1.42-1.71 |
| male | 1.32±0.49* | 1.15-1.49 | 1.06±0.27 | 0.96-1.16 |
| LDL cholesterol (mmol/l) | | | | |
| female | 2.35±0.43 | 2.12-2.58 | 2.64±1.08 | 2.08-3.20 |
| male | 2.88±0.98 | 2.54-3.21 | 3.27±0.90 | 2.93-3.60 |
| Triglyceride (mmol/l) | | | | |
| female | 1.03±0.66 | 0.67-1.38 | 1.01±0.34 | 0.83-1.19 |
| male | 1.26±0.77 | 0.99-1.52 | 1.83±1.09* | 1.42-2.24 |
| Creatinine (µmol/l) | | | | |
| female | 66.62±8.50 | 62.09-71.15 | 75.65±9.90* | 70.55-80.74 |
| male | 80.48±9.39 | 77.26-83.71 | 92.80±13.98* | 87.58-98.02 |
| Albumin (g/l) | | | | |
| female | 41.00±2.96 | 39.42-42.58 | 41.97±2.63≈ | 41.89-43.87 |
| male | 42.20±2.95 | 41.19-43.21 | 43.53±2.91 | 42.44-44.62 |
| Fibrinogen (g/l) | | | | |
| female | 2.89±0.57 | 2.59-3.19 | 2.42±0.69≈ | 2.06-2.77 |
| male | 2.54±0.84 | 2.23-2.81 | 2.11±0.59≈ | 1.88-2.32 |

** $p<0.0001$. * $p<0.01$. ≈ $p<0.05$ (p values are for same sex comparisons).

As shown in table 9, blood glucose was higher in male than in female diabetic subjects ($p=0.23$). There was no significant difference in HbA1c% between female and male diabetic subjects ($p=0.93$). As it was expected these values were significantly higher in diabetic subjects compared with control subjects ($p<0.0001$ for both females and males). There was a positive correlation between HbA1c% and blood glucose in patients with diabetes ($p<0.01$ and Pearson correlation =0.41) in spite of blood glucose being measured non-fasting (figure 2).

**Figure 2- HbA1c% by blood glucose in diabetic subjects***




*Note: The line is called fit line, which finds β0 and β1 for the straight line that fits the points to minimize the residual sum of squares.

Stepwise regression analysis was used to examine the relationship between education and income level with HbA1c% in diabetic patients when the model was adjusted for age and sex. Income level did not enter into this model while education entered but did not contribute significantly in the model ($p<0.1$). The result of this analysis is shown in table 10.

The similar analysis also indicated that none of the variables related to diabetes history i.e. duration of diabetes, dose of insulin and number of insulin injections had an independent significant association with HbA1c% when the models were adjusted for age and sex. But when education and income level were entered into the model, both of them had some but insignificant effects ($R^2=0.19$ and $p<0.05$ for the whole model).

**Table 10- Results from multiple regression analysis of HbA1c% on education and income level in diabetic subjects**

| | model 1 | model 2 |
|---|---|---|
| Intercept | 11.68** | 11.40** |
| sex | 0.02 | -0.05 |
| age | -0.10* | -0.09* |
| Education (3-2&1) | - | -0.48 |
| Education (1-2) | - | (•) |
| Income (3&2-1) | - | (•) |
| Income (3-2) | - | (•) |
| N | 51 | 51 |
| $R^2$ | 0.09 | 0.15 |
| Adjusted $R^2$ | 0.05 | 0.09 |
| p value of whole model | <0.1 | <0.1 |

The figures in front of each variable are the estimates.- not available in this model. (•) available but not entered into the model. ** $p<0.01$. * $p<0.05$.

In both groups HbA1c% significantly decreased with age ($p=0.03$ and Pearson correlation=-0.30 for diabetic subjects and $p=0.04$ and Pearson correlation=-0.29 for control subjects). When this association was adjusted for BMI in a regression model, this effect was not longer observed.

All the variables like anthropometric measurements, blood pressure, diabetes history, and the blood results were used as an independent variable in several models with HbA1c% in the diabetic group. No association was found.

No significant differences were observed between lipid and lipoprotein levels in female subjects in two groups (table 9). However male diabetics had significantly higher HDL cholesterol ($p=0.01$) and lower triglyceride ($p=0.01$) than male controls. In both groups females had significantly higher HDL cholesterol ($p<0.05$ and $p<0.001$ respectively for diabetic and control subjects) and lower LDL cholesterol ($p=0.05$ in diabetic and $p<0.05$ in control subjects) than males.

There were no significant differences between cholesterol and triglyceride levels in female and male subjects with diabetes but in control subjects the triglyceride level was significantly higher in male subjects compared to females. The mean of lipid and lipoprotein levels in both groups were within reference ranges (Appendix 27).

Four regression models that allowed for interaction within two groups, were performed for blood lipids with anthropometric measurements as independent variables while adjusted for age and sex ($p<0.01$ for the whole models). The models indicated that at least two of these measurements had some positive effects in the prediction of total cholesterol, LDL cholesterol and triglyceride while BMI had negative effect on HDL cholesterol in diabetic and control subjects (table 11). It should be mentioned that except the negative effect of BMI on HDL cholesterol the effect of other variables on blood lipids and lipoproteins did not reach to a significant level.

**Table 11- Results from multiple regression analysis of blood lipids and lipoproteins on the anthropometric measurements in diabetic and control subjects**

| | Total cholesterol | | HDL cholesterol | | LDL cholesterol | | Triglyceride | |
|---|---|---|---|---|---|---|---|---|
| | model 1 | model 2 | model 1 | model 2 | model 1 | model 2 | model 1 | model 2 |
| Intercept | 4.03** | 1.03 | 1.38** | 2.00** | 2.34** | -0.50 | 0.82 | -2.76 |
| group | 0.20 | 0.22* | -1.10* | -0.10* | 0.21* | -0.89 | 0.21 | 0.21 |
| sex | 0.19 | 0.22 | -0.21** | -0.19** | 0.28** | 0.28 | 0.26 | 0.04 |
| age | 0.02 | 0.01 | 0.00 | 0.00* | 0.01 | 0.00 | 0.01 | 0.01 |
| sexxgroup | - | (•) | - | (•) | - | (•) | - | (•) |
| agexgroup | - | (•) | - | (•) | - | (•) | - | (•) |
| BMI | - | (•) | - | -0.03* | - | 0.01 | - | 0.06 |
| BMIxgroup | - | (•) | - | (•) | - | 0.04 | - | (•) |
| Body fat% | - | 0.04 | - | (•) | - | 0.03 | - | (•) |
| Body fat%xgroup | - | (•) | - | (•) | - | (•) | - | (•) |
| WHR | - | 2.87 | - | (•) | - | 2.25 | - | 2.69 |
| WHRxgroup | - | (•) | - | (•) | - | (•) | - | (•) |
| N | 98 | 97 | 98 | 97 | 98 | 97 | 98 | 97 |
| $R^2$ | 0.06 | 0.15 | 0.20 | 0.27 | 0.12 | 0.24 | 0.13 | 0.27 |
| Adjusted $R^2$ | 0.03 | 0.11 | 0.41 | 0.24 | 0.09 | 0.18 | 0.10 | 0.23 |
| p value of whole model | <0.1 | <0.01 | <0.01 | <0.01 | <0.01 | <0.01 | <0.01 | <0.01 |

The figures in front of each variable are the estimates.- not available in this model. (•) available but not entered into the model. ** $p<0.01$. * $p<0.05$.

As shown in table 9 blood creatinine and albumin levels were lower in diabetic compared to control subjects. Females and males in the control group had significantly higher creatinine level than females and males in the diabetic group ($p=0.01$ and $p<0.001$ for females and males respectively).

Blood albumin was significantly higher among female control subjects (p=0.04) than female diabetic subjects (table 9). Creatinine and albumin levels were higher among males than in females in both groups. This difference was significant for creatinine (p<0.001 males vs females in both groups). But all the values for creatinine and albumin in both groups were within the normal range (Appendix 27).

Diabetes history (including duration of diabetes, number of insulin injections and insulin dose), body measurements, blood pressure and biochemical variables did not enter into a model with creatinine. Similar results was found from regression analysis of blood albumin on different variables, except when the regression analysis was done with blood lipids and lipoproteins which HDL cholesterol found to have negative effect in the model (table 12).

**Table12- Results from multiple regression analysis of albumin on blood lipids and lipoproteins in diabetic subjects**

| | model 1 | model 2 |
|---|---|---|
| Intercept | 45.50* | 49.26 |
| sex | 0.59 | -0.22 |
| age | -0.12 | -0.13* |
| Total cholesterol | - | (•) |
| HDL cholesterol | - | -2.27** |
| LDL cholesterol | - | (•) |
| Trglyceride | - | (•) |
| N | 51 | 51 |
| $R^2$ | 0.10 | 0.25 |
| Adjusted $R^2$ | 0.06 | 0.20 |
| p value of whole model | <0.1 | <0.01 |

The figures in front of each variable are the estimates.- not available in this model. (•) available but not entered into the model. ** p <0.01. * p<0.05.

As it is shown in table 9, diabetic subjects had significantly higher fibrinogen than control subjects (p=0.04 for females and p=0.02 for males), although for both groups these values were within normal range.

In subjects with diabetes the level of fibrinogen was not related significantly to age and HbA1c%, but it rose with increasing body mass index (p<0.01). Similar associations could not be found within control group.

Regression analysis of fibrinogen on diabetes history did not show any significant associations when the model was adjusted for age and sex. But with anthropometric measurements, WHR and body fat% entered into the model. Only body fat% had significant effect in the model (table 13).

No association was found when a model was fitted for fibrinogen and blood pressure, adjusted for age, sex and HbA1c% in diabetic group.

Stepwise regression analysis of fibrinogen on blood lipids and lipoproteins in diabetic and control groups showed that allowing for interaction between two groups and adjusting for age and sex, triglyceride had significant positive effect ($p<0.001$) and HDL cholesterol negative but insignificant effect in the model (table 14).

**Table 13- Results from multiple regression analysis of fibrinogen on anthropometric measurements in diabetic subjects**

| | model 1 | model 2 |
|---|---|---|
| Intercept | 1.89** | -0.94 |
| sex | -0.18 | 0.02 |
| age | 0.02 | 0.02 |
| BMI | - | (•) |
| Body fat% | - | 0.05* |
| WHR | - | 2.12 |
| N | 51 | 51 |
| $R^2$ | 0.09 | 0.23 |
| Adjusted $R^2$ | 0.05 | 0.17 |
| p value of whole model | <0.1 | <0.05 |

The figures in front of each variable are the estimates.- not available in this model. (•) available but not entered into the model.. ** $p<0.01$. * $p<0.05$.

**Table 14- Results from multiple regression analysis of fibrinogen on blood lipids and lipoproteins in diabetic and control subjects**

| | model 1 | model 2 |
|---|---|---|
| Intercept | 2.01** | 2.20** |
| group | -0.18 | 0.75* |
| sex | -0.17 | -0.27** |
| age | 0.02 | 0.01 |
| sexxgroup | - | (•) |
| agexgroup | - | (•) |
| Total cholesterol | - | (•) |
| Total cholesterolxgroup | - | (•) |
| HDL cholesterol | - | -0.29 |
| HDL cholesterolxgroup | - | -0.40* |
| LDL cholesterol | - | (•) |
| LDL cholesterolxgroup | - | (•) |
| Triglyceride | - | 0.30** |
| Triglyceridexgroup | - | -0.37** |
| N | 98 | 98 |
| $R^2$ | 0.14 | 0.37 |
| Adjusted $R^2$ | 0.12 | 0.32 |
| p value of whole model | <0.01 | <0.001 |

The figures in front of each variable are the estimates.- not available in this model. (•) available but not entered into the model. ** p <0.01. * p<0.05.

## 4.4- Biochemical Results of Urine Sample

Spot urine samples were tested for sugar and ketones by reagent stripes. Thirty-seven urine samples of diabetic subjects were sugar positive (from + to ++++) and 10 were ketone positive (trace). None of the urine samples of controls reacted positive to either sugar or ketone tests.

Albumin and creatinine were measured in both spot and early morning urine samples of diabetic and control subjects. The ratio between these two values was calculated as albumin (mg/l)/creatinine (mmol/l).

The concentrations of albumin and creatinine on spot urine samples of diabetic and control groups are shown in table 15. The ratio of albumin/creatinine that is also indicated in the table was logarithmically transformed before analysis because of its positively skewed distribution and then changed to actual figure.

This ratio was significantly higher in diabetic group than in control group ($p<0.001$).

**Table 15- Albumin, creatinine and their ratio in spot urine sample in diabetic and control subjects**

| | diabetic n=51 | control n=47 |
|---|---|---|
| albumin (mg/l) | | |
| mean±SD | 11.76±19.07 | 7.38±4.36 |
| 95%CI | 2.67-17.13 | 6.09-8.66 |
| creatinine (µmol/l) | | |
| mean±SD | 10456.18±6462.62 | 14012.49±6650.46 |
| 95%CI | 8647.54-12282.81 | 12089.21-15935.77 |
| Albumin (mg/l)/creatinine (mmol/l) | | |
| mean±SD | 0.79±2.04* | 0.51±1.62 |
| 95%CI | 0.64-0.95 | 0.44-0.59 |

* $p<0.001$.

Early morning urine samples were collected from 29 patients and 28 controls. The rest of the subjects did not bring their samples. The results of albumin, creatinine and the ratio are presented in table 16. The ratio in this table was logged and the values presented are transformed to actual figures.

There was a positive correlation between the mean of logged albumin/creatinine ratio in spot urine sample of those who had collected early morning urine sample and the ratio of the spot urine sample of the whole group ($p<0.005$ and Pearson correlation=0.53 for diabetic subjects and $p<0.05$ and Pearson correlation=0.37 for control subjects).

**Table 16- Albumin, creatinine and their ratio in early morning urine sample in diabetic and control subjects**

| | diabetic n=29 | control n=28 |
|---|---|---|
| albumin (mg/l) | | |
| mean±SD | 9.29±8.29 | 6.41±3.92 |
| 95%CI | 6.07-12.37 | 4.89-7.94 |
| creatinine (μmol/l) | | |
| mean±SD | 12629.14±5802.86 | 15271.11±5985.61 |
| 95%CI | 10421.86-14836.41 | 12950.15-17592.07 |
| Albumin (mg/l)/creatinine (mmol/l) | | |
| mean±SD | 0.61±2.01* | 0.39±1.38 |
| 95%CI | 0.47-0.81 | 0.34-0.44 |

* $p<0.005$.

In diabetic subjects regression analysis was performed to examine the importance of different variables on the logarithmically transformed albumin/creatinine ratio calculated from spot and early morning urine samples. No significant effect was found when the analysis was performed with duration of diabetes, dose of insulin and level of HbA1c% when the model was adjusted for age and sex.

The regression of these ratios on anthropometric measurements adjusted for age and sex indicated that with both ratios, WHR was the most important independent predictor but its positive effect was not significant in these models.

Similar models with systolic and diastolic blood pressure as independent variables and adjusted for age and sex, indicated that diastolic blood pressure and systolic blood pressure entered respectively into models constructed with spot and early morning urine samples, but their effects were not significant in these models.

HDL cholesterol entered into a model with both of these ratios when the analysis was adjusted for age and sex, and blood lipids and lipoproteins were used as independent variables. Its negative effect was significant when the analysis was performed with the ratio on early morning urine sample (table 17).

**Table 17- Results from multiple regression analysis of log of albumin/creatinine ratio of early morning urine sample on blood lipids and lipoproteins in diabetic subjects**

| | model 1 | model 2 |
|---|---|---|
| Intercept | 0.26 | 0.70 |
| sex | -0.09 | -0.14* |
| age | -0.01 | -0.01 |
| total cholesterol | - | (•) |
| HDL cholesterol | - | -0.27* |
| LDL cholesterol | - | (•) |
| triglyceride | - | (•) |
| N | 29 | 28 |
| $R^2$ | 0.15 | 0.27 |
| Adjusted $R^2$ | 0.08 | 0.18 |
| p value of whole model | 0.12 | <0.05 |

The figures in front of each variable are the estimates.- not available in this model. (•) available but not entered into the model. * $p<0.05$.

### 4.5-Dietary Intakes

Dietary intake of diabetic and control subjects were determined by diet history and the assistance of food models and cup and spoon measures. Both food pattern questionnaire and a food checklist were used as a double check for the assessment of their dietary intakes. The results are shown in tables 18 to 25. These are the results of dietary intake of 54 diabetic subjects (17 female and 37 male) and 47 control subjects (17 female and 30 male).

The macronutrients and alcohol intake of diabetic and control subjects are shown in table 18. The amount of starch in the diet of diabetic subjects was significantly higher than that in the diet of control subjects ($p=0.01$ and $p<0.01$ for females and males respectively). Also grams of fibre intake by male diabetic subjects was significantly more than that consumed by male control subjects ($p<0.01$). The intake of other nutrients and alcohol by diabetic subjects were similar to their intake by control subjects except for protein and carbohydrate intake which females with diabetes consumed more but did not differ significantly.

As it was expected in both groups the means of protein, total fat and carbohydrate intakes in males were significantly higher than in females (for all the nutrients in two groups $p<0.01$). But when the amount of protein intake in relation to body weight was compared within diabetic subjects, males had consumed significantly more protein than females ($p=02$). This difference was not significant when females and males were compared within control group. No significant difference was found between female and male diabetic subjects regarding sugar intake, but within control males consumed more sugar than females ($p<0.05$).

**Table 18- Macronutrients and alcohol intake of diabetic and control subjects**

| | Diabetic (n=54) | | Control (n=47) | |
|---|---|---|---|---|
| | Mean±SD | 95%CI | Mean±SD | 95%CI |
| Protein (g) | | | | |
| female | 100.5±25.3 | 87.4-113.5 | 89.7±17.8 | 80.6-98.9 |
| male | 143.2±35.0 | 131.6-154.9 | 138.6±56.3 | 117.5-159.6 |
| Pro (g)/bwt (kg)[a] | | | | |
| female | 1.5±0.5 | 1.2-1.7 | 1.4±0.3 | 1.3-1.6 |
| male | 1.8±0.5 | 1.7-2.0 | 1.8±1.0 | 1.4-2.2 |
| Total fat (g) | | | | |
| female | 56.7±27.3 | 42.7-70.8 | 63.0±30.35 | 47.4-78.6 |
| male | 98.3±34.2 | 87.0-109.7 | 106.2±28.1 | 95.7-116.7 |
| Carbohydrate(g) | | | | |
| female | 245.6±56.5 | 216.6-274.7 | 213.5±48.5 | 188.5-238.4 |
| male | 328.7±91.1 | 298.3-359.1 | 298.3±78.0 | 269.1-327.4 |
| Starch (g) | | | | |
| female | 131.9±34.7** | 114.1-149.8 | 101.5±41.8 | 80.0-123.0 |
| male | 203.4±63.9** | 182.0-224.6 | 152.8±47.4 | 135.1-170.6 |
| Sugar (g) | | | | |
| female | 111.2±32.7 | 94.4-128.0 | 110.5±28.6 | 95.8-125.2 |
| male | 122.5±50.6 | 105.6-139.3 | 142.0±60.3 | 119.5-164.5 |
| Fibre(g) | | | | |
| female | 30.7±7.6 | 26.8-34.6 | 30.3±10.9 | 24.7-35.9 |
| male | 41.6±11.1** | 37.9-45.3 | 32.4±10.2 | 28.5-36.2 |
| Alcohol(g) | | | | |
| female | 3.3±6.7 | 0.1-6.7 | 7.6±15.4 | 0.3-15.5 |
| male | 8.3±15.9 | 3.2-13.8 | 9.4±12.5 | 4.8-14.1 |

[a]Protein (g)/Body weight (kg). ** $p<0.01$.

Table 19 summarizes the results of dietary intake regarding energy intake and contribution of macronutrients and alcohol in the usual daily diets of the two

groups. It shows that none of the same-sex between group comparisons was statistically significantly different. For instance total fat (energy%) intake was lower in male diabetic subjects than its intake in male controls (p=0.08).

Among people with diabetes, energy intake and the contributions of fat and alcohol in the daily energy intake of females were lower than in males. Percent energy from carbohydrate in the diet of female subjects with diabetes was higher than this ratio in the diets of male subjects with diabetes and protein (energy%) was similar for two sexes. These differences, except for energy intake (p<0.0001) were not statistically significant. Similar results were found when control males and control females were compared.

**Table 19- Energy intake and contribution of macronutrients and alcohol in daily diet of diabetic and control subjects**

| | Diabetic (n=54) | | Control (n=47) | |
|---|---|---|---|---|
| | mean±SD | 95% CI | mean±SD | 95% CI |
| Energy (kJ) | | | | |
| female | 7975.7±2033.5 | 6930.2-9021.3 | 7644.1±1758.4 | 6739.9-8548.2 |
| male | 11837.5±2549.9 | 10987.3-12687.6 | 11424.1±1986.7 | 10682.2-12165.9 |
| Energy (kcal) | | | | |
| female | 1904.6±485.5 | 1654.9-2154.2 | 1826.3±420.1 | 1610.4-2042.3 |
| male | 2827.0±609.1 | 2624.8-3030.9 | 2731.1±473.7 | 2554.2-2907.9 |
| Fat (energy%) | | | | |
| female | 28.3±10.0 | 23.1-33.4 | 30.1±9.5 | 25.3-35.1 |
| male | 31.2±6.7 | 28.9-33.4 | 34.1±7.1 | 31.5-36.8 |
| CHO [a] (energy%) | | | | |
| female | 47.7±13.6 | 40.7-54.7 | 46.2±8.0 | 42.0-50.3 |
| male | 45.2±7.9 | 42.6-47.9 | 42.4±6.7 | 39.9-44.9 |
| Protein (energy%) | | | | |
| female | 21.5±4.4 | 19.2-23.8 | 20.6±4.9 | 19.1-22.2 |
| male | 21.1±4.1 | 19.8-22.6 | 20.7±5.8 | 18.5-22.9 |
| Alcohol (energy%) | | | | |
| female | 1.6±3.9 | 0.3-3.7 | 2.7±5.6 | 0.1-5.6 |
| male | 2.3±4.1 | 0.7-3.6 | 2.5±3.7 | 1.2-3.9 |

[a] Carbohydrate.

In addition to diet history data and food pattern questionnaire, the quantity and frequency of alcoholic drink consumption by diabetic and control subjects were assessed with 2 questions (Appendix 17). Table 20 shows the results. Fifty-four diabetic subjects and 46 control subjects answered the question on the frequency of alcoholic drinks while 47 diabetic subjects and 41 control subjects answered to the second question relating to the number of drinks consumed.

No significant differences were found when the number of patients and controls were compared regarding alcohol intake frequency except for those who drink 3 or 4 days and/or 5 or 6 days a week. However, few people were classified in these two categories. In addition the number of drinks were compared between the two groups. Only the number of control subjects who used to drink 9 to 12 drinks was significantly higher than the comparable number of patients. However, the numbers were too small to draw any conclusion.

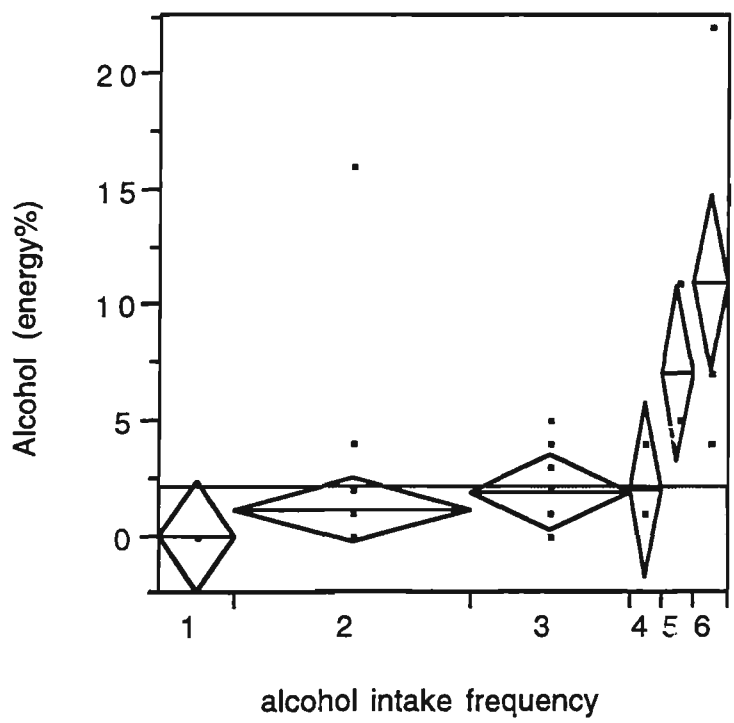
Among diabetic subjects it was found that HbA1c% was the lowest for those who drink less (1-2 drinks each time) and it was the highest for a patient who used to drink more than 20 drinks 1-2 days/week. But no other conclusion could be drawn in relation to different number of drinks in other groups. The trend for the whole population was not significant ($p=0.21$).

As it was mentioned before alcohol intake was also calculated from the diet history data. It was found that there was a significant correlation between these two sets of information ($p<0.0001$, Pearson correlation=0.59) (figure 3).

**Table 20- Alcohol intake of diabetic and control subjects**

| | diabetic n (%) | control n (%) | p value |
|---|---|---|---|
| **Alcohol intake frequency** | | | 0.23 |
| I don't drink alcohol | 7 (12.9) | 5 (10.9) | |
| Less than once a week | 22 (40.7) | 14 (30.4) | |
| On 1 or 2 days a week | 16 (29.6) | 16 (34.8) | |
| On 3 or 4 days a week | 3 (5.6) | 8 (17.4) | |
| On 5 or 6 days a week | 3 (5.6) | 0 (0.0) | |
| Every day | 3 (5.6) | 3 (6.5) | |
| **How many drinks** | | | 0.44 |
| 1 or 2 drinks | 17 (36.2) | 16 (39.1) | |
| 3 or 4 drinks | 19 (40.4) | 11 (26.8) | |
| 5 to 8 drinks | 8 (17.0) | 8 (19.5) | |
| 9 to 12 drinks | 2 (4.3) | 6 (14.6) | |
| 13 to 20 drinks | 0 (0.0) | 0 (0.0) | |
| more than 20 drinks | 1 (2.1) | 0 (0.0) | |

**Figure 3- Correlation of alcohol intake frequency and percentage of energy from alcohol***




* The horizontal line shows total response sample mean. In each diamond the width represents the group sample size and the top and bottom points of the means diamond show the upper and lower 95% confidence points for each group.

The intakes of polyunsaturated, monounsaturated and saturated fatty acids, their ratios and cholesterol intake of diabetic and control subjects are presented in table 21. It is shown that the ratio of polyunsaturated to saturated fatty acids were significantly higher among diabetic males than control males.

On the other hand control males consumed more saturated fatty acids in their diets than diabetic males did. No significant differences were found when females from the two groups were compared. The contributions of fatty acids in daily energy intake of female and male diabetic subjects were not significantly different. Females with diabetes had a lower intake of cholesterol in their daily diet than males with diabetes ($p<0.01$).

**Table 21- Fatty acids and cholesterol intake of diabetic and control subjects**

| | Diabetic (n=54) | | Control (n=47) | |
|---|---|---|---|---|
| | Mean±SD | 95% CI | Mean±SD | 95% CI |
| PUFA [a] (g) | | | | |
| female | 9.1±7.2 | 5.3-12.7 | 9.8±8.4 | 5.5-14.1 |
| male | 17.2±11.2 | 13.5-20.9 | 13.5±6.6 | 11.1-16.0 |
| PUFA (Energy%) | | | | |
| female | 5.6±3.8 | 3.6-7.6 | 5.3±2.9 | 3.8-6.7 |
| male | 6.1±2.7 | 5.2-7.0 | 4.9±2.2 | 4.1-5.8 |
| MUFA[b] (g) | | | | |
| female | 18.5±10.7 | 12.9-24.0 | 20.3±9.5 | 15.4-25.2 |
| male | 32.8±12.9 | 28.5-37.1 | 37.0±13.0 | 32.2-41.9 |
| MUFA (Energy%) | | | | |
| female | 11.0±5.6 | 8.1-13.9 | 11.1±3.8 | 9.1-13.1 |
| male | 11.9±3.0 | 10.9-12.9 | 13.4±3.6≈ | 12.1-14.7 |
| SAFA[c] (g) | | | | |
| female | 21.5±9.6 | 16.5-26.4 | 25.6±13.3 | 18.7-32.4 |
| male | 36.0±13.1 | 31.7-40.4 | 43.6±13.1* | 38.7-48.5 |
| SAFA (Energy%) | | | | |
| female | 12.8±5.3 | 10.1-15.5 | 13.8±5.1 | 11.2-16.5 |
| male | 13.2±3.5 | 12.0-14.3 | 15.9±3.7** | 14.5-17.3 |
| P:S[d] | | | | |
| female | 0.44±0.19 | 0.34-0.54 | 0.43±0.23 | 0.31-0.54 |
| male | 0.50±0.28** | 0.41-0.60 | 0.32±0.19 | 0.26-0.40 |
| M:S[e] | | | | |
| female | 0.85±0.16 | 0.76-0.93 | 0.83±0.21 | 0.73-0.94 |
| male | 0.93±0.22 | 0.86-1.00 | 0.86±0.23 | 0.78-0.95 |
| Cholesterol (mg) | | | | |
| female | 178.2±65.9 | 144.3-212.1 | 200.1±92.4 | 152.4-247.7 |
| male | 299.3±137.4 | 253.4-345.1 | 351.0±164.7 | 289.5-412.5 |

[a]polyunsaturated fatty acids. [b]monounsaturated fatty acids. [c]saturated fatty acids. [d]polyunsaturated to saturated fatty acids ratio. [e]monounsaturated to saturated fatty acids ratio. ** $p<0.01$. * $p<0.05$.

Vitamin intakes including retinol equivalent (A), ascorbic acid (C), thiamin (B1), riboflavin (B2) and niacin (PP) were determined and the results are presented in table 22. There was no significant difference of any one of the vitamin intakes when similar sexes from two groups compared except for niacin intake which was significantly higher for male diabetic subjects ($p<0.01$).

When females and males from the same group were compared, significant differences found in vitamin B1, B2 and pp intakes. Males consumed significantly more of these vitamins in both groups than females ($p=0.03$ for B1, $p=0.03$ for B2

and $p<0.01$ for PP in diabetic subjects and $p=0.05$ for B1, $p=0.05$ for B2 and $p<0.01$ for PP in control subjects).

**Table 22- Vitamin intakes of diabetic and control subjects**

| | Diabetic (n=54) | | Control (n=47) | |
|---|---|---|---|---|
| | Mean±SD | 95% CI | Mean±SD | 95% CI |
| Retinol Eq(μg) | | | | |
| female | 1671.2±773.3 | 1273.6-2068.8 | 1978.9±1462.6 | 1227.0-2730.9 |
| male | 1788.7±1043.7 | 1440.7-2136.7 | 1861.8±802.9 | 1562.0-2161.6 |
| Vitamin C(mg) | | | | |
| female | 171.9±128.9 | 105.7-238.3 | 255.5±267.2 | 118.1-392.8 |
| male | 226.7±138.4 | 180.6-272.9 | 258.1±359.7 | 123.8-392.5 |
| Thiamin(mg) | | | | |
| female | 1.8±0.7 | 1.4-2.2 | 1.6±0.8 | 1.2-2.0 |
| male | 2.5±1.3 | 2.1-3.0 | 2.1±0.7 | 1.8-2.3 |
| Riboflavin(mg) | | | | |
| female | 2.7±1.1 | 2.1-3.2 | 2.4±1.3 | 1.7-3.1 |
| male | 3.6±1.6 | 3.1-4.2 | 3.1±1.1 | 2.7-3.5 |
| Niacin Eq (mg) | | | | |
| female | 41.6±9.0 | 36.9-46.2 | 34.6±14.3 | 27.2-42.0 |
| male | 59.9±17.3** | 54.1-65.7 | 47.8±14.8 | 42.3-53.3 |

** $p<0.01$.

Sodium, potassium, calcium, iron and zinc intake of diabetic and control subjects are shown and compared in table 23. The intakes of potassium, calcium, iron and zinc in females and males from two groups did not show any significant differences. But the sodium intake of male diabetic subjects was significantly higher than that of control males ($p<0.01$). This difference was not seen when females were compared. Within the diabetic group the sodium intake of males was significantly higher than that of females ($p<0.01$). It should be mentioned that sodium intake from added salt was not included in these calculations.

There was no significant difference in the potassium and calcium intakes of female and male diabetic subjects ($p=0.07$ for potassium and $p=0.78$ for calcium). Iron and zinc intakes were significantly higher in male diabetic subjects than in female diabetic subjects ($p=0.00$ for iron and zinc).

Control males had significantly higher intakes of sodium, potassium, calcium, iron, and zinc than control females (P=0.03, p=0.02, p=0.03, p=0.01 and p=0.00 for sodium, potassium, calcium, iron and zinc respectively).

**Table 23- Mineral intakes of diabetic and control subjects**

| | Diabetic (n=54) | | Control (n=47) | |
|---|---|---|---|---|
| | mean±SD | 95% CI | mean±SD | 95% CI |
| Sodium (mg) | | | | |
| female | 2447.1±1941.7 | 1448.7-3445.4 | 2234.5±1019.6 | 1710.2-2758.7 |
| male | 3935.2±1245.8** | 3519.8-4350.5 | 2993.4±1138.6 | 2568.3-3418.6 |
| Potassium(mg) | | | | |
| female | 4294.1±1061.4 | 3748.3-4839.8 | 3640.9±965.9 | 3144.3-4137.6 |
| male | 4921.1±1222.9 | 4513.4-5328.9 | 4524.5±1286.1 | 4044.3-5004.7 |
| Calcium(mg) | | | | |
| female | 1157.8±655.3 | 820.9-1494.8 | 924.7±298.4 | 771.2-1078.1 |
| male | 1197.3±396.9 | 1064.9-1329.6 | 1196.1±440.2 | 1031.7-1360.4 |
| Iron(mg) | | | | |
| female | 13.4±3.0 | 11.8-14.9 | 15.1±4.8 | 12.6-17.6 |
| male | 21.3±6.0 | 19.2-23.3 | 19.8±6.7 | 17.3-22.3 |
| Zinc(mg) | | | | |
| female | 11.8±4.0 | 9.7-13.9 | 11.4±3.7 | 9.4-13.3 |
| male | 18.7±6.1 | 16.6-20.7 | 17.1±6.6 | 14.7-19.5 |

** p<0.01.

Regression analysis was performed to examine the effects of independent variables on dietary intakes. No association was found between carbohydrate (energy%), fat (energy%) or protein (energy%) and age, sex, education, and income level in diabetic and control subjects. Duration of diabetes, dose of insulin, number of injections, HbA1c% did not enter into the models with the above mentioned dependent variables in diabetic subjects when the analysis was adjusted for age and sex.

As part of Food Pattern Questionnaire (Q13) (Appendix 13), meal time and place of consuming the meals were asked from diabetic and control subjects. The usual time of meals and the number of diabetic and control subjects who did not have the meals are indicated in table 24.

Six patients had an additional snack, one at 2:00 pm, three at 8:30 pm and two at 24:00. Three controls had their additional snack at 5:00 pm, one at 1:00 pm, one at 8:00 pm and one at 11:00 pm.

The majority of diabetic subjects had their meals either at home or take it from home while more controls would buy at least some of their meals from takeaway outlets. As it is shown controls skip their meals especially their breakfasts and snacks more frequently than patients.

**Table 24- Usual time and snack pattern in diabetic and control subjects**

| | Usual time of meal | | Do not eat | |
|---|---|---|---|---|
| | Diabetic n=55 | Control n=47 | Diabetic n (%) | Control n (%) |
| Breakfast | 7:00 | 7:45 | 0 (0) | 6 (13) |
| Morning snack | 10:15 | 10:15 | 1 (2) | 9 (19) |
| Lunch | 12:30 | 12:45 | 0 (0) | 2 ( 4) |
| Afternoon snack | 15:30 | 15:30 | 4 (7) | 6 (13) |
| Dinner | 18:15 | 17:00 | 0 (0) | 0 ( 0) |
| Supper | 21:15 | 20:45 | 2 (4) | 3 ( 6) |

### 4.6- Dietary Adherence

The contribution of macronutrients in the daily diets of diabetic and control subjects were compared to the Australian Guidelines. The numbers of individuals from the diabetic and control groups that met the Australian guidelines for the contribution of macronutrients in the daily diet are shown in table 26.

Significantly more subjects with diabetes reached the Australian guidelines for fat. However the number of controls who met the dietary guideline for protein was significantly higher than that for diabetic subjects. No significant difference was found regarding carbohydrate (energy%) between groups. Overall 4 individuals from the diabetic group (3 males and 1 female) could meet all the guidelines for the macronutrients (energy%) in their diets.

**Table 25- Diabetic and control subjects meeting the Australian dietary guidelines for diabetes**

| Macronutrient | Guidelines (energy%) | Diabetics n (% of group) | controls n (% of group) |
|---|---|---|---|
| Fat | <30 | 27 (50.0)* | 15 (31.9) |
| Carbohydrate | >50 | 18 (33.3) | 12 (25.5) |
| Protein | <20 | 20 (37.0) | 27 (57.4)* |

* $p<0.05$

The means of vitamin and mineral intakes of the subjects from the diabetic and control groups were greater than the Australian Recommended Dietary Intakes (RDI) (table 26). Although most of the subjects had adequate nutrient intakes, 7 female diabetics and 5 female controls had a mean intake of iron below the RDI for adults. Also the intake of zinc was lower than the RDI in 10 diabetic subjects and 10 controls, and 3 diabetic subjects and 5 controls did not meet the recommendations for calcium.

**Table 26- Comparison of micronutrient intakes of diabetic and control subjects with the Australian RDI**

| Nutrient | RDI | Diabetic* | Control* |
|---|---|---|---|
| Thiamin (mg) | 1.0 | 2.3 | 1.9 |
| Riboflavin (mg) | 1.5 | 3.3 | 2.8 |
| Niacin Eq (mg) | 16.3 | 54.1 | 43.0 |
| Retinol Eq (µg) | 750.0 | 1751.7 | 1904.2 |
| Vitamin C (mg) | 30.0 | 209.5 | 257.2 |
| Iron (mg) | 12.0 | 18.8 | 18.1 |
| Zinc (mg) | 12.0 | 16.5 | 15.2 |
| Calcium (mg) | 800.0 | 1184.9 | 1097.9 |

* Data are means of micronutrient intakes.

The above mentioned comparisons could provide some information about intake of each nutrient. In order to get the overall diet quality of diabetic and

control subjects their diets were assessed by two ways. Firstly the diets of diabetic and control subjects were evaluated on the basis of food group consumption and secondly their diets were scored by comparison with a number of the Australian and American dietary recommendations.

For this purpose each diet history collected from diabetic and control subjects was evaluated for the consumption of 5 basic food groups including, dairy, meat, fruit, vegetable and grain products. Additionally, the variety of food items within each food group was assessed. The results for diabetic and control subjects and their food patterns were summarized in table 27.

It can be seen that all of the patients consumed milk in their usual daily diet. In addition to milk, cheese and to the lesser extent yogurt, was used as well. The meat group included both animal and plant protein sources (eg, beef, pork, lamb, poultry, fish, shellfish, dried beans and peas, and nuts and seeds). One patient did not have red meat or chicken in her diet but did eat fish and marine foods. Others had a variety in their consumption of red and white meat. With regard to the consumption of legumes (dried peas and beans) only one individual in diabetic group had reported its use in her usual diet. When food pattern questionnaires were checked 29 patients (53.7%) never, 13 patients (24.1%) have them 1 to 3 times per month and the rest (22.2%) had legumes 1 to 3 times a week.

Two controls did not have red or white meat in their diet but they used egg as an alternate. Among them one mentioned that he consumes legumes. But when their food pattern questionnaire were checked, it was found that 16 (33.3%) of them never used to have legumes in their diet. Also 26 controls (55.3%) 1 to 3 times a month and 6 controls (12.8%) have them 1 to 3 times a week in their diets.

Among patients 8 (14.8%) people did not consume fruit in their usual daily diet or they did not have it in an adequate amount. An addition of 3 patients used either dried fruit or fruit juice instead of fresh fruit. Diabetic subjects consumed significantly more other kinds of fruit than controls ($p=0.03$).

All the diabetic subjects had at least two kinds of vegetable in addition to potato in their usual diet in either fresh or cooked form. The majority had mashed or boiled potato on a daily basis or 4 to 6 times per week. Breakfast cereals, including WeetBix and Cornflakes were consumed by 33 subjects. Bread, rice and pasta provided the rest of cereal group alternately.

Fourteen (29.1%) controls did not have either fruit in their diets or they had it in less than recommended amount. The consumption of vegetable group by controls was similar to diabetics. They had potato and other vegetable in their usual daily diet. Among controls Just Right, Cocopops, Weetbix and Sultanabran were used as a breakfast cereal. One person consumed Allbran in his breakfast.

**Table 27- Pattern of basic food groups consumption***

| | diabetic n=54 | control n=47 | p value |
|---|---|---|---|
| 1.Milk | 48 (88.9) | 46 (97.9) | 0.52 |
| 2.Meat | | | |
| meats | 53 (98.1) | 45 (95.7) | 0.86 |
| nuts | 8 (14.8) | 4 (8.5) | 0.18 |
| legumes | 1 (1.8) | 1(2.1) | 0.88 |
| 3.Fruit | | | |
| Citrus fruits | 27 (50.0) | 25 (53.2) | 0.75 |
| others | 46 (85.2) | 28 (59.6) | 0.03 |
| 4.Vegetables | | | |
| potato | 37 (68.5) | 28 (59.6) | 0.43 |
| others | 53 (98.1) | 47 (100) | 0.89 |
| 5.Bread and cereals | 54 (100) | 47 (100) | 1.00 |

*values (%) are the number of subjects who had consumed at least 2 exchanges from milk, meat, fruit and vegetable groups and 4 exchanges from bread and cereal group.

Secondly the goals for the dietary intake as recommended by the Australian and American dietetic Associations were used as cutoffs for scaling. Individuals who met a goal were arbitrarily given a score of 3 in recommendations 1-4 and 9, and score of 2 for goals 5-8. The total maximum score is 23 and the minimum is 9. The number and percent of subjects meeting the diet goals are illustrated in table 28.

There was no significant difference between diabetic and control subjects regarding the scores for the protein (energy%). There were significantly more diabetics who had consumed less than 30% of energy from total fat. The number of diabetic subjects who had 45-50% carbohydrate was significantly more than control subjects.

The scores for fatty acids and cholesterol intake were not significantly different between the groups except for MUFA (energy%). Significantly more subjects in control group had greater than 10% of their energy intake as monounsaturated fatty acids.

The number of diabetic subjects who had consumed more than 30 grams of dietary fibre in their diet was greater than control subjects. On the other hand the number of controls whose sodium intake was within recommendation was significantly higher than the number of diabetic subjects.

The mean±SD of overall score for diabetic subjects was 15.74±1.81 and for controls was 15.29±2.36 which were not significantly different. In both groups females had higher dietary score than males, this difference was significant among diabetic subjects ($p=0.03$).

**Table 28- Distribution of dietary score on the basis of recommendations in diabetic and control subjects**

| Recommendations | score | intake | diabetic | control | p value |
|---|---|---|---|---|---|
| 1.Limit protein intake to 10-15 (energy%) | 3 | 10-15% | 4 (7.4%) | 5 (10.6%) | 0.45 |
| | 2 | 15-20% | 17 (31.5%) | 20(42.5%) | |
| | 1 | >20% | 33 (61.1%) | 22(46.9%) | |
| 2.Reduce total fat intake to 30% or less of energy | 3 | ≤30% | 27 (50.0%) | 15 (31.9%) | 0.18 |
| | 2 | 31-35% | 13 (24.1%) | 15 (31.9%) | |
| | 1 | >36% | 14 (25.9%) | 17 (36.2%) | |
| 3.Increase CHO[a] intake to 55% of energy | 3 | 50-55% | 18 (33.3%) | 12 (25.5%) | <0.01 |
| | 2 | 45-50% | 20 (37.0%) | 6 (12.8%) | |
| | 1 | ≤44% | 16 (29.7%) | 29 (61.7%) | |
| 4.Limit PUFA[b] intake to 6-10% of energy | 3 | 6-10% | 22 (40.7%) | 14 (29.8%) | 0.27 |
| | 2 | <6% | 30 (55.6%) | 36 (68.1%) | |
| | 1 | >10% | 2 (3.7%) | 1 (2.1%) | |
| 5.Increase MUFA[c] intake to more than 10% of energy | 2 | ≥10% | 33 (61.1%) | 36 (76.6%) | 0.09 |
| | 1 | <10% | 21 (38.9%) | 11 (23.4%) | |
| 6.Reduce SAFA[d] intake to less than 10% of energy | 2 | ≤10% | 14 (25.9%) | 8 (17.0%) | 0.28 |
| | 1 | >10% | 40 (74.1%) | 39 (83.0%) | |
| 7.Reduce cholesterol intake to 300 mg daily | 2 | ≤300 | 32 (59.2%) | 26 (55.3%) | 0.69 |
| | 1 | >300 | 22 (40.8%) | 21 (44.7%) | |
| 8.Increase fibre intake to more than 30 grams | 2 | ≥30 | 41 (75.9%) | 26 (55.3%) | 0.03 |
| | 1 | <30 | 13 (24.1%) | 21 (44.7%) | |
| 9.Limit sodium intake between 1150-2300 mg daily | 3 | 1150-2300 | 11 (20.4%) | 18 (38.3%) | 0.10 |
| | 2 | <1150 | 1 (1.8%) | 0 (0%) | |
| | 1 | >2300 | 42 (77.8%) | 29 (61.7%) | |

[a] Carbohydrate. [b] Polyunsaturated fatty acid. [c] Monounsaturated fatty acid.
[d] Saturated fatty acid.

Stepwise regression analysis was used to relate independent variables including income and education level to the diet score while the analysis allowed for interaction between two groups and adjusted for age and sex. It was found that

education level (tertiary schooling finished) had significant positive effect in the model (table 29).

In diabetic subjects the association between HbA1c% and the grams of alcohol and diet score was examined by regression analysis which was adjusted for age, sex and dose of insulin. The result showed that the grams of alcohol and the diet score entered into the model with negative but insignificant effects ($R^2$=0.19 and $p<0.05$ for the whole model). No associations were found when regression analysis was done with diet score on biochemical findings and blood pressure adjusted for age and sex in diabetic subjects.

A stepwise regression analysis was performed with the dependent variables being HbA1c% and the independent variables being units of insulin per body weight (kg) and diet score. Diet score had negative and insignificant effect ($p<0.1$) in the model, which was adjusted for age and sex (table 30).

**Table 29- Results from multiple regression analysis of diet score on income and education level in diabetic and control subjects**

| | model 1 | model 2 |
|---|---|---|
| Intercept | 16.07** | 16.57** |
| group | -0.27 | -0.40 |
| sex | -0.54* | -0.47* |
| age | -0.01 | -0.03 |
| sexxgroup | - | (•) |
| agexgroup | - | (•) |
| Education (1&2-3) | - | -0.62** |
| Education (1-2) | - | (•) |
| Education (1&2-3)xgroup | - | (•) |
| Education (1-2)xgroup | - | (•) |
| Income (1&2-3) | - | (•) |
| Income (1-2) | - | (•) |
| Income (1&2-3)xgroup | - | (•) |
| Income (1-2)xgroup | - | (•) |
| N | 101 | 101 |
| $R^2$ | 0.07 | 0.15 |
| Adjusted $R^2$ | 0.04 | 0.11 |
| p value of whole model | <0.1 | <0.01 |

The figures in front of each variable are the estimates.- not available in this model. (•) available but not entered into the model. ** p <0.01. * p<0.05.

**Table 30- Results from multiple regression analysis of HbA1c% on diet score and units of insulin per body weight (kg)**

| | model 1 | model 2 |
|---|---|---|
| Intercept | 11.68** | 16.02** |
| sex | 0.01 | -0.14 |
| age | -0.09* | -0.10* |
| insulin (units)/body weight (kg) | - | (•) |
| diet score | - | -0.26 |
| N | 51 | 51 |
| $R^2$ | 0.09 | 0.14 |
| Adjusted $R^2$ | 0.54 | 0.09 |
| p value of whole model | <0.1 | <0.1 |

The figures in front of each variable are the estimates.- not available in this model. (•) available but not entered into the model. ** $p < 0.01$. * $p < 0.05$.

In addition to above data that were derived from diet history, food pattern and food preparation questionnaires, dietary adherence of diabetic subjects was determined by a special questionnaire (Appendix 15). The responses of 55 diabetic subjects (18 females and 37 males) to three questions related to dietary adherence are summarized in table 31. As is indicated in the table, 27% of subjects answered that they always follow their carbohydrate portion plan. The majority of them (46%) usually followed the carbohydrate portion plan.

About 8% of them either sometimes or not very often used to follow the plan, 7% did not follow the carbohydrate portion plan, 2% did not know about the diet plan and the same ratio did not answer to this question. These results reveal that the number of the patients who always followed carbohydrate portion plan is significantly higher than those who do not do so ($p<0.0001$)

In response to why they did not follow the plan, 7% said that it did not help them and 16% were tired of following the carbohydrate portion plan. Work was too hectic for 23% of them to follow the portion plan. For 14% of patients family life made the following of carbohydrate portion plan difficult and 2% of the respondents said that family/friend were not supporting them in following the carbohydrate portion plan. About 23% craved foods they should not eat and 10% gave other reasons for not following the carbohydrate portion plan. Two of them

gave more than one reason to why they didn't follow this plan. About 2%, 15%, 30%, 31% and 22% generally found following carbohydrate portion plan, very difficult, moderately difficult, neither difficult or easy, moderately easy and very easy respectively.

For the third question, a significant difference ($p<0.0001$) was found when the number of those who find carbohydrate portion plan very difficult was compared with the number of patients who find it very easy. Most of them believed that this plan is neither difficult nor easy or it was moderately easy.

**Table 31- Dietary adherence of patients with type 1 diabetes**

| | female n (%) | male n (%) | Total n (%) |
|---|---|---|---|
| 1.Follow carbohydrate portion plan | | | |
| always | 6(33) | 9 (24) | 15(27) |
| usually | 7(39) | 17(46) | 24(44) |
| sometimes | 2(11) | 3( 8) | 5( 9) |
| not very often | 2(11) | 3( 8) | 5( 9) |
| no | 0( 0) | 4(11) | 4( 7) |
| don't know | 0( 0) | 1( 3) | 1( 2) |
| no answer | 1( 6) | 0( 0) | 1( 2) |
| 2.Why didn't follow carbohydrate portion? | | | |
| It didn't help | 1( 5) | 3( 8) | 4( 7) |
| tired of following | 3(17) | 6(16) | 9(16) |
| work is too hectic | 5(28) | 8(21) | 13(23) |
| family life makes it difficult | 3(17) | 5(13) | 8(14) |
| family/friend are not supportive | 0( 0) | 1( 3) | 1( 2) |
| crave food shouldn't eat | 7(39) | 6(16) | 13(23) |
| other | 3(17) | 7(18) | 10(18) |
| 3.Generally find it | | | |
| very difficult | 0( 0) | 1( 3) | 1( 2) |
| moderately difficult | 2(13) | 6(16) | 8(15) |
| neither difficult or easy | 5(31) | 11(29) | 16(30) |
| moderately easy | 5(31) | 12(31) | 17(31) |
| very easy | 4(25) | 8(21) | 12(22) |

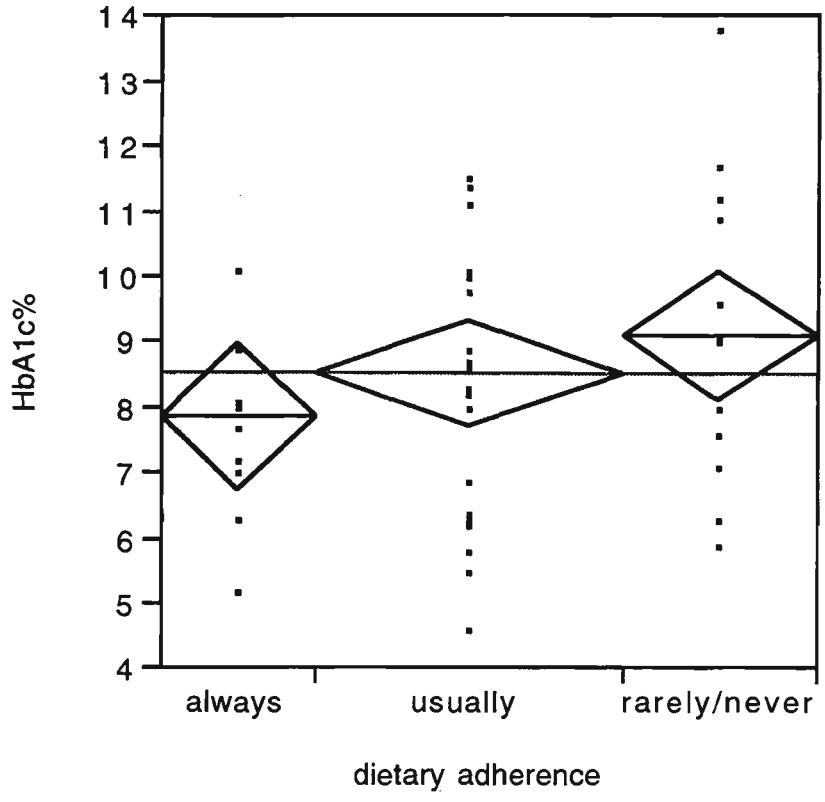
To compare the glycemic control of patients with their dietary adherence, the HbA1c% of each group in question 1 was compared and shown in table 32. Those who always followed the carbohydrate portion plan had lower HbA1c%. The patient who did not know about carbohydrate portion plan had the highest HbA1c%. No significant difference was found between first 2 groups.

Because the number of patients with type 1 diabetes in the last four groups in table 32 was small, the numbers were grouped as patients who rarely or never followed carbohydrate portion plan. As shown in figure 4, there was an increasing trend in HbA1c%, but this was not significant (p=0.26). A stepwise regression analysis, adjusted for age and sex demonstrated that dietary adherence and diet score had negative effects in the model with HbA1c% but their effects were not significant ($R^2$=0.18 and p<0.05 for the whole model).

**Table 32- HbA1c% of different groups of diabetic subjects in regard to their dietary adherence**

| | HbA1c% | | |
|---|---|---|---|
| | mean±SD | 95% CI | n |
| Follow carbohydrate portion plan | | | |
| always | 7.85±1.32 | 7.02-8.69 | 12 |
| usually | 8.53±2.01 | 7.69-9.39 | 24 |
| sometimes | 9.86±2.83 | 6.33-13.38 | 5 |
| not very often | 8.43±2.16 | 5.65-11.02 | 5 |
| no | 8.57±1.80 | 5.70-11.44 | 4 |
| don't know | 11.70 | - | 1 |

**Figure 4- Dietary adherence by HbA1c% in diabetic subjects***




* The horizontal line shows total response sample mean. In each diamond the width represents the group sample size and the top and bottom points of the means diamond show the upper and lower 95% confidence points for each group.

## 4.7- Weight Control and Physical Exercise

The diabetic and control subjects were asked if they currently intended to reduce, maintain or increase their weights and indicate their ideal weight (Appendix 16). Forty-seven diabetic subjects and 37 controls completely answered to these questions. The results of this survey are presented in table 33.

**Table 33- Weight control and ideal body weight of diabetic and control subjects**

| | Diabetic | Control |
|---|---|---|
| reduce weight | 15 (32) | 23 (62) |
| maintain weight | 26 (55) | 11 (30) |
| increase weight | 6 (13) | 3 ( 8) |
| ideal weight* | 73.13±12.51 | 70.38±12.66 |

The figures are the numbers (%) of subjects who had answered "yes" to the questions. *Values are mean±SD.

The diabetic and control subjects responses to the weight maintenance in relation to their BMI are presented in tables 34 and 35 respectively. These two tables show that in both groups those who intended to reduce their weight were overweight and needed to do so. Again the average BMI of those who gave a negative answer to maintaining their body weight, was more than 25. Six patients and 3 controls intended to increase their body weight while their BMIs were not below the normal range.

**Table 34- BMI of patients by their attitude toward weight maintenance**

| | Negative answers | | Positive answers | |
|---|---|---|---|---|
| | n (%) | BMI | n (%) | BMI |
| reduce | 37 (71.2) | 24.66±2.40 | 15 (28.8) | 28.32±5.27 |
| maintain | 27 (51.9) | 26.61±0.89 | 25 (48.1) | 24.66±2.34 |
| increase | 46 (88.4) | 26.51±4.70 | 6 (11.6) | 23.60±1.05 |

**Table 35- BMI of controls by their attitude toward weight maintenance**

| | Negative answers | | Positive answers | |
|---|---|---|---|---|
| | n (%) | BMI | n (%) | BMI |
| reduce | 24 (54.5) | 23.40±3.88 | 20 (45.5) | 27.95±3.51 |
| maintain | 33 (75) | 26.41±4.40 | 11 (25) | 22.65±2.64 |
| increase | 41 (93.2) | 25.48±4.01 | 3 (6.8) | 21.58±1.81 |

Diabetic and control subjects were asked about the degree and extent of their physical exercise and activity (Appendix 18). The results are shown in table 36.

**Table 36- Physical exercise in diabetic and control subjects**

| | Diabetic | Control |
|---|---|---|
| **Vigorous exercise** | | |
| Number(%) | 27(49.1) | 28(58.3) |
| Session/2wk | 4.2±2.7 | 5.4±4.1 |
| Time (hr/2wk) | 6.50±5.30 | 5:06±3.8 |
| **Less vigorous exercise** | | |
| Number(%) | 25(46.3) | 28(58.3) |
| Session/2wk | 3.8±3.1 | 5.8±3.4 |
| Time(hr/2wk) | 7.48 | 6.30 |
| **Walking** | | |
| Number(%) | 33(60) | 28(58.3) |
| Times of 20min.walk/2wk | 6.1±5.4 | 5.1±3.5 |
| **Vigorous activity** | | |
| Number(%) | 30(54.5) | 26(54.2) |
| Session/2wk | 4.6±3.2 | 3.7±3.1 |
| Time(hr/2wk) | 15.0 | 8.3 |

There were no significant differences between the number of patients with diabetes and controls when comparing within the levels of exercise. HbA1c% and logged transformed albumin: creatinine ratio on spot and early morning urine sample, of diabetic subjects who had performed different kinds of sport, were compared with the values found in those who were not engagedin those kinds of sports. HbA1c% was not different but the ratio on urine samples was higher among those performing a kind of physical activity, but they did not differ significantly.

Also diabetic subjects were asked if they increase their carbohydrate intake before exercise (Q6 in Food Pattern Questionnaire) (Appendix 13). Thirty-four of diabetic subjects used to increase their intake while 17 did not take more carbohydrate before performing an exercise.

### 4.8- Quality of Life

The quality of life of diabetic subjects was determined using two measures: Diabetes Quality of Life Measure (DQOL) (produced by the DCCT) and the Medical Outcome Study Health Survey 36-Item Short Form (SF-36) (Appendices 19-20). Controls completed only the SF-36 form and their results were compared with the results obtained from diabetic subjects.

The scores for the DQOL were examined separately by sex, and are presented in table 37. Total and individual scores show that all the subjects were generally satisfied and not worried about their diabetes. Diabetic males reported better scores than females except for the satisfaction subscale. No significant differences were found between males and females.

**Table 37- DQOL subscales by sex in diabetic subjects**

| | total n=51 | female n=16 | male n=35 |
|---|---|---|---|
| Total | 76.79±10.78 | 74.59±8.88 | 77.89±11.59 |
| Impact | 73.97±11.36 | 73.60±9.55 | 74.16±12.33 |
| Satisfaction | 73.91±15.48 | 76.47±11.57 | 72.66±17.07 |
| Worry:diabetes related | 74.22±17.06 | 70.31±17.07 | 74.25±19.81 |
| Worry:social/vocational | 81.38±15.25 | 77.32±15.89 | 81.92±16.64 |

It was found that the DQOL subscales all had significant positive correlation with each other. The strongest association was found between the diabetes worry and social worry scales. The correlations and their significant levels are shown in table 38.

The internal consistency of the DQOL subscales, were determined by Cronbach's $\alpha$. These values were compared with the internal consistency of the DQOL subscales which were obtained in the DCCT (table 39).

in this model when it was adjusted for age and sex (table 43). While duration of diabetes enetered into the model but did not have significant effect ($p<0.1$). Regression analysis of SF-36 subscales on diabetes history did not reveal any significant effect of these independent variables in the models.

**Table 43- Results from multiple regression analysis of diabetes worry subscale on diabetes history data**

| | model 1 | model 2 |
|---|---|---|
| Intercept | 72.77** | 118.08 |
| sex | 2.84 | 2.30 |
| age | 0.01 | -0.43 |
| Duration of diabetes | - | -1.45 |
| HbA1c% | - | -2.61* |
| N | 51 | 51 |
| $R^2$ | 0.02 | 0.18 |
| Adjusted $R^2$ | -0.01 | 0.10 |
| p value of whole model | 0.55 | <0.1 |

The figures in front of each variable are the estimates.- not available in this model.
** $p < 0.01$. * $p<0.05$.

The effects of marital status, BMI and WHR were examined in the regression models with SF-36 subscales in diabetic and control subjects. These analysis allowed for interaction between two groups and adjusted for age and sex. The results of these analyses are illustrated in table 44. This table shows that at least one of the anthropometric measurements had negative effect in each model.

Regression analysis was performed in order to examine the effects of dietary adherence on quality of life of patients. It was found that dietary adherence had negative effect on the DQOL and SF-36 subscales but when the models adjusted by age and sex these associations vanished.

**Table 44- Results from multiple regression analysis of SF-36 subscales on marital status and body measurements in diabetic and control subjects**

| | Physical functioning | | Bodily pain | | General health | | Vitality | | Mental health | |
|---|---|---|---|---|---|---|---|---|---|---|
| | model 1 | model 2 | model 1 | model 2 | model 1 | model 2 | model 1 | model 2 | model 1 | model 2 |
| Intercept | 100.39** | 165.87** | 69.86** | 118.44** | 58.89** | 134.68** | 53.00** | 76.37** | 66.42** | 115.24** |
| group | -0.28 | 2.04 | 1.26 | 4.81 | 3.19 | 28.35* | -0.76 | 25.97* | 1.47 | 2.16 |
| sex | -0.69 | 5.03* | 2.03 | 5.90** | -0.64 | 5.95* | 3.62 | 4.98* | 0.10 | 3.79 |
| age | -0.26 | -0.16 | 0.31 | 0.31 | 0.37 | 0.47 | 0.36 | 0.65 | 0.38 | 0.47 |
| sexxage | - | (•) | - | (•) | - | (•) | - | (•) | - | (•) |
| agexgroup | - | 0.52 | - | (•) | - | (•) | - | (•) | - | (•) |
| marital status | - | (•) | - | (•) | - | (•) | - | -4.32 | - | (•) |
| marital statusxgroup | - | (•) | - | (•) | - | (•) | - | (•) | - | (•) |
| BMI | - | -0.88* | - | -0.45 | - | -0.86 | - | -0.99* | - | -0.75 |
| BMIxgroup | - | -0.65 | - | -0.90* | - | 0.94* | - | -1.04* | - | (•) |
| WHR | - | -56.20* | - | -46.11 | - | -70.74* | - | (•) | - | -40.23 |
| WHRxgroup | - | (•) | - | 23.79 | - | (•) | - | (•) | - | (•) |
| N | 98 | 96 | 99 | 97 | 99 | 97 | 99 | 98 | 98 | 96 |
| $R^2$ | 0.01 | 0.22 | 0.04 | 0.16 | 0.03 | 0.20 | 0.05 | 0.15 | 0.02 | 0.11 |
| Adjusted $R^2$ | -0.02 | 0.16 | 0.00 | 0.09 | 0.00 | 0.15 | 0.02 | 0.09 | -0.00 | 0.07 |
| p whole model | 0.79 | <0.01 | 0.29 | <0.05 | 0.36 | <0.01 | 0.17 | <0.05 | 0.54 | <0.05 |

The figures in front of each variable are the estimates.- not available in this model. (•) available but not entered into the model.
** p <0.01. * p<0.05.

# Chapter 5

# Discussion

This project is one of the first studies which examines the glycemic control (assessed by HbA1c%) of adults with insulin dependent diabetes in relation to different variables in an urban Australian area. Most of the published studies in Australia are in relation to dietary intake and management of children and adolescents with type 1 diabetes.

The discussion will review glycemic control and the impact of a number of factors (anthropometric measurements, blood pressure, biochemical findings, socioeconomic status and physical exercise) on indices of control and on cardiovascular disease (CVD). The relationships between dietary intake, and adherence to a dietary prescription, glycemic control and quality of life will also be discussed.

## 5.1 - Representativeness of the Subjects and Their General Characteristics

Generalisability of the results of the study presented here depends on the representative nature of the sample. The selection of subjects in this study relied on records proved to be out of date. Counting the frequency of insulin dependent diabetes mellitus is critical for the understanding of the etiology and natural history of disease and for public health actions. Therefore effective monitoring is needed not only for the prevention of disease, but for the allocation of limited health-care resources (LaPorte et al. 1993). Standardized incidence registries permit the direct comparisons of type 1 diabetes in individual areas and globally (WHO Diamond Project Group 1990). For this purpose it is agreed that at least 90% of the cases in a population should be counted (Diabetes Epidemiology

Research International Group 1988). The cost of identifying the constant incidence and prevalence of the disease in this detail manner is quite high.

There are different methods for counting the cases of type 1 diabetes. In traditional methods multiple sources of ascertainments like hospital records, doctors' medical files, diabetes associations, schools and laboratories and so on are used to collect data on the number of patients with type 1 diabetes. Duplicate names are eliminated. This way of counting is incomplete and there is a problem of missing cases.

The newer method of capture-recapture has revolutionized the counting approach. The capture-recapture methodology was first applied in the study of fisheries and wildlife biology, and it has been adapted and used in epidemiologic studies in human populations (International Working Group for Disease Monitoring and Forecasting 1995a). In this method attention is paid to the duplicates. The number of recaptured people is used to estimate the degree of undercount which yields an ascertainment corrected rate (LaPorte et al. 1992a, LaPorte et al. 1992b). The assumption is that each estimate is independent of each other estimate.

The simplest capture-recapture model is two-sample model. In this way the first sample provides the cases for marking and the second sample provides the recaptures. But this should be used with caution because of the possible dependency of the sources. The number of cases can be calculated by the formula (LaPorte et al. 1993, Cochi et al. 1989):

$$N = \frac{(M+1)(n+1)}{(m+1)} - 1$$

N= Estimate of the number
M= Number in first sample
n= Number in second sample
m= Number of "marked" items in second sample

$$Var(N) = \frac{(M+1)(n+1)(M-m)(n-m)}{(m+1)2(m+2)}$$

$$95\%CI = \pm 1.96\sqrt{Var(N)}$$

With this technique, in order to have accurate and unbiased data about diabetes incidence, multiple independent sources should be used. These may include diabetic clinic and family physicians, prescriptions for insulin, reagent strips and insulin syringes, and hospitals (Bruno et al. 1990, Bruno et al. 1994). In this way source dependencies can be modeled and taken into consideration when estimating the number of cases. It is possible to allow for dependency among the lists and heterogeneity in the population (International Working Group for Disease Monitoring and Forecasting 1995a).

It has been reported that virtually all papers published after 1987 have used the capture-recapture method to check case ascertainment. Most of the studies have used 2 information sources. The remaining have used several sources and shown ascertainment adjusted rates (International Working Group for Disease Monitoring and Forecasting, 1995b).

In this thesis we have used p values and confidence intervals for hypothesis testing. The cut-off levels for statistical significance were chosen arbitrarily; $P<0.05$ as significant. Two possible errors may arise when using P to make a decision. We can obtain a significant result and reject the null hypothesis (type I error or $\alpha$) or we may obtain a non significant result when the null hypothesis is not true (type II error or $\beta$). If $\beta$ is the probability of committing a type II error, then $1-\beta$ is called the power of the test of hypothesis. In other words, the power is the probability of avoiding type II error. The value of $\beta$ depends upon the size of effect that one is interested in and also the sample size. The greater the power of the study the more sure we can be, but a greater power requires a larger sample. Therefore in order to have a high probability of finding a true effect of a given magnitude an appropriate sample size is required.

The interpretation of the p value is problematic, it cannot convey all the necessary information therefore the confidence interval is also needed. The confidence interval is a range of values of which we can be confident that it includes the true value. A wide confidence interval is an indication of low power (Pagano and Gauvreau 1993, Altman 1995).

Fifty six people or 65.1% of those asked to take part agreed to become subjects in this study. Little is known about the non-responders in this study. Those who did not take part in the study might have poor glycemic control or there might be other factors which distinguish non-responders (for instance, they were busy with their jobs and it was not convenient for them to take part in the study during weekdays and weekends). In a study in New Zealand it was found that establishing the incidence and prevalence of type I diabetes amongst adults is less easy than for children (Scott and Brown 1991). This may be the case for the group who were eligible to participate in our study too.

This response rate is comparable to that reported in similar studies. In Sydney in a study on the validation of a modified food frequency questionnaire in adolescents with type 1 diabetes, out of 211 eligible individuals, 60 were approached to join the study. Thirty five of them completed the 4 day food record and the one week food frequency questionnaire, a response rate of 58.3% (Garnett et al. 1994).

A random sample selected from a diabetic clinic was used in a study in Ireland on the dietary intake of adults with type 1 diabetes. Overall, 190 patients were invited to participate but 31 were not contactable and 23 refused to participate, fourteen either did not return or failed to complete their food records. The response rate for this study was therefore 64.2% (Humphreys et al. 1994). In another dietary study in diabetic children, out of 234 children attending clinics 170 agreed to take part in the study and 168 completed dietary records, a response rate of 71.8% (Hackett et al. 1988).

In the study reported in this thesis people with diabetes were asked to recruit a control subject (similar age and sex). Only 16 controls were recruited by the patients and the rest were volunteers from the University, the public hospital and industry. Many volunteers declined when they were told that they needed to give a blood sample. For this reason there was not enough opportunity to select people with similar age. Other studies have noted similar difficulties in recruiting a control group. For instance, in Sydney only 16 children without diabetes were recruited by the families of 38 diabetic children (Ballinger et al. 1994).

Control subjects in this study were overall slightly younger than the patients, but other demographic characteristics ie sex, marital status, education and income level and country of birth were satisfactorily matched. In a study in Sweden the social situation of 91 young adults (37.2 years) with type 1 diabetes since childhood was compared to that of an age- and sex- matched group of 189 healthy persons (Ingberg et al. 1996). This study showed that there were very small differences between diabetic adults (without severe complications) and controls concerning education, occupation, housing conditions, and civil state.

In the study reported in this thesis all of the patients were taking insulin injections 2 times or more every day. Moses et al. (1995) reported a similar finding that the mean number of injections was 2.7 for the patients with type 1 diabetes (age 13-39 years) in the Illawarra area. The mean ± SD insulin units per kilogram of body weight in our study was 0.68±0.29 which was lower than that reported in Moses et al. (1995) study (0.8±0.3), suggesting that the populations sampled in the two studies were different.

The family history of the patients showed that 51.8% of them had a family member with the disease, 18.1% of the subjects had one or more affected first degree relatives, 3.6% had an affected mother and 7.2% had an affected father. These proportions were higher than those reported in a study on the incidence of childhood diabetes in New South Wales (Verge et al. 1994), with 1% and 2.0% for mother and father affected respectively. In another study in Boston, on the risk of

type 1 diabetes in offspring of a parent with the disease, it was reported that the risk of type 1 diabetes by age 20 was 5.4±0.9% when the father was affected and it was 2.1±0.5% when the mother was affected (El-Hashimy et al. 1995).

## 5.2 - Glycemic Control

The level of glycemic control (HbA1c%) in diabetic subjects participating in our study (8.52±2.21% and 8.56±1.91% for females and males respectively) were similar to the levels reported in other community based samples. HbA1c% was 5.22±0.56% and 5.39±0.55% for female and male controls respectively. In the Illawarra area in a report on diabetic control in patients with type 1 diabetes (age 13-39 years) HbA1c% was 3.7 SD above the mean of the reference range for non-diabetic people which compares favourably with the selected and intensively treated patients of the DCCT (Moses et al. 1995). In the New Zealand study the HbA1c% calculated from fructosamine values was 8.15±1.7% (Dunn 1996).

It was reported that in the DCCT with intensive therapy HbA1c% decreased to 6.9% after 6 months. This was 2% lower than that with conventional treatment (9% versus 7%) (Nathan 1996). It was shown that HbA1c% and body weight (% of ideal body weight) correlated positively with total cholesterol, LDL cholesterol, and triglyceride levels.

The mean HbA1c% of patients with type 1 diabetes who took part in a Spanish study was 7.7% and 7.9% for men and women respectively while 36% of the patients had an HbA1c% value >8.0% (The Diabetes and Nutrition Study Group of Spanish Diabetes Association, 1997). In another study it was found that HbA1c% was higher among patients who progressed to microalbuminuria than those who were normoalbuminuric (9.3±1.6% vs 8.4±1.4%) (Mathiesen et al. 1995).

## 5.3 - Cardiovascular Risk Factors

Patients with type 1 diabetes have excess mortality predominantly because of cardiovascular disease (CVD) (Kannel and McGee 1979). In the general population, overweight and an elevated waist to hip ratio (WHR) are associated with higher blood pressure and lipid disturbances which are risk factors for CVD.

In our study there were no differences in measurements of obesity between people with diabetes and controls. We showed that BMI for diabetic females and males were 26.1 $kg/m^2$ and 25.70 $kg/m^2$ respectively.

Weight gain is not characteristic of most people with type 1 diabetes. Reported populations with type 1 diabetes have generally had BMI measurements within an age and sex corrected reference ranges (Riley and Blizzard 1995, Dunn 1996, Dubrey et al. 1993, Shimakawa et al. 1993, Fontvieille et al. 1992, Muhlhauser et al. 1995, The Diabetes and Nutrition Study Group of the Spanish Diabetes Association 1997, Al-Muhtaseb 1991, Koivisto et al. 1996, Gill et al. 1995).

However, people with type 1 diabetes who have had intensive insulin treatment tend to gain weight. In the DCCT the mean body weight was reported as 103% of ideal weight (the DCCT Research Group (1995) considered men and women were overweight if their BMI was $\geq$27.8 $kg/m^2$ and $\geq$27.3 $kg/m^2$ respectively). On the other hand in the DCCT, in intensively treated patients there was a 2.1 fold increased risk of major weight gain which is a risk factor for cardiovascular disease in individuals with type 2 diabetes and nondiabetic individuals (Hubert et al. 1983).

The reason for weight gain with intensive therapy is not clear. This may be because of alteration in food habits (Rodin et al. 1985), or perhaps a reduction in metabolic rate (Leslie et al. 1986).

In our study the means of systolic and diastolic blood pressure in diabetic and control subjects were within the normal range for each sex. This has been

shown in other studies (Koivisto et al. 1996, The DCCT Research Group 1995, Gill et al. 1995, Mathiesen et al. 1995, Al-Muhtaseb et al. 1991). However in a study of identical twins it was found that systolic blood pressure of diabetic twins was significantly higher ($p<0.05$) than in nondiabetic twins and nondiabetic controls (duration of diabetes 18.1± 9.0 years). Diastolic blood pressure was similar in all groups (Dubrey et al. 1994). The mean duration of diabetes in this study was higher than that in our study and in other studies.

Our study demonstrated a positive relationship between means of obesity and systolic and diastolic blood pressure after correcting for age and sex. BMI entered into a model with systolic blood pressure while WHR had a significant positive effect with the model fitted with diastolic blood pressure. The models in the present study allowed for the interaction between the two groups, therefore it could be concluded that the effects of increasing BMI and WHR on blood pressure is similar for diabetic and healthy subjects. Blood pressure in diabetic subjects did not have any association with the duration of diabetes.

In a major European study, BMI in women with type 1 diabetes was associated with increased prevalence of CVD. In men increasing WHR was related to a number of adverse CVD effects (Koivisto et al. 1996).

This positive relation of body weight and blood pressure in men and women with type 1 diabetes was shown by the WHO Multinational Study of Vascular Disease in Diabetes (Chaturvedi et al. 1995). Body weight was positively associated with blood pressure and in men, with blood cholesterol. Fasting blood glucose was higher in the most obese groups in women only.

In the DCCT macrovascular events in the group treated with intensive therapy were reduced by 42%. However the number of events was small and the difference with those on conventionally therapy was not statistically significant (the DCCT Research Group 1993a). One reason may be that the mean age of the DCCT population at the end of the trial was 34 years which is relatively low. In addition, in this trial there was a reduction in both LDL cholesterol and triglyceride

which both are known as an atherogenic factors. Although there was an increased risk of weight gain in intensive treatment, the DCCT Research Group suggested that overall there is a beneficial effect of intensive treatment on the risk of developing macrovascular disease (the DCCT Research Group 1995).

Alteration in serum lipid and lipoprotein concentrations is a well established risk factor for coronary heart disease both in nondiabetic and diabetic populations. A number of studies have shown that in type 1 diabetes the serum LDL cholesterol concentration tends to be normal or low and HDL cholesterol normal or high (Kennedy et al. 1978, Nikkila and Hormila 1978, Mattock 1979, Durrington 1980, Sosenko et al. 1980, Winocour 1986).

In the present study we found that the mean plasma lipid and lipoprotein levels were within normal range and were similar to the age-adjusted values for the general population. The mean HDL cholesterol was higher in diabetic subjects than in control subjects (significant in males). In both groups females had higher HDL and lower LDL cholesterol than males, however all the values were within normalrange.

Reasonably accurate estimation of LDL cholesterol is an important aim in view of the significance of this measure as a risk factor for coronary heart disease. However the desire for a rapid, convenient, and inexpensive procedure for LDL cholesterol for clinical and epidemiological use has led to the application of the Friedwald formula. The method has the drawback that it includes intermediate density lipoprotein (IDL), which accumulates in diabetes, in the calculation (Senti et al. 1991), and the assumption is that true fasting triglyceride concentrations have been determined.

It has been shown in postprandial samples of healthy subjects, that plasma triglyceride concentration was increased, the plasma cholesterol unchanged, HDL cholesterol significantly decreased and LDL cholesterol was significantly decreased compared to measurements in the fasted states for all subjects (Cohn et al. 1988). In another study on circulating lipids in diabetic children, it was concluded that in

postprandial samples, increases in triglyceride and other lipoproteins were found in control subjects although significant changes did not occur in diabetic subjects, apart from a fall in LDL cholesterol (Azad et al. 1994). However, it should be mentioned that postprandial lipid changes in healthy individuals have been found to be age and sex dependent (Cohn et al. 1988)

Other studies also revealed that patients with type 1 diabetes had similar or slightly less atherogenic lipid profiles than similarly aged non-diabetic subjects (Dunn 1996, The DCCT Research Group 1992, Muhlhauser et al. 1995, The Diabetes and Nutrition Study Group of the Spanish Diabetes Association 1997, Dubrey et al. 1994).

It has been postulated that in insulin deficient patients reduced activity of lipoprotein lipase results in defective removal of triglyceride-rich lipoproteins from the circulation (Pyakalisto et al. 1975). Insulin therapy usually corrects this abnormality.

Weight gain is mainly thought to be undesirable because obese people tend to have higher blood lipids and lipoproteins. This was indicated in our study when it was shown that in people with type 1 diabetes and controls, at least one of the anthropometric measurements entered into the models fitted with total cholesterol, LDL cholesterol, HDL cholesterol and triglyceride. Except for BMI which had significant negative effect in the model with HDL cholesterol, the rest had positive but insignificant effects in the models. Studies reviewed in the literature indicate the relationship of lipid and lipoprotein levels with the degree of obesity within patients with type 1 diabetes (Chaturvedi et al. 1995, Koivisto et al. 1997).

In our study the mean fibrinogen level was significantly higher in diabetic subjects than that in control subjects. In diabetic subjects fibrinogen increased significantly with increasing BMI which this relation were not found in control subjects. Although regression analysis of fibrinogen on anthropometric measurements (adjusted for age and sex) in subjects with diabetes showed that

body fat% and WHR entered into the model and only body fat% had significant positive effect.

Regression analysis with blood lipids and lipoproteins, indicated that triglyceride had positive and HDL cholesterol had negative associations with fibrinogen when the analysis allowed for interaction between two groups and adjusted for age and sex. Similar results have also been found in other studies in patients with type 1 diabetes (Dubrey et al. 1994 and Ganda and Arkin 1992, Greaves et al. 1997).

Reviewing literature shows that increased rate for cardiovascular disease associated with type 1 diabetes could result from an increased blood pressure and/or a thrombogenic tendency (Dubrey et al. 1994). Fibrinogen level was an independent predictor of vascular complications in type 1 diabetes, and perhaps also in microvascular disease (Ganda and Arkin 1992, Greaves et al. 1997).

Finally the results of anthropometric measurement, blood pressure and plasma cholesterol found in our study were compared with the values reported for the Australian (non-diabetic) population (20-39 years) in the Risk Factor Prevalence Study (National Heart Foundation of Australia 1989) (table 45). All these measurements were similar between those with type 1 diabetes and in the wider population. Also the number of subjects with high blood pressure (diastolic blood pressure≥95 mmHg) were comparable with the findings of this study (5.9% of diabetic females, 13.5% of diabetic males, 0% of control females and 3.3% of control males).

**Table 45- Comparison of measurements in diabetic and control subjects in this study with measurements on a reference (non-diabetic) population in the Risk Factor Prevalence Study***

| | the RFPS* n=3065 | | Control n=47 | | Diabetic n=54 | |
|---|---|---|---|---|---|---|
| | M | F | M | F | M | F |
| WHR [a] | 0.87 | 0.74 | 0.90 | 0.74 | 0.88 | 0.76 |
| BMI ($kg/m^2$) [b] | 24.8 | 23.3 | 23.9 | 26.1 | 25.7 | 26.0 |
| sBP (mmHg) [c] | 123.4 | 112.7 | 124.7 | 117.2 | 125.4 | 113.9 |
| dBP (mmHg) [d] | 77.9 | 70.8 | 74.5 | 69.7 | 77.0 | 67.9 |
| Cholesterol (mmol/l) [e] | 5.20 | 5.02 | 5.17 | 4.67 | 4.78 | 4.47 |

* The Risk Factor Prevalence Study (National Heart Foundation of Australia, 1989). [a] Waist to hip ratio. [b] Body mass index. [c] Systolic blood pressure. [d]Diastolic blood pressure. [e] n=51 for diabetic subjects in our study.

### 5.4 - Effects on the kidneys

Diabetic nephropathy is a major cause of increased morbidity and mortality in patients with type 1 diabetes (Dorman et al. 1984, Borch-Johnsen et al. 1985, Borch-Johnsen et al. 1987). Recently it has been shown that a slightly elevated urinary albumin excretion rate (microalbuminuria) is an early predictor for later development of manifest diabetic nephropathy (Mogensen et al. 1992, Bongstad et al. 1993, Ellis et al. 1993, Viberti 1994).

The association between nephropathy and CVD in patients with type 1 diabetes has been demonstrated in many studies (Jensen et al. 1987, Borch-Johnsen and Kreiner 1987, Winocour et al. 1987, Deckert et al. 1989, Dullaart et al. 1989, Jay et al. 1991). The relative mortality rate from CVD increases several fold in patients with proteinuria but the mechanisms are not clear. The EURODIAB IDDM Complications Study Group showed that in men micro- and macro- albuminuria appeared as significant risk factors for CVD (Koivisto et al. 1996). This was not shown for women.

Measurement of an albumin/creatinine ratio in an early morning sample has been recommended for screening purposes (Row and Gatling 1997). Gatling et al. (1985) examined different methods of urine specimen collection for detection of microalbuminuria in diabetic subjects. They concluded that an albumin/creatinine ratio performed on an early morning sample was a useful initial screening test to detect an overnight AER >30 ug/min they used >3.5 mg/mmol as a cut-off point which showed 80% sensitivity and 81% specificity.

Others had shown that in non-diabetic subjects a random urine sample was a good indicator of 24 hour albumin excretion (Shaw et al. 1983). Later Gatling et al. (1988) also used >3.5 mg/mmol as a cutoff point. Hutchison et al. (1988) used >3.0 mg/mmol as cutoff point. Marshall and Alberti (1986) used >4.5 mg/mmol.

All of the mentioned studies were done on early morning urine samples and sensitivity and specificity were at least 80% for all the studies. Watts et al. (1986) used random urine specimen and the cut off point was >8.0 mg/mmol. They found 100% sensitivity and 96% specificity.

In our study in order to know albumin excretion rate in patients with type 1 diabetes, albumin:creatinine ratio was calculated in spot and overnight urine samples, expressed as mg/mmol and compared with that in control subjects. In diabetic subjects mean albumin: creatinine ratio was 0.79±2.04 and 0.61±2.01 mg/mmol in spot and early morning urine samples respectively. Both of these ratios were significantly higher than the ratios found for control subjects (0.51±1.62 in spot and 0.39±1.38 mg/mmol in early morning urine samples).

The prevalence of microalbuminuria in patients with type 1 diabetes was estimated in a study by measurement of albumin: creatinine ratio in an early morning urine sample (≥3.5 mg/mmol was used as cut-off point). All patients with microalbuminuria had a duration of diabetes of 5 years or more. In this study microalbuminuia was associated with elevated blood pressure and longer duration of diabetes (Microalbuminuria Collaborative Study group 1992).

The findings of the DCCT Research Group show conclusively that intensive therapy may delay and perhaps prevent diabetic nephropathy (The DCCT Research Group 1993a). Also it has been reported that the risk of microalbuminuria in patients with type 1 diabetes increases abruptly above a HbA1c% level of 8.1% suggesting that efforts should be focused on the patients with HbA1c% values above this threshold (Krolewski et al. 1995). It is also suggested if the ratio is 3.6 to 9.9 mg/mmol there is a high risk of more rapid progression to definite microalbuminuria and the patient should be rescreened more frequently (Marshall 1991).

In our study regression analysis with albumin:creatinine ratios indicated that systolic blood pressure and diastolic blood pressure had some effects in the spot and early morning urine samples respectively, but their effects were not significant. A similar result was found in a study in which diabetic patients with proteinuria were compared with diabetic subjects without proteinuria (Jensen et al. 1987 and Coonrod et al. 1993). There was also a positive correlation between HbA1c% and albumin creatinine ratio, but it did not reach to the significant level. In our study duration of diabetes was not a predictor of albumin: creatinine ratio in diabetic subjects.

In Denmark 200 insulin dependent diabetics with normal blood pressure were followed up for 10 years. Baseline urinary albumin excretion and glycated hemoglobin but not blood pressure were the predictors of development of microalbuminuria (Mathiesen et al. 1995).

In a longitudinal study in patients with type 1 diabetes in Denmark, the baseline characteristics of progressors and nonprogressors to microalbuminuria were compared with the healthy control subjects. It was found that there were no significant differences in BMI and serum creatinine between these three groups. Although duration of diabetes and HbA1c% were higher in progressors the difference did not reach statistical significance. This study showed that 24 h

ambulatory blood pressure measurement had a close association with changes in blood pressure and urinary albumin excretion (Poulsen et al. 1994).

The Pittsburgh Epidemiology of Diabetes Complications Study showed that HbA1%, LDL cholesterol, duration of type 1 diabetes, and sBP were significant independent predictors of micralbuminuria overall (Coonrod et al. 1993). Some studies have reported a significantly higher HbA1c% in microalbuminuric individuals (Mathiesen et al. 1989, Wiseman et al. 1984, Klein et al. 1992), whereas others have reported no significant differences (Parving et al. 1988).

In a follow-up study it was revealed that no significant correlation was seen between albumin excretion rate and age, sex, or systolic and diastolic blood pressure. A weak but significant correlation was found with the duration of diabetes. All of the type 1 diabetes patients had normal serum creatinine concentrations (Viberti et al. 1982). This last finding was also found in our study.

Previously in England it was reported that patients with type 1 diabetes and proteinuria were similar in regard to age, age at onset and duration of diabetes, body mass index and insulin dose to those patients with normal albumin excretion. There was no excess of alcohol consumption in those with proteinuria. Glycemic control and serum albumin concentrations were similar in the two groups. Serum creatinine concentration increased moderately. Systolic and diastolic blood pressures were higher in the patients with proteinuria. Concentrations of serum cholesterol, serum triglycerides, LDL cholesterol tended to be higher in patients with proteinuria but without reaching levels of significance (Winocour et al. 1987).

The EURODIAB IDDM Complications Study Group and the WHO Multinational Study of Vascular Disease in Diabetes Study Group have reported that the increased cardiovascular risk (raised blood pressure and total cholesterol) associated with microalbuminuria in patients with type 1 diabetes for more than 5 years was also apparent in those with diabetes for 1-5 years. Microalbuminuria before 5 years was more likely to be transient or reversible (Stephenson et al.

1994). In Finland it was shown that hospitalization for diabetic nephropathy was very low during the first 8 years of diabetes duration, after which it increased to a maximum of 1.6 to 2.0% of the patient population per year (Tuomilehto et al. 1997).

In a study about exercise testing as a predictor of microalbuminuria among type 1 diabetes patients it was found that the postexercise urinary albumin creatinine ratio was positively associated with this ratio after 10 years. This association was independent of HbA1, systolic blood pressure, body mass index, and duration of diabetes (O'Brien et al. 1995).

## 5.5- Effect of Socio-Economic Status on Metabolic Control in Diabetic Subjects

In our study after correcting for age, sex and income, we showed that the higher the educational achievement the lower HbA1c% level in people with diabetes. Although this association was not significant ($p<0.1$).

It has been reported that people with type 1 diabetes in lower socio-economic groups tend to have poorer glycemic control (Virtanen 1992). Other studies have not shown any difference in glycemic control between socio-economic groups (Robinson et al. 1984, Lundvigsson 1977, Marteau et al. 1987). All of these studies were performed on relatively few subjects and may not have had sufficient power to demonstrate a relationship between diabetic control and socio-economic class. This may be true for our study too.

Recently these relationships have been examined in 2387 European patients. The mean percentage of HbA1c% was highest in the primary educated men and women (ie those in the lowest educational group). Total triglyceride and cholesterol levels were higher in primary educated than in college-educated men. College educated people were most likely to partake in vigorous exercise (Chaturvedi et al. 1996). Others have shown that the proportion of patients with

three or more risk factors for cardiovascular disease was much higher in the lowest socio-economic categories (Connolly and Kesson 1996).

### 5.6 - Dietary Intake

Evaluation of food and nutrient intake is an important part of the appraisal of the management of adults with type 1 diabetes. Diet has been a cornerstone in the management of type 1 diabetes, but there are few published data on the relationship between dietary intake of adults with type 1 diabetes and metabolic control. The main problem with dietary intake estimation is the measurement techniques.

In our study dietary intake was assessed by diet history (Burke method). This method was chosen because it was applied in the DCCT for the assessment of usual dietary intake of patients with type 1 diabetes (age 13-39 years).

As part of the DCCT the long-term reproducibility of the meal-based diet history method was demonstrated. Trained interviewers performed diet histories at two intervals at the end of year 1 and year 2 of the trial. Mean daily intakes of energy, protein, carbohydrate, total fat, saturated fatty acids, monunsaturated fatty acids, polyunsaturated fatty acids, cholesterol, and dietary fibre were determined. No statistically significant differences in energy and nutrient intakes were observed between the intensive and conventional treatment group at either year 1 or year 2. The strong correlation between two diet histories obtained 1 year apart in the same subjects indicated good reproducibility of the instrument and the stability of the diets of the people in two groups during the study. The major strength of the diet history, compared with dietary assessment methods, is its focus on usual intake. It was believed that diet history was the best method to assess usual intake of a single person over an extended period, and this was the most appropriate method for investigating associations between dietary intake and biochemical or other parameters reflecting long-term dietary habits. It was shown

that diet history was reproducible at least in the DCCT population, which represents both sexes and a broad age range. Validation studies of the method were difficult because of the lack of a gold standard for measuring usual dietary intake (Schmidt et al 1994).

In another study a modified food frequency questionnaire in adolescents with insulin dependent diabetes was validated in Sydney. Thirty five subjects 12-18 years completed the 4 day food record and the one week food frequency questionnaire. The mean nutrient intakes from both dietary methods were compared with the National Dietary Survey and the Dietitians Association of Australia dietary recommendations. Energy, protein, fibre and vitamin C intakes were higher among the subjects with type 1 diabetes. It was concluded that the use of a food frequency questionnaire was easier and more attractive to adolescents and its use is suggested for the measurement of short-term dietary intakes (Garnett et al. 1994).

Dietary intakes of subjects from 30 centres who had participated in the EURODIAB IDDM Complications Study were assessed by a validated 3-day food record. However this method did not give adequate information on long-term energy requirements (Toeller et al. 1996). The results of this study are compared with ours and presented in table 46. The method of dietary assessment, age and cultural background of the subjects in these studies were different from ours, therefore the dietary intakes should be interpreted with caution.

**Table 46- Comparison of energy and nutrient intake in patients with type 1 diabetes in the EURODIAB IDDM Complications Study and the present study**

| | EURODIAB | | present study | |
|---|---|---|---|---|
| | F (n=1410) | M (n=1458) | F (n=17) | M (n=37) |
| Protein (energy%) | 17.8±3.7 | 17.4±3.3 | 21.5±4.4 | 21.1±4.1 |
| Fat (energy%) | 37.9±7.2 | 37.9±7.3 | 28.3±10.0 | 31.2±6.7 |
| CHO[a] (energy%) | 43.1±7.2 | 41.9±7.3 | 47.7±13.6 | 45.2±7.9 |
| Alcohol (energy%) | 1.2 | 2.7 | 1.6 | 2.7 |
| Fibre (g/day) | 16.8±6.5 | 19.3±7.7 | 30.7±7.6 | 41.6±11.1 |
| SAFA[b] (energy%) | 13.8±3.5 | 13.8±3.6 | 12.8±5.3 | 13.2±3.5 |
| cholesterol (mg/day) | 331±173 | 413±207 | 178.2±65.9 | 299.3±137.4 |
| Energy (kcal/day) | 2064±545 | 2706±702 | 1904±485 | 2827±609 |
| Energy (kJ/day) | 8668±2289 | 11365±2948 | 7976±2033 | 11837±2550 |
| Protein (g)/bwt (kg)[c] | 1.5 | 1.6 | 1.5 | 1.8 |

[a]carbohydrate. [b]saturated fatty acids. [c]protein (g)/body weight (kg).

Recently the nutritional pattern of Spanish people with insulin dependent diabetes was studied and its relationship with targets of metabolic control was explained. In this study seven-day food diaries were used. Energy intake was between 1623 and 2217 kcal/day (6.8-9.3 kJ/day), distributed as follows: 39.5-40.0% from carbohydrate, 19.0-20.0% from protein, 40.5-41.5% from fat (13.2-13.9% saturated fatty acids, 5.2-5.8% polyunsaturated fatty acids, and 20.7-21.9% monounsaturated fatty acids). It was concluded that consumption of the Mediterranean diet in Spain with high MUFA content might have helped to maintain good diabetic control (7.7 in males and 7.9 in females) among these patients (The Diabetes and Nutrition Study Group of the Spanish Diabetes Association 1997). Comparing the intake of monounsaturated fatty acids

(energy%) with that of our study shows that patients in our study consumed much lower amount of MUFA (11.0-11.9%).

Our results showed that there were no significant differences in regard to energy and the contribution of macronutrients in daily energy intake, between individuals with diabetes and control group. This was also found in the Nurses Health Study, which the dietary intake of women with type 1 diabetes was compared with nondiabetic women. Dietary information was obtained by a self-administered semiquantitative food frequency questionnaire, which asked how often the individual consumed a specified amount of various foods on average over the preceding year. Compared with control women, diabetic women consumed diets high in protein and fat and lower in sucrose. These differences were small yet statistically significant. Diabetic women tended to avoid desserts and sweets, sugar containing beverages, and alcoholic beverages but consumed more meat and meat products. Intake of foods high in complex carbohydrates (e.g. bread, rice, pasta, and potatoes), vegetables and fruits were similar in diabetics and control women (Shimakawa et al. 1993).

Similar result was reported in another study in Ireland. The daily energy intake was similar in adult patients with type 1 diabetes and in the age-matched subjects in the Irish National Survey (Humphreys et al. 1994). The consumption of starch and dietary fibre by diabetic subjects was significantly higher than in the general poulation. This was found in our study too when the consumption of starch and dietary fibre was compared in diabetic and control subjects.

Other methods also have been used for the assessment of dietary intake among individuals with type 1 diabetes. In Tasmania the validity of a food frequency questionnaire (FFQ) was studied in female and male adults with type 1 diabetes (age 17.5-73.8 years). There was a significant decrease in dietary energy and macronutrients estimates upon readministration of the questionnaire. The dietary intake also was assessed by 2 days of weighed dietary records (Riley and Blizzard 1995). The comparison between the Tasmanian and present study are

shown in table 47. As it is shown there are some similarities and dissimilarities between these two sets of results. The number of male and female subjects and the age ranges were different in the two studies and as mentioned before, the method for dietary assessment in these two studies was different.

**Table 47- Comparison of dietary intake in patients with type 1 diabetes in the Tasmanian study and present study**

| | Tasmanian study (n=84) | | Present study |
|---|---|---|---|
| | FFQ[a] | Weighed record | (n=54) |
| Energy (kJ) | 8621±2875 | 8533±2448 | 10622±2990 |
| Fat (g) | 93.3±40.1 | 86.5±33.4 | 85.3±37.3 |
| Protein (g) | 95.5±31.6 | 88.5±23.6 | 129.8±37.8 |
| Carbohydrate (g) | 210±65.8 | 224±67.9 | 302±90.1 |
| Saturated fat (g) | 37.1±18.1 | 38.1±17.6 | 31.4±13.8 |
| Cholesterol (mg) | 264±117 | 260±133 | 261±131 |

[a]Food Frequency Questionnaire.

In a Danish review it was concluded that long-term high protein intake may have a pathogenic influence on the development of late diabetic complications (Pedersen et al. 1990). In our study the mean contribution of protein in daily energy intake was 21.5% for females and 21.1% for males. In a study the average protein intake of a population with type 1 diabetes by 7 day recall interviews was found to be 19-20% of total energy intake (Palving et al. 1987). Similar results was found in other studies (The Diabetes and Nutrition Study Group of the Spanish Diabetes Association 1997, Shimkawa et al. 1993). However in the Tasmanian study protein contributed 19% of energy intake when diet was assessed by FFQ and 18% by weighed food record.

Available data suggest that alcohol produces little or no immediate effect on blood glucose levels in patients with type 1 diabetes (Koivisto et al. 1993, Gin et al. 1992). The alcohol consumption by patients with type 1 diabetes and control group in our study was similar in respect to the amount, its frequency and number of drinks. Results of different studies also show that many people with type 1 diabetes as in the general population consume alcohol on a regular basis. The metabolic response to alcohol depends upon the quantity of alcohol consumed. In our study only one of those diabetic patients who drank more than 20 drinks each time had a high HbA1c% (11.5%). Alcohol consumption by patients with type 1 diabetes in poor nutritional state and/or dependent on alcohol is associated with an increased risk of hypoglycemia (Meeking and Cavan 1997).

Others have shown that patients with type 1 diabetes appear to develop an increase in blood pressure in response to dietary sodium more readily than controls. In one study there was no significant difference in the various blood pressure measurements between patients and controls, but the increment in blood pressure upon transition from low to high salt diet was significantly higher with respect to 24h and daytime mean arterial pressure (Strjek et al. 1995). The effects of moderate sodium reduction on blood pressure was assessed in 16 patients with type 1 diabetes, and it was concluded that reduction of sodium resulted a slight reduction in diastolic blood pressure (Muhlhauser et al. 1995). Results from our study indicated that male patients with type 1 diabetes had a significantly higher intake of sodium in their diet than those in the control group. Their intake was also higher than the recommended dietary intake.

Finally the dietary intake of diabetic and control subjects were compared with the intakes of people in New South Wales ( NSW Health Department 1994) and shown in tables 48 and 49. The differences must be looked at with caution because the intakes in these studies and our study were assessed by different methods and the sample sizes are extremely different. These studies were done several years ago and in different areas. However in regard to fat (g) females in our

study consumed much less but control males fat intake was similar to the results of these studies. Alcohol intake in our subjects was less than that in people in the other studies. There was a big difference in the amount of dietary fibre in all subjects in our study compared to its intake among subjects in the two studies. Perhaps people are becoming aware of the beneficial effects of vegetables and fruits in their diets. The contribution of protein to daily energy intake was higher among diabetic and control subjects in our study.

On the other hand a more recent data has been reported on a survey of dietary intakes of the Australian population which is much more comparable to our results (Baghurst et al. 1996). These findings show the trends in lower fat intake and higher carbohydrate intake among Australians (table 50).

**Table 48- Comparison of mean daily intake, energy and selected nutrients in diabetic and control subjects with the intakes in NSW**

| Nutrient | NDSA[a] | | WSDS[b] | | Control | | Diabetic | |
|---|---|---|---|---|---|---|---|---|
| | M | F | M | F | M | F | M | F |
| | n=698 | n=721 | n=206 | n=309 | n=30 | n=17 | n=37 | n=17 |
| En (MJ)[c] | 11.17 | 7.53 | 10.76 | 8.31 | 11.42 | 7.64 | 11.84 | 7.97 |
| Fat (g) | 113 | 78 | 104 | 79 | 106 | 63 | 98 | 57 |
| SFA (g)[d] | 45 | 31 | 42 | 31 | 44 | 26 | 36 | 22 |
| P:M:S | 0.36: | 0.39: | 0.41: | 0.43: | 0.32: | 0.43: | 0.50: | 0.44: |
| Ratio[e] | 0.96: | 0.94: | 0.86: | 0.87: | 0.86: | 0.83: | 0.93: | 0.85: |
| | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 |
| Sug (g)[f] | 120 | 92 | 153 | 126 | 142 | 111 | 123 | 111 |
| Alc (g)[g] | 26.7 | 9.5 | 13.8 | 4.6 | 9.4 | 7.6 | 8.3 | 3.3 |
| Ch (mg)[h] | 419 | 319 | 349 | 268 | 351 | 200 | 299 | 178 |
| fibre (g) | 23.8 | 19.6 | 25.2 | 23.4 | 32.4 | 30.3 | 41.6 | 30.7 |
| sod (mg)[i] | na | na | 3429 | 2506 | 2993 | 2234 | 3935 | 2447 |
| cal (mg)[j] | 856 | 667 | 963 | 835 | 1196 | 924 | 1197 | 1157 |

[a] National dietary survey of adults, Sydney subsample, 1983. [b] Western Sydney dietary dietary survey, 1989-1990. [c] Energy, d Saturated fat, [e] Polyunsaturated: monounsaturated: saturated fatty acid ratio, [f] sugars, [g] alcohol, [h] dietary cholesterol, [i] sodium, [j] calcium

**Table 49- Comparison of contribution of macronutrients to total energy intake, NSW with those in diabetic and control subjects**

| | NDSA [a] | | WSDS [b] | | Control | | Diabetic | |
|---|---|---|---|---|---|---|---|---|
| Nutrient | M | F | M | F | M | F | M | F |
| (Energy%) | n=698 | n=721 | n=206 | n=309 | n=30 | n=17 | n=37 | n=17 |
| Fat | 36.8 | 37.7 | 36.4 | 35.9 | 34.1 | 30.1 | 31.2 | 28.3 |
| SFA [c] | 15.1 | 15.5 | 14.6 | 14.0 | 15.9 | 13.8 | 13.2 | 12.8 |
| CHO [d] | 39.7 | 41.1 | 43.0 | 44.9 | 42.4 | 46.2 | 45.2 | 47.7 |
| Protein | 16.7 | 17.6 | 16.8 | 17.6 | 20.7 | 20.6 | 21.1 | 21.5 |
| Alcohol | 6.6 | 3.5 | 3.8 | 1.6 | 2.5 | 2.7 | 2.3 | 1.6 |

[a] National dietary survey of adults, Sydney subsample, 1983. [b] Western Sydney dietary survey, 1989-1990. [c] saturated fatty acids. [d] carbohydrate.

**Table 50- Comparsison of energy and nutrient intake of the Australian population (1993) with those of diabetic and control subjects**

| | Australian population | | Control | | Diabetic | |
|---|---|---|---|---|---|---|
| | M | F | M | F | M | |
| | n=736 | n=997 | n=30 | n=17 | n=37 | n=17 |
| Energy (kJ) | 10400 | 8044 | 11424 | 7644 | 11837 | 7976 |
| Energy (kcal) | 2476 | 1915 | 2731 | 1826 | 2827 | 1904 |
| % energy as: | | | | | | |
| protein | 16.8 | 17.8 | 20.7 | 20.6 | 21.1 | 21.5 |
| total fat | 33.9 | 32.5 | 34.1 | 30.1 | 31.2 | 28.3 |
| SAFA | 13.8 | 13.2 | 15.9 | 13.8 | 13.2 | 12.8 |
| MUFA | 11.7 | 11.2 | 13.4 | 11.1 | 11.9 | 11.0 |
| PUFA | 5.7 | 5.5 | 4.9 | 5.3 | 6.1 | 5.6 |
| total CHO | 44.5 | 46.5 | 42.4 | 46.2 | 45.2 | 47.7 |
| alcohol | 3.1 | 1.5 | 2.5 | 2.7 | 2.3 | 1.6 |
| P:S ratio | 0.47 | 0.47 | 0.32 | 0.43 | 0.50 | 0.44 |
| chol (mg) | 311 | 307 | 351 | 200 | 299 | 178 |
| fibre (g) | 28.4 | 34.8 | 32.4 | 30.3 | 41.6 | 30.7 |
| sodium (mg) | 3113 | 3036 | 2993 | 2234 | 3935 | 2447 |

## 5.7 - Dietary Adherence

It has been shown that dietary adherence is an essential component of successful diabetes management. However the the goals of the recommendations have been controversial and changed during periods of years (American Diabetes Association 1998).

In our study the recommendations on dietary total fat, carbohydrate and protein were achieved in 50%, 33% and 37% of patients respectively. Only 4 individuals could meet all the recommendations for macronutrients (energy%) in their diets. When these results were compared with those in controls, it was found that the number of controls who had met the criteria for protein (<20%) was significantly higher than that of patients. Although half of the patients had less than 30% of energy from fat in their diets, but overall 74% of the patients were found to consume 10% or more of energy from saturated fatty acids.

Lower fat intake among these patients and control subjects may be the result of a national trend in the general population towards lower fat intake. It was reported that total fat (energy%) decreased significantly and total carbohydrate (energy%) increased significantly among male and female Australians from 1988 to 1993 (Wahlqvist and Kouris-Blazos 1997). Similar changes have also been seen in British people (Stephen and Sieber 1994).

The EURODIAB IDDM Complications Study Group showed that the recommendation for total fat, saturated fatty acids and carbohydrate were met by only 14%, 14% and 15% of patients respectively (Toeller et al. 1996). In Ireland only 11% of subjects consumed total fat less than 30% of total energy (48% of fat intake as saturated fatty acids). Carbohydrate intake (42% of total energy) was lower than recommended in all age groups. Only 6.5% achieved the dietary goal of carbohydrate. The intake was similar in diabetic patients and the general population. It was concluded that the dietary recommendations for diabetic patients had not been achieved (Humphreys et al. 1994).

In our study no model could be constructed when the dependent variable was either dietary carbohydrate (energy%), fat (energy%) or protein (energy%) and independent variables were age, sex, education, income level. Duration of diabetes, dose of insulin, number of injections and HbA1c% did not enter into a model with the above mentioned dependent variables in diabetic subjects. In the Irish study also, there was no correlation between HbA1c% and dietary intakes (Humphreys et al. 1994).

In Sydney an education program was conducted to improve dietary compliance in patients with type 1 diabetes. The analysis of data showed that the number of patients who satisfied compliance criteria (at least 45% of energy from complex carbohydrates) increased significantly after 6 months education program (13.9% vs 38.9%). Those who achieved the recommended goal of a high carbohydrate/low fat diet were more likely to have adequate glycemic control. It was also concluded that subjects who complied with one aspect of the dietary goals did not necessarily comply with other aspects (Webb et al. 1984). Others studied the understanding of and compliance with a controlled carbohydrate diet of patients with type 1 diabetes (14-85 years). The pattern of eating of 178 patients was established by a seven-day food record and their dietary knowledge by a questionnaire. It was found that in this group of patients the understanding of a controlled carbohydrate diet and adherence to it were associated with better glycemic control but adequate compliance was rare (McCulloch et al. 1983).

Similar studies have been conducted in children. For instance in Sydney the dietary intake of children (5-10 years) with type 1 diabetes was studied and compared with the dietary recommendations. These children were selected from the diabetic clinic and who had been educated to have a diet with over 50% of energy from carbohydrate and less than 30% of energy from fat. Dietary intakes were determined by a three day food diary. Children with diabetes did not meet the Australian dietary guidelines for individuals with diabetes for complex carbohydrate, fibre, fat, saturated fat or salt, but did meet the recommended

restriction of cholesterol and refined sugar. These children compared to their controls had higher intakes of most nutrients and could meet the Australian dietary guidelines. Regular dietary assessment by a dietitian was suggested to ensure that nutrient requirements and treatment goals continue to be met (Ballinger et al. 1994).

A group in England used a 3-day food record to evaluate diet, and found that the amount of energy consumed by diabetic children was similar to those expected of non-diabetic children. They ate more fat and fibre, but less sugars and carbohydrates. The amount of carbohydrate was less than the dietary recommendations. The comparison of actual intake with the dietary recommendations suggested that the dietary advice were being applied only to newly diagnosed patients more than to those with long standing disease (Hackett et al. 1986).

Later dietary variation of children with type 1 diabetes was studied. There were no significant differences in the day to day or within subject variations in intakes over the three days either between the sexes and the social class, and day to day variation in the energy and carbohydrate was less than non-diabetic children. It was found that compliance with the dietary prescription deteriorated during the day. The results support the idea that dietary advice for diabetic subjects should be qualitative or at most semiquantitative (Hackett et al. 1988).

In England the dietary intake of 63 adult diabetic patients requiring insulin or oral hypoglycemic agents was assessed by a 7-day food diary. These patients had never been taught about carbohydrate exchanges in their diets. It was indicated that 70% achieved the dietary recommendations for fat intake and 30% the recommendations for carbohydrate intake. Protein contributed 20% of daily energy intake. Those with BMI of more than 30 reported lower energy intakes than the other subjects. Eleven per cent of the subjects had cholesterol intake higher than the recommendations. Only two of the subjects reported consuming

alcohol during the 7-day period, which supposed to be underreported (Pearson et al. 1996).

The majority of patients in our study had 2 insulin injections every day. In Germany a 6-year follow up study was undertaken to document that a strict dietary regimen is not necessary when patients are on intensified insulin treatment. Six hundred and thirty six patients were re-examined (out of 771 in the original program). Patients with a higher degree of diet liberalization injected insulin or used an insulin pump more frequently. It was concluded that intensified therapy along with a teaching program and liberlization of the diabetes diet was not associated with an adverse effect on glycemic control. No clinically significant associations were found between the extent of diet liberalization and metabolic control (Muhlhauser et al. 1995).

In our study the food items in each food group in the daily diets that contribute to the overall dietary intake of patients with type 1 diabetes and control group were identified. It was found that there was no difference between diabetic and control subjects in regard to food consumption except fruits which diabetic subjects consumed significantly more. In both group the consumption of low glycemic index foods like legumes was very low. Similarly it has been reported that legume intake in Australia in 1993 was less than 5 gram/day (Baghurst et al. 1996).

The beneficial effect of lowering the glycemic index of diet in diabetic subjects has been studied in patients with type 2 and type 1 diabetes. In 12 insulin dependent diabetic patients the consumption of a low glycemic diet improved fructosamine, fasting blood glucose, 2-h postprandial blood glucose and serum triglycerides, but no significant improvement were found in body weight, HbA1c%, insulin requirement and other circulating lipids (Fontvieille et al. 1992). Earlier studies could not show any beneficial effects of a low glycemic index on the glycemic control of patients with type 1 diabetes (Calle-Pascual et al. 1988).

The overall diet quality of the subjects in our study was assessed by comparing the subjects' dietary intakes with several Australian and American dietary goals and recommendations. It has become clear that focusing on one single food or nutrient does not provide a complete picture of the total diet quality (Drenowski et al. 1996). We could detect no difference in quality of the diet of patients and controls. Females in both groups had higher dietary score and among dibetic subjects the difference between two sexes was significant.

In order to see the relation between the diet quality and glycemic control, regression analysis was performed. We showed that the diet quality of patients had a negative effect in a model with HbA1c%, after correcting for age, sex, insulin dose and number of insulin injections. But this effect was not significant ($p<0.1$).

It was also interesting that diet quality of patients with type 1 diabetes and control group had significant positive correlation with the level of education. Different studies have shown that people with higher socioeconomic status had diets more consistent with current dietary guidelines (Aro et al. 1986, Pietinen et al. 1988, Baghurst et al. 1990, Bolton-Smith et al. 1991, Hulshof et al. 1991, Steele et al. 1991, Prattala et al. 1992, Smith and Baghurst 1992, Shimakawa et al. 1994).

Insulin replacement is the main therapy for patients with type 1 diabetes but the optimal treatment requires a careful balance of food, insulin, and physical activity. Poor diabetes control is frequently attributed to either inadequate dietary advice (Knowles et al. 1965) or poor adherence to dietary prescription (Christensen et al. 1983).

Only 27% of the diabetic subjects in our study followed their prescribed diets. We also showed that those who always adhered to their diet had lower level of HbA1c% compared to those rarely/never did so (this negative trend was not significant).

Non-compliance with diet has long been documented as one of the most difficult parts in diabetes management and people with diabetes regard diet as one of the biggest problems of their disease (Lockwood et al. 1986). Even later when a

liberalized diet was prescribed for patients on intensified insulin therapy, diet remained a problem for the patients (Muhlhauser et al. 1995). On the other hand diet is one of the most controversial subjects in control of diabetes and its risk factors. It has been reported that instructions varied from clinic to clinic and the prescribed pattern changed with time. This made many patients confused and cynical about this aspect of their management ( Anonymous 1990).

In all people with diabetes, but especially in young patients, dietary advice should be part of a sensitive and careful education program that involves the family and aims to deviate as little as possible from customary habits (Anonymous 1990).

Earlier reports have shown that there were a variety of reasons for poor dietary adherence, including both inadequate knowledge of dietary management and inadequate motivation (Hinkle 1961, Glanz 1979, Stone 1961, West 1973). In a camp for diabetic subjects the relationship between dietary knowledge and eating behavior was studied and the patients' actual intake was recorded at regular meals (Lorenz et al. 1985). It was found that the majority of diabetic children demonstrated deficits in diet-related knowledge and skills, despite previous instruction and several years of experience in dietary management. The data implied that health care providers might often underestimate the complexity of disease management behaviors required of patients. Thus the time and the effort devoted to teach patients were probably not enough.

A poor rate of attendance for dietary review has been found in many studies (Humphreys et al. 1993, Close et al. 1992). Perhaps it might be because of limited funding and/or lack of awareness of its importance among doctors. For example most diabetic clinics in UK do not have the dietetic support to see patients even once a year to motivate, monitor or reinforce dietary advice (Page and Tattersall 1994).

In a study on dietary adherence of adults with type 1 diabetes, it was concluded that health educators should assess obstacles to dietary adherence.

Then it may be overcome only with patience, skill, practice, and guidance on the part of the patient and the dietitian (Schlundt et al. 1994). In an audit of dietary advice to diabetic patients it was found that the patients who had never been taught carbohydrate exchanges were more compliant with the dietary recommendations than those reported by other studies (Pearson et al. 1996).

Furthermore in an education program it was found that dietary compliance in patients with type 1 diabetes was only related to the initial pattern of dietary intake (Webb et al. 1984). Therefore it has been recommended that the diet therapy for diabetic subjects should be individualized, with consideration given to usual eating habits and other lifestyle factors (American Diabetes Association 1998).

In the DCCT, dietitians were involved in many activities to help foster participants enthusiasm and adherence. They assessed subjects for the existence of an eating disorder or any related underlying psychological issues. For the improvement of the dietary adherence, the dietitians counsel participants using a variety of behavioral and cognitive approaches (DCCT Research Group 1993b).

### 5.8 - Physical Exercise

Regular physical exercise is recognized to have several benefits to health, not only for those with insulin dependent diabetes but for everyone. Physical activity may have a beneficial effect on several cardiovascular risk factors: exercise enhances insulin sensitivity, decreases insulin requirements, alters the lipid profile in an antiatherogenic direction, and may also reduce blood pressure (Koivisto and DeFronzo 1986, Kelemen et al. 1990, Ekelund et al. 1988, Salonen et al. 1988). It may increase strength and flexibility and lead to an improved sense of well being and enhanced quality of life. In a recent study in Finland metabolic control and insulin sensitivity of athletes with type 1 diabetes and sedentary men with type 1 diabetes were compared. They found that in athletes insulin requirements

decreased and metabolic control was impaired but insulin sensitivity was not enhanced. It was concluded that an intense glucose monitoring and education may be required for the maintenance of better glycemic control in athletes with type 1 diabetes (Ebeling et al. 1995).

Others in Switzerland showed that increasing physical activity is safe and does not result in more hypoglycemic episodes in well-controlled patients with type 1 diabetes. They also showed that there was a relationship between increased physical activity and loss of abdominal fat a decrease in blood pressure and lipid-related cardiovascular risk factors (Lehmann et al. 1997).

In our study the extent and duration of physical exercise was very similar within diabetic and control subjects. No significant association was found between extent and duration of diabetes and glycemic control. Before beginning exercise 61% of individuals with diabetes had a high carbohydrate snack.

### 5.9 - Quality of Life

It is well known that the combination of clinical information with those of quality of life, could provide a useful criteria for evaluating the medical outcome. For this reason a specific health-related quality of life measure (the Diabetes Quality of Life Measure) and a multidimensional questionnaire as the SF-36 were applied in our study.

The Diabetes Quality of Life (DQOL) measure was developed by the DCCT Research Group (1988). Its reliability and validity in a group of patients with type 1 diabetes (n=192) have been shown. It was found to be useful for evaluating the quality of life in patients with type 1 diabetes.

The use of the MOS Short-Form General Health Survey to measure functioning and well being of patients with chronic conditions like type 1 and type 2 diabetes has been suggested (Stewart et al. 1989). These findings emphasize the potential usefulness of generic (non-disease-specific) health measures for the

purpose of monitoring progress and for use as outcomes in studies of patients with chronic conditions.

Later the effects of type 1 diabetes on patients (n=240) perception of their quality of life was examined and compared to the psychometric properties of the Medical Outcome Study Health Survey 36-item (SF-36) versus the DQOL (Jacobson et al 1994). The reliability coefficients (Cronbach'α) for the DQOL subscales ranged from 0.47 to 0.87 and from 0.78 to 0.91 on the SF-36 subscales. The findings, except for those relating to the diabetes worry subscale (α=0.47), were very similar to those reported previously (Stewart et al. 1989, Ware and Sherbourne 1992, Wachtel et al. 1992, Wu et al. 1991). This confirms our results that the reliability coefficient (Cronbach'α) for diabetes worry subscale was the lowest (α=0.51).

Comparing the DCCT results with those of our study reveals that the DCCT Research Group (1988) scored satisfaction subscale from 1 (very satisfied) to 5 (very dissatisfied) and impact and worry subscale rated from 1 (no impact or never worried) to 5 (always affected or always worried). In our study the method proposed by Jacobson et al. (1994) was applied where 0 represents the lowest possible quality of life and 100 represents the highest. It was shown that a mean satisfaction score of 1.93 (almost equal to 77 on transformed score) is a score that indicates a high level of satisfaction (Jacobson and the DCCT Research Group 1995).

In these studies scales had a high degree of internal consistency as measured by Cronbach'α. Diabetes worry subscale revealed lower a level of internal consistency. Like the DCCT findings the intercorrelations between the DQOL subscales were the strongest between the impact and satisfaction scales and between the two worry scales. It was suggested that the lower level of internal consistency of worry items might reflect a tendency of patients to focus on a particular concern, thereby exhibiting relatively less worry about their issues. Another explanation was that the diabetes-related worry scale consisted of only 4

items, whereas the satisfaction scale had 15 items, the impact scale 20 items, and the social worry scale 7 items. It was concluded that the DQOL could be used as a screening measure to detect unstated or understated concerns of patients about their diabetes.

In the study of Jacobson et al. (1994) the diabetes and social worry subscales of the DQOL were typically less strongly correlated with the SF-36 subscales than the satisfaction and impact subscales. On the other hand, the pain and physical functioning subscales were the least strongly correlated with the subscales of the DQOL. In our study it was found that the physical functioning was the least and social functioning less correlated with the subscales of DQOL.

In the above patients, quality of life measures were not influenced by sex or education level. Older individuals reported worse physical functioning but no effects emerged in relationship to other SF-36 scales. The association of marital status and quality of life suggested that separated or divorced individuals generally experienced worse quality of life than those who were single or married. Our study did not show similar results with marital status for patients with type 1 diabetes, although similar results were found among controls but the differences were not significant.

The findings of Jacobson et al. (1994) indicated that the SF-36 was less sensitive than the DQOL to lifestyle issues while it was appeared to be more sensitive to changes in the number and severity of complications. The findings suggested that these two approaches to measure quality of life overlap but were not mutually exclusive. Therefore incorporating both measures may be useful. It was found that patients with type I diabetes without complications or other impairing conditions, will typically rate themselves on the DQOL as generally satisfied or only slightly impacted (a rating of about 2.0 on the original scale and a rating of 70-75 on the 100-point scale), and concluded that these two measures complement one another. Similarly our study showed that the mean of the DQOL subscale for female and male patients were between 74-81, showing that this

group of patients had good quality of life. In other word these patients did not have any serious complications which affects their quality of life.

As part of the Pittsburgh Epidemiology of Diabetes Complications Study, the psychological factors associated with complications of type 1 diabetes were investigated (Lloyd et al. 1992). Application of the DQOL measure showed that the quality of life of patients (diagnosed for more than 25 years), was significantly poorer among those with macrovascular disease or nephropathy compared with patients free from all complications (mean scores on QL questionnaire: 35.1 vs.30.1 and 33.6 vs. 30.1 respectively). A multiple regression analysis adjusted for sex, duration and glycemic control demonstrated that the presence of nephropathy was the most important independent predictor of quality of life. The most significant independent predictor of depression was the combination of retinopathy and macrovascular disease.

Quality of life in patients with type 1 diabetes who had pancreas/kidney transplantation was assessed (Nathan et al. 1991). It was found that the quality of life of these patients (measured by the DQOL) improved significantly after the transplantation and follow up. The averages of the 4 subscales and total score were greater than 4, with 1 representing worst score and 5 representing the best.

In another study quality of life of patients with type 1 diabetes was examined and compared with those of patients with type 2 diabetes (Mayou et al. 1990). Fifty-seven patients with type 1 diabetes were recruited from two diabetic clinics in Oxford, England. The Social Difficulty questionnaire and the Profile of Mood States scale were used. The pattern of quality of life was different from those reported by the group with type 2 diabetes. The number of patients with type 1 diabetes who mentioned moderate or great effects on at least one leisure area was significantly less, and that with some aspects of work affected was significantly more than that of individuals with type 2 diabetes. Subjects with more episodes of hypoglycemia reported more worry for their spouses especially coping at night. They were more likely to report that "diabetes affects my whole

life style". Retinopathy was not related with more reported social or psychological difficulties. The results showed that psychological factors were not associated with the duration of diabetes.

In Sweden, quality of life and metabolic control in patients with type 1 diabetes who used pen treatment instead of injections were studied at follow up 9-13 months. Metabolic control improved in those patients who had one or two injections before and quality of life of patients improved consistently (Hornquist et al. 1990). After an interval of 2 years it was found that for a great majority well being and life satisfaction was fairly stable (Hornquist et al. 1995).

The effect of intensive versus conventional diabetes treatment on quality of life measured by the DQOL, the Symptom Checklist-90R, SF-36 and intercurrent psychosocial events were examined in a greater depth (The DCCT Research Group 1996). The models using DQOL score as the outcome did not demonstrate an association of hypoglycemia with an adverse change in quality of life. Sex in the intensive treatment group and age in the conventional group were the significant variables. The adverse effect on quality of life was most evident for patients who had three or more hypoglycemic episodes that resulted in coma or seizure.

In England, quality of life of patients with type 1 diabetes (12-25 years) together with the relationship between demographic and disease variables was studied (Eiser et al. 1991). Quality of life of patients was measured by a version of the DQOL. Sixty nine patients out of 93 contacted agreed to participate in the study, and returned completed questionnaires. Nonresponders had poorer attendance records and higher fructosamine levels. The three subscales (impact/worry, social relationship, diabetes concerns) correlated highly with each other. There was no correlation between any of the quality of life subscales and either age or diabetes knowledge. Diabetes satisfaction correlated with lower insulin dose. Females reported more negative impact than males and also they tended to obtain higher score for diabetes knowledge.

In a study about depression in type 1 diabetes patients (age 26.2±9.7 year) using the Beck Depression Inventory Scale (BDI) in Greece it was concluded that the patients without chronic complications had no more depression than an age- and sex- matched healthy population (Liamou et al. 1994).

Outcome management of patients with type 1 and type 2 diabetes was assessed by the SF-36, diabetes data, and physician's ratings of patient's health status (at baseline and at 6 months follow up). The SF-36 scores ranged from a low of 53 (energy) to a high of 81 (physical functioning). The general pattern of results was different among type 1 and type 2 dibetic patients. The only variable that was associated with perceived health status was number of daily insulin injections. More frequent daily injections were associated with higher ratings of health in general. There was a discrepancy in patient and physician ratings of health status, physician's ratings being higher than those of patients themselves (Nerenz et al. 1992).

In Sweden the quality of life of type 1 diabetic patients was assessed and it was shown that overall quality of life and metabolic control were not related to either age or duration of the diabetes (Wikby et al. 1991).

The perception of well-being among 192 adult patients with type 1 diabetes was assessed through a mailed questionnaire. Analysis of results showed that only a minority of patients reported that the disease caused them considerable problems in daily life. Younger subjects had significantly more problems in daily life and more worries about complications. They felt more anxiety, lack of freedom, insecurity and reduced self-esteem (Lundman et al. 1990).

In most of the other studies there was no relationship between diabetes duration and quality of life in subjects with type 1 diabetes. In our study it was found that duration of diabetes and HbA1c% had negative effect in a model with diabetes worry scale. In which HbA1c% had significant negative effect and duration of diabetes had insignificant negative effect ($p<0.1$).

On the other hand the results of SF-36 in the current study were compared with the interim norms for Australian data (Stevenson 1996). Comparison of these two sets of data showed that the scores of SF-36 were higher in diabetic and control subjects than the Australian norms except for general health that was the lowest for the diabetics. In controls BMI had significant negative effect in the models with SF-36 subscales. Similar results were reported with the Australian norms, where SF-36 scores were decreased with increasing in body weight. Also consistent with the Australian norms, married and single subjects had higher scores when compared with separated or divorced individuals.

Also in our study regression analysis allowed for interaction between patients and controls showed that BMI enetred in the models with physical functioning, bodily pain, general health, vitality and mental health subscales with negative effect. While WHR had its negative effect in the models with physical functioning, bodily pain, general health and mental health. Their effects were significant for physical functioning. This might again suggest the importance of maintenance of ideal weight among this group of patients and controls.

# Chapter 6

# Conclusion and Recommendation

Patients in this study were very similar to the individuals who participated in the DCCT, healthy volunteers with type 1 diabetes and without severe complications. More than half of the patients were taking insulin injections two times a day, in other words were conventionally treated.

In this chapter I try to answer the questions posed at the time of proposing this research:

1- What is the glycemic control of these diabetic subjects as measured by HbA1c%?

The mean HbA1c% in our study were 8.52% (SD 2.21) and 8.56% (SD 1.91) for female and male diabetic subjects respectively. These values were higher than the DCCT intensively treated patients but lower than the DCCT conventionally treated patients.

2- Is glycemic control associated with likely determinants such as age, sex, duration of diabetes and socio-economic status?

HbA1c% significantly decreased with age. There was no significant difference in HbA1c% between diabetic males and females. No association was found with the duration of diabetes. This study showed that socio-economic differences affected diabetes control, those with the least education had the poorest diabetic control. The mean HbA1c% was associated negatively with income and education (when considered with duration of diabetes, dose of insulin, and number of insulin injections). Though these trends did not reach to the significant level.

3- Are diabetic people able to maintain optimal levels of known determinants of some of the possible complications, determinants such as weight and related measures, blood pressure, plasma lipid levels, and indicators in blood and urine samples?

The means of body measurements, blood pressure and plasma lipid levels in diabetic and control subjects were within normal ranges. This study showed that there was not much difference between people with type 1 diabetes and the control group in respect of measures including body measurements, blood lipids and lipoproteins. We showed that systolic blood pressure was significantly associated with BMI and diastolic blood pressure with WHR in men and women patients with type 1 diabetes and control subjects.

Dyslipidemia was uncommon in type 1 diabetes patients. Obesity or overweight had insignificant positive associations in regression models with total cholesterol, LDL cholesterol and triglyceride and significant negative association with HDL cholesterol, ie obesity or overweight was associated with an adverse blood lipid and lipoprotein pattern.

Plasma fibrinogen concentration in people with diabetes was significantly higher than in the control group. It rose with increasing BMI and a regression analysis of fibrinogen on anthropometric measurements showed that Body fat% and WHR entered into the model and only body fat% had significant effect. Also regression analysis with blood lipids demonstrated that triglyceride was a significant positive predictor in the model with fibrinogen, while HDL cholesterol entered into the model but its negative effect was not significant. This again suggests that to reduce fibrinogen, maintenance of ideal weight is very important along with other measures (though of course, as demonstration of an association does not necessarily imply causality, we cannot be sure from this evidence that weight maintenance will help).

The albumin: creatinine ratio in spot and early morning urine samples of patients with diabetes was also significantly higher than in the control group.

WHR entered into the regression models with the albumin: creatinine ratio in spot and early morning urine samples but its effects were not significant. HDL cholesterol had an inverse significant relationship with this ratio in early morning urine samples.

4- Is there any association between the level of glycemic control and these indicators of potential complications?

All the variables like anthropometric measurements, blood pressure, diabetes history and the blood and urine results were evaluated as independent variables in several models with HbA1c% in the diabetic group. No statistically significant associations were found.

5- How does dietary intake of diabetic subjects compare to those in control subjects? Is there any relation between dietary intake and glycemic control in diabetic subjects?

Except for the intake of starch and dietary fibre that were significantly higher in the usual daily diet of diabetic subjects, the intake of other macronutrients and alcohol by patients did not differ significantly from the control subjects. None of the same sex comparisons of energy intake and the contribution of macronutrients was statistically significantly different. No conclusion could be drawn when energy, macronutrients and micronutrients were considered in relation to diabetic control.

6- What is the adherence of both diabetic and control subjects to the recommended dietary intake and food group consumption? Is there any relation between dietary adherence and glycemic control in diabetic subjects?

Significantly more subjects with diabetes than controls reached the Australian guidelines for fat. While patients' total fat intake was very close to the recommended amount, the majority of those with diabetes consumed saturated

fatty acids at a level greater than the recommended dietary intakes. The number of controls who met the dietary guidelines for protein was significantly higher than that for diabetic subjects. Protein intake of patients, in terms of grams per kg of body weight and its contribution to total energy intake, was high. It appeared that the reduced fat intake was in part being substituted for by an increase in protein intake. It can be concluded that the patients' dietary intake compared to the Australian and American guidelines needs some modifications.

Diabetics in this study consumed a quite satisfactory amount of dietary fibre, suggesting that they may perhaps be aware of the beneficial effects of having vegetables and fruits in their diets.

The average intakes of vitamins and minerals for both diabetic and control subjects were greater than the recommended dietary intakes (RDI). Sodium intake in diabetic subjects was higher than in control subjects and the recommendations.

When the overall diet quality was scored on the basis of several dietary recommendations, it was found that there was no difference in quality of diet of patients and controls. Females in both groups had higher dietary score and among diabetic subjects the difference between two sexes was significant.

It was found that the diet score had a negative but insignificant effect in the model with HbA1c% after correcting for age, sex, insulin dose and number of insulin injections. Regression analysis indicated that in diabetic and control subjects education had significant positive association with the diet score, ie better educated people ate a "better" diet.

Significantly more diabetic subjects consumed other kinds of fruits in addition to citrus fruits. Very few people in both the diabetic and control group used legumes and nuts (as a meat alternate) on a regular basis in their diets.

Most people with diabetes had meals at regular times, and very few bought foods from takeaway outlets. Not all the patients followed a carbohydrate portion plan, although only one patient found it difficult to follow. The reasons for the lack of adherence varied. This study showed that those who comply to

their prescribed diet tended to have better diabetic control although this improvement was not statistically significant. Regression analysis showed that the frequency of dietary adherence and diet score both entered in a model with HbA1c% but their negative associations were not significant.

In general the patients' attitude toward weight control was quite correct but still some of them need more information about the importance of weight control in prevention of complications. This group of patients with type 1 diabetes should be encouraged to increase their physical exercise although there was no differences between their rate of exercise and those of controls. Those patients who do not take a carbohydrate snack before exercise should be informed about its effects.

7- What is the quality of life of patients with type 1 diabetes and how does it compare to a reference group?

The results of the Diabetes Quality of Life Measure in these patients were comparable with those found in the DCCT. This indicates that both of these groups as it was mentioned before did not have severe complications and their quality of life was probably quite good. Duration of diabetes and HbA1c% were negatively associated with the diabetes worry subscale. This shows the importance of diabetic control in achieving a better quality of life in this group of patients. In diabetic and control subjects it appeared that the most important variables which affect negatively the quality of life (SF-36 subscales) were at least one of the anthropometric measurements.

*Conclusion*

Although some of the associations like the associations between socioeconomic and diet quality with glycemic control, did not reach to the significant level ($p<0.05$) but it can be assumed that with the greater number of patients and/or follow up studies these associations could be established. In conclusion it can be said that nutritional management of these diabetic subjects, who have been in treatment for some time, could be improved. It can be assumed from this study that such improvement might reduce their risk of complications.

*Future programs and studies*

There are several limitations to the present study that should be recognized. Firstly caution should be taken in generalizing from these results, given the relatively small sample size. Larger scale studies are needed to confirm the generalisability of this study. For this purpose counting the frequency of type 1 diabetes in this area by capture-recapture method is recommended. Secondly this study was cross-sectional. Longitudinal studies are necessary if a causal relationship between variables, or between variables and diabetes outcomes, are to be demonstrated. More comprehensive studies involving assessments of other diabetes related complications like retinopathy, neuropathy and macrovascular disease would be very valuable. Despite these possible limitations this study is one of few, thus providing useful leads for future research among individuals with type 1 diabetes in this area and in Australia.

As a result of the DCCT, the American and British Diabetes Associations recommend that a major aim of the management of type 1 diabetes should be glycemic control at least equal to that achieved in the intensively treated group of the DCCT. In the DCCT patients received all their diabetes supplies (syringes, blood glucose tests and insulin) free together with free medical care. Resourcing diabetes care satisfactorily in our health care system is an unsolved problem.

A complete organized medical record system is essential for the management of every patient with diabetes to provide ongoing care of these people. At each visit the patient's progress in achieving treatment goals should be evaluated by the health care team and problems that have occurred could be reviewed. If goals are not being met the management plan needs to be revised and/or the goals need to be reassessed. Patients should be given explicit goals with feedback about how well they are doing. It has been suggested that one should send a copy of letters to the patient, or even write personally to the patient (Tattersall 1990).

Access to health care may also play a role. It is clear that high quality health care substantially reduces the risk of diabetes related complications, so that improving access to such care for vulnerable groups may be an important measure in achieving the goals. In order to achieve goals of treatment more specially trained diabetes health staff are needed to motivate and educate the patients. Patients with long experience in treatment may also be used in the education of other patients.

The need for extensive and effective diabetes education becomes apparent when the results of regression analysis are examined. Diabetic patients like other individuals also forget much of what they had originally learned and need regular refresher courses. Re-education is essential but it is rarely provided by diabetes units. In addition to education and re-education, patients with type 1 diabetes need motivation and encouragement.

In the short term teaching and evaluation may help to improve compliance and glycemic control (McCulloch et al. 1983) but in the DCCT the need for a continued medical input was emphasized for long-term motivation. Individualized nutrition recommendations and instructions preferably by a registered dietitian familiar with the components of diabetes medical nutrition therapy might be very effective. These instructions should include fat and protein modifications that are as important to patient's long term health as carbohydrates. Factual information

and technical skills can be taught relatively easily either individually or in group sessions but regular reassessment of knowledge and technical skills is vital to maintain motivation and adequate skill levels. Dietary advice must be tailored to the individual social and cultural needs of each patient. Many patients find diets based on carbohydrate exchanges or the glycemic indexes of different foods difficult to understand, and simple advice is often as effective (Albraira et al. 1980, Mitchell et al. 1990).

In general monitoring of glucose and glycated hemoglobin, lipids, blood pressure and renal status is essential to evaluate nutrition-related outcomes. Although adherence to nutrition and meal planning principles is one of the most challenging aspects of diabetes care, nutrition therapy is an essential component of successful diabetes management. If goals are not met, changes must be made in the overall diabetes care and management plan. Helping people in the coping process is an important aspect of diabetes education and one can conclude from the evidence that simple human interaction may be the best way of achieving this.

Future research needs to be directed at determining the impact of socioeconomic status on metabolic control. The adequacy of ongoing help and support may be an important but overlooked factor in overall control of the disease. In particular the attitude of the individuals in contrast with access to high quality health care should be examined to inform policy makers about how to improve outcomes for all those with type 1 diabetes.

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# Appendices

UNIVERSITY OF WOLLONGONG
Office of the Vice-Principal (Administration)

Academic & Student Services Branch

In reply please quote: DC:KM HE95/24
Further Information: Karen McRae (Ext 4457)

29 August 1995

Ms Farideh Tahbaz
Medical Research Unit

Dear Ms Tahbaz,

Thank you for your response to the Committee's requirements for your Human Research Ethics application HE95/024 "Management of diabetes by people aged 30 years or less with insulin-dependent diabetes."

Your response meets with the requirements of the Committee and your application is now formally approved.

Chairperson
Human Research Ethics Committee

cc. Professor G.D. Calvert, Supervisor

Address: Northfields Avenue Wollongong 2522 Australia
Telephone (042) 21 3555, Facsimile – Student Services: (042) 21 4322, Research/Secretariat: (042) 21 4338

**Appendix 2: Letter to the patients**

Date
<<name>>
<<address>>

Dear <<name>>

As part of the effort to improve the management of diabetes mellitus, we are about to conduct a study on the way people with type 1 diabetes in the Illawarra manage their disease. We hope to contact all younger adults (aged 18-40 initially) with this type of diabetes in the Illawarra. I obtained your name from the Diabetes Education Unit, to which you were referred. This letter is written to ask if you would take part in this study, which will give you information on your diabetes management.

The study involves an interview, in which one of our interviewers asks questions about diabetes, a questionnaire to be filled in (at home, if you wish) and, if you agree, a blood test. We want to find out about diet (what does the person with diabetes normally eat?), insulin, the degree to which diabetes is controlled ( for which diabetes is controlled (for which a blood test is needed) and factors influencing "quality of life". All this is confidential information, and no identifying information will be given to anyone without your specific consent. (We shall ask whether you would like us to send your results to a GP or medical specialist.) Neither you nor your doctor will be identified in any report arising from this study. The study is not primarily aimed at being an assessment of your diabetic control. Rather, we will use the group results to assess current management strategies throughout the Illawarra area. Your results will of course be passed on to you, as will the group results if you wish.

We are working in collaboration with a steering committee with representatives from the Illawarra Area Health Service, the IAHS Diabetes Education Unit, the NSW Health Department, the Illawarra Division of General Practice, and a local endocrinologist.

If you do not want to be part of this study I would be very pleased if you could let us know as early as possible. Please write to, or phone, my secretary, Mrs Elaine Knight, at the address below (phone 266 594). If you are happy to continue, you will be contacted by a nutritionist, Mrs Farideh Tahbaz, and she will forward further information and/or make an appointment to have these aspects of your diabetes management checked by one of our team. In order to have a good picture of current diabetic management, it is important to have input from as many people as possible, whether or not they have good diabetic control.

I believe that this is an important step in working to improve diabetes management in Australia. I hope you will be able to help.

Yours sincerely

Dennis Calvert
Professor of Medicine and Public Health

Farideh Tahbaz
PhD Student

## Appendix 3: Letter to the doctors

Date

Dear Dr

We in association with the Illawarra Area Health Service Diabetes Education Unit are currently conducting a study into the way in which younger adults (aged 18-40) with type 1 diabetes manage their diabetes with particular emphasis on their diet. We want to make this a true population-based study, ie to contact (if possible) all such people with diabetes in the community, rather than just those with problems. In this way the study will have greater generalisability and vitality. Ultimately we want to re-examine the dietary advice we give people with diabetes with a view to optimising it - and this study is designed to give us very important and necessary baseline information about what actually happens in the field.

The names and contact phone numbers and addresses of these patients with diabetes were provided by the Diabetes Education unit, to whom they were originally referred by their doctor, in confidence to me. Unfortunately, in some cases these patients are no longer at these addresses, and we have been unable to contact them to invite them to participate in our study. I am writing to ask if you can help us contact them.

A list of the names and our record of phone numbers and addresses of those patients with whom you have referred to the Diabetes Education unit and whom we can no longer contact is attached.

I would be very pleased if you could help us contact these patients - either by providing us with a contact number or address, or by asking them to contact us. to let you know what we would like to ask of them I have enclosed a sample copy of the letter and information sheet given to the patients. We would appreciate it if you could contact us as soon as possible about this problem by phone (266 594), fax (265 130) or letter, as we need to complete this study within fairly strict time limitations.

Your help will be greatly appreciated.

Yours sincerely

GD Calvert
Professor of Medicine and Public health

## Appendix 4: Letter to the controls

Date

<<name>>
<<address>>

Dear <<name>>

As part of the effort to improve the management of diabetes mellitus, we are planning a study on the kinds of diabetes treatment given to people with type 1 diabetes in the Illawarra, and on the ways they cope with diabetes. It will be the first in what is called an outcome study - how good is current treatment, and does it achieve its aims?

We are working in collaboration with a steering committee with representatives from the Illawarra Area Health Service, the IAHS Diabetes Education Unit, the NSW Health Department, the Illawarra Division of General Practice, and a local endocrinologist.

Ms/Mr..... who is your relative / neighbour / friend has nominated you to participate in this study as a case in the control group. This is of course highly confidential information; we have an ethical obligation not to reveal any information that might identify you to any unauthorized person, and we will not do so.

If you do not want to be part of this study I would be very pleased if you could let us know as early as possible. Please write to, or phone, my secretary, Mrs Elaine Knight, at the address below (phone 266 594). If you are happy to continue, you will be contacted by a nutritionist, Mrs Farideh Tahbaz, and she will forward full information including an information sheet and a form seeking your consent to the study.

I hope you will be able to help.

Yours sincerely

Dennis Calvert
Professor of Medicine and Public Health

Farideh Tahbaz
PhD Student

## Appendix 5: Message sent to recruit controls

To: All 18-40 years Australian, Illawarra Residents

A study " Assessment of type 1 diabetes management" is being carried out at the Medical Research Unit. We are currently seeking participants to be control subjects.

If you are "Australian", that is, on the Australian electoral roll, a resident of the Illawarra, aged between 18-40 years, and non-diabetic we would welcome you in this study. Your usual diet will be analysed and its nutritional content quantified. The results of all these measurements will be sent to you.

This study takes place at the Illawarra regional hospital (Wollongong Hospital), though we may be able to conduct the diet interview elsewhere.

If you are interested in taking part or would like to discuss it, please contact Mrs Farideh Tahbaz or professor Dennis Calvert at the Medical Research unit, phone 266 594, or leave a message with Mrs Elaine knight at this number.

## Appendix 6: Patient's information sheet

### INFORMATION SHEET

ASSESSMENT OF TYPE 1 DIABETES MANAGEMENT

We plan to carry out an evaluation of the way in which people with type 1 diabetes mellitus manage the diabetes. We hope as a result of this evaluation to be able to recommend ways in which management guidelines or services may be improved to provide the best possible outcomes for people with diabetes.

We have explained to you how we obtained your name, and we have reassured you that this information we discover about you, is confidential and will not be released to anybody, unless you give us specific consent to pass information to your doctor. Any other information about this study that is published or passed to other bodies (for instance, the NSW Health Department) will be in such a form that no individuals can be identified. We shall, of course, send you a copy of your results, and (if you wish) the group results when they are available.

We will ask if we can interview you. Interviews will be conducted by Mrs Farideh Tahbaz, who is a nutritionist with a Master degree in nutrition or a graduate in nutrition who is studying for a Master degree. Mrs Tahbaz, or a colleague will give you a standard questionnaire to fill out, which contains information on your own circumstances, on the way you manage your diabetes, on the way in which insulin is prescribed, and on the way you feel you manage your diabetes and your reactions to diabetes.

You will be asked if you can give a blood and urine specimen, to check the degree to which your diabetes is controlled, and have your height and weight and degree of fatness estimated. Blood would normally be taken from a vein in the arm. You will be asked for further information on the details of your usual diet.

It should be clear that there are no right or wrong answers on diet or diabetes management; we wish to obtain an accurate picture of current management, in its diversity, in the Illawarra.

Please feel free to ask Mrs Tahbaz any questions that occur to you. We will ask you if we can write to your doctor and let him/her know the results of your blood test and if you wish, the dietary analysis.

If there are any outstanding questions, please ring Professor Dennis Calvert phone 266 594. If you have any queries regarding the conduct of the research, please contact the Secretary of the Human Research Ethics Committee on 214 457.

**Appendix 7: Control's information sheet**

## INFORMATION SHEET

### ASSESSMENT OF TYPE 1 DIABETES MANAGEMENT

We plan to carry out an evaluation of the way in which people with type 1 diabetes mellitus manage the diabetes. We hope as a result of this evaluation to be able to recommend ways in which management guidelines or services may be improved to provide the best possible outcomes for people with diabetes.

We have explained to you how we obtained your name, and we have reassured you that this information we discover about you, is confidential and will not be released to anybody, unless you want to know about your dietary intake and physical fitness. Any other information about this study that is published or passed to other bodies (for instance, the NSW Health Department) will be in such a form that no individuals can be identified.

We will ask if we can interview you. Interviews will be conducted by Mrs Farideh Tahbaz, who is a nutritionist with a Master degree in nutrition or a graduate in nutrition who is studying for a Master degree. Mrs Tahbaz, or a colleague will give you a standard questionnaire to fill out, which contains information on your own circumstances. They will have an interview with you to get your diet history and estimate your estimate your height, weight and the degree of fatness.

You will be asked if you can give blood and urine specimen. Blood would normally be taken from a vein in the arm.

Please feel free to ask Mrs Tahbaz any questions that occur to you. We shall, of course, send you a copy of your results if you wish.

If there are any outstanding questions, please ring Professor Dennis Calvert phone 266 594. If you have any queries regarding the conduct of the research, please contact the Secretary of the Human Research Ethics Committee on 214 457.

**Appendix 8: Patient's consent form**

## CONSENT FORM

FOR PARTICIPANTS WITH DIABETES

ASSESSMENT OF TYPE 1 DIABETES MANAGEMENT

This research on the current management of diabetes in the Illawarra is being conducted by a group of clinicians and scientists supported by a steering committee with representatives from the Illawarra Area Health Service, the NSW Health Department, and the medical profession. Professor Dennis Calvert in the Medical Research Unit (Illawarra Area Health Service/University of Wollongong) heads the group, and Mrs Farideh Tahbaz is coordinating.

Information relating to this study is detailed in the attached information sheet.

You are free to withdraw from all or part of this research program at any time without penalty, and without compromising in any way your treatment or access to services.

The ethical aspets of this study have been approved by the University of Wollongong Human Research Ethics Committee, which is responsible for the ethical aspects of research involving people in the Illawarra. If you have any enquiries regarding the conduct of the research please contact the Secretary of the University of Wollongong Human Research Ethics Committee on 214 457.

**********

I understand that the information collected in this research will be used for the assessment of type 1 diabetes management and I consent for the data to be used in that manner.

If you wish to take part in this research please sign below

| ..... | ..... | ...../...../..... |
|---|---|---|
| Name | Signature | Date |

**Appendix 9: Control's consent form**

## CONSENT FORM

FOR PARTICIPANTS WITHOUT DIABETES

ASSESSMENT OF TYPE 1 DIABETES MANAGEMENT

This research on the current management of diabetes in the Illawarra is being conducted by a group of clinicians and scientists supported by a steering committee with representatives from the Illawarra Area Health Service, the NSW Health Department, and the medical profession. Professor Dennis Calvert in the Medical Research Unit (Illawarra Area Health Service/University of Wollongong) heads the group, and Mrs Farideh Tahbaz is coordinating.

Information relating to this study is detailed in the attached information sheet.

You are free to withdraw from all or part of this research program at any time without penalty.

The ethical aspects of this study have been approved by the University of Wollongong Human Research Ethics Committee, which is responsible for the ethical aspects of research involving people in the Illawarra. If you have any enquiries regarding the conduct of the research please contact the Secretary of the University of Wollongong Human Research Ethics Committee on 214 457.

**********

I understand that the information collected in this research will be used for the assessment of type 1 diabetes management and I consent for the data to be used in that manner.

If you wish to take part in this research please sign below

| ..... | ..... | ...../...../..... |
|---|---|---|
| Name | Signature | Date |

**UNIVERSITY OF WOLLONGONG**

**MEDICAL RESEARCH UNIT**

**TYPE 1 DIABETES STUDY**

Please indicate your answer by ticking the appropriate box ❑ or by writing your answer in the space provided. If you are uncertain about the answer to any of the questions leave them blank and ask the receptionist to help you.

Office use only

**Characteristics of the subject:**

1. Sex: Female ❑1 / Male ❑2 — ❑ 1

2. Marital Status:

| | | |
|---|---|---|
| Single | ❑1 | ❑ 2 |
| Married | ❑2 | |
| Separated/Divorced | ❑3 | |
| Widowed | ❑4 | |

3. Date of Birth: Day: ❑❑ Month: ❑❑ Year: 19❑❑ — ❑ 3

4. Country of Birth: Australia ❑1 / Not Australia ❑2 — ❑ 4

If not Australia, what is your country of birth? ----------

5. How long have you been resident in Australia? Months ❑ Years ❑ — ❑ 5

6. Where were members of your family born?

- Your father ---------- ❑ 6
- Your father's father (paternal grandfather) ---------- ❑ 7
- Your father's mother (paternal grandmother) ---------- ❑ 8
- Your mother ---------- ❑ 9
- Your mother's father (maternal grandfather) ❑ 10
- Your mother's mother (maternal grandmother) ❑ 11

Office use only

7. Are you of Aboriginal or Torres Strait Islander origin?
(If of mixed origin indicate the one to which you belong)

| | | |
|---|---|---|
| No | ❑1 | ☐ 12 |
| Yes, Aboriginal | ❑2 | |
| Yes, Torres Strait Islander | ❑3 | |

**Appendix 11: Diabetes and medical history**

**DIABETES HISTORY:**

1.What date was diabetes diagnosed? Mo❑/Yr❑❑ ☐ 13

2. What is the name and address of your doctor who normally treats your diabetes? ☐ 14

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---

3. Do you want us to send any results to your doctor (eg. diet and blood test results)?

| | | |
|---|---|---|
| No | ❑1 | ☐ 15 |
| Yes | ❑2 | |

4. Have you ever taken oral drugs (tablets) for diabetes?

| | | |
|---|---|---|
| No | ❑1 | ☐ 16 |
| Yes | ❑2 | |

a. If yes, are you currently taking oral drugs (tablets)?

| | | |
|---|---|---|
| No | ❑1 | ☐ 17 |
| Yes | ❑2 | |

b. If no, how long ago did you stop taking oral drugs (tablets)?

| | | |
|---|---|---|
| Mo ❑ Yr | ❑❑1 | ☐ 18 |
| Unknown | ❑2 | |

5. Are you currently taking insulin?

| | | |
|---|---|---|
| No | ❑1 | ☐ 19 |
| Yes | ❑2 | |

6. When did you begin permanent use of insulin?

| | | |
|---|---|---|
| Mo ❑ Yr | ❑❑1 | ☐ 20 |
| Unknown | ❑2 | |

Office use Only

7. What is your current total daily dose of insulin: ----- ------ units ☐ 21

8. Are you currently taking oral drugs and insulin?

No ❑1
Yes ❑2 ☐ 22

If yes to #5 or #8, what is your current insulin regimen? (answer one)

| | | | |
|---|---|---|---|
| one injection daily | ❑1 | pump❑4 | ☐ |
| two injections daily | ❑2 | other ❑5 | 23 |
| three or more injections daily | ❑3 | Specify:---------- | |

9. Have you ever been hospitalized for diabetes ketoacidosis?

No ❑1
Yes ❑2
Unknown ❑3 ☐ 24

**MEDICAL HISTORY:**

A. Eye problems:

Have you ever been told by a health care professional that you have or had:

1. Any diabetes related eye problems?

No ❑1
Yes ❑2 ☐ 25
Unknown ❑3

If yes please specify: _____

2. Laser treatment?

No ❑1
Yes ❑2 ☐ 26
Unknown ❑3

3. Impairment of vision?

No ❑1
Yes ❑2 ☐ 27
Unknown ❑3

Office use only

4. Cataracts?

No ❑1
Yes ❑2
Unknown ❑3

☐ 28

5. Detached retina?

No ❑1
Yes ❑2
Unknown ❑3

☐ 29

B. Kidney problems:

Have you ever been told by a health care professional that you have or had:

1. Diabetic kidney problem?

No ❑1
Yes ❑2
Unknown ❑3

☐ 30

2. Protein or albumin in the urine?

No ❑1
Yes ❑2
Unknown ❑3

☐ 31

Have you ever had:

3. Kidney transplant?

No ❑1
Yes ❑2
Unknown ❑3

☐ 32

4. Kidney dialysis?

No ❑1
Yes ❑2
Unknown ❑3

☐ 33

C. Cardiovascular (heart or circulation) problems:

Have you ever been told by a health care professional that you have or had:

Office use only

1. Any problems with heart or blood vessels?

No ❑1
Yes ❑2
Unknown ❑3

☐ 34

If yes, please specify: ----------

2. Abnormal Electrocardiogram?

No ❑1
Yes ❑2
Unknown ❑3

☐ 35

Have you ever had:

3. Heart pains or angina?

No ❑1
Yes ❑2
Unknown ❑3

☐ 36

4. Heart attack?

No ❑1
Yes ❑2
Unknown ❑3

☐ 37

5. Coronary bypass surgery?

No ❑1
Yes ❑2
Unknown ❑3

☐ 38

6. Stroke?

No ❑1
Yes ❑2
Unknown ❑3

☐ 39

7. High blood pressure?

No ❑1
Yes ❑2
Unknown ❑3

☐ 40

Office use only

8. Drug treatment for high blood pressure?

No ❑1
Yes ❑2
Unknown ❑3

☐ 41

If yes, are you currently receiving drug treatment?

No ❑1
Yes ❑2
Unknown ❑3

☐ 42

D. Peripheral vascular complications:

Have you ever been told by a health care professional that you have or had:

1. Any trouble with circulation in legs?

No ❑1
Yes ❑2
Unknown ❑3

☐ 43

2. Foot ulcers?

No ❑1
Yes ❑2
Unknown ❑3

☐ 44

3.Gangrene?

No ❑1
Yes ❑2
Unknown ❑3

☐ 45

Have you ever had:

4. Non-traumatic amputation?

No ❑1
Yes ❑2
Unknown ❑3

☐ 46

Office use only

E. Other major medical disease?

1. Do you have any serious medical problems not mentioned yet?

No ❑1
Yes ❑2
Unknown ❑3

☐ 47

Specify: ----------

F. Are there any people with diabetes in your family?

☐ 48

No ❑1
Yes ❑2

If yes what is his/her relation with you? ----------

**Information on your background:**

Office use only

1. **Education**

What is the highest level of your education?
(Please tick and complete level if appropriate)

| | | |
|---|---|---|
| commenced primary school | ❑ | ☐ |
| finished primary school | ❑ | 49 |
| commenced high school | ❑ | |
| finished high school | ❑ level ---- | |
| university or other tertiary schooling (eg. TAFE) started | ❑ | |
| university or other tertiary schooling (eg. TAFE) finished | ❑ level ---- | |

2. **Economic data:**

2.1 What is the total estimated family income before taxes?

| | | |
|---|---|---|
| less than $12000 | ❑1 | ☐ |
| $12000 -$15000 | ❑2 | 50 |
| $15001 -$18000 | ❑3 | |
| $18001 -$22000 | ❑4 | |
| $22001 -$26000 | ❑5 | |
| $26001 -$32000 | ❑6 | |
| $32001 -$40000 | ❑7 | |
| $40001 -$50000 | ❑8 | |
| $50001 and over | ❑9 | |

2.2 Occupation

What is your current occupation (if applicable)? ---------- ☐ 51

Do you want a summary of the study results when available ?

No ❑ ☐
Yes ❑ 52

Contact address (to send you a summary of the results if you wish, and for future follow up): ☐ 53

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Tel:----------

# FOOD PATTERN QUESTIONNAIRE JULY 1995

**Diabetes Control and Complications Trial (DCCT) - Australian Version***

This questionnaire asks general questions about your food choices and eating habits. Answer as best you can. If you have any questions about the form you can ask the researcher. More information will be collected during the clinic visit.

Thank you for your co-operation in providing this information.

1. Has your general pattern of eating changed in the last year?

☐ yes ☐ no If yes, describe:

_____

_____

_____

2. Are you or have you in the past year been on any special diet in addition to a diabetic diet? (such as low salt, vegetarian, weight loss etc).

☐ yes ☐ no If yes, describe this diet:

_____

_____

_____

3. Are you currently increasing or decreasing your intake of any particular foods or beverages (such as foods high in fibre, caffeine, alcohol etc)?

☐ yes ☐ no If yes, describe:

4. Does your meal pattern tend to vary from week to week? (due to shift work, sports activities, weekends etc).

☐ yes ☐ no If yes, describe:

5. In the last year, have you taken any vitamin and/or mineral supplements?

☐ yes ☐ no If yes, specify brand name, amount and how often taken

6. Do you change your meal pattern/insulin routine when you exercise? (e.g do you eat additional carbohydrate before exercise or change your insulin dose etc...)

☐ yes ☐ no If yes, describe how:

_____

_____

_____

_____

7. How do you treat hypos ( low blood sugar)?

List food/beverages and amounts consumed:

_____

_____

_____

_____

8. Do you use sugar or an artificial sweetener?

☐ yes ☐ no

If yes, specify which foods/beverages you add it to (such as cereal, fruit, coffee, tea, other):

_____

_____

_____

If you use an artificial sweetener, specify brand name:

_____

9. Do you add salt to your food at the table?

☐ always ☐ occasionally ☐ never, Go to Q11

10. How would you rate the amount of salt you add?

☐ light ☐ moderate ☐ heavy

11. Do you use a salt substitute at the table such as Lite, Co-salt, No-salt etc?

☐ always ☐ occasionally ☐ never

If used, specify brand name: _____

_____

12. Do you regularly use other salt seasonings at the table such as Chicken salt, onion salt, garlic salt?

☐ yes ☐ no

Specify kind(s):

_____

_____

_____

13. Indicate below your usual meal and snack patterns:

For each meal state the usual time you eat it, for example breakfast at 7:30am and then state the number of times a week you would eat it at home, take from home etc.. Repeat this for each meal time.

| | Usual Time of Meal | Eat at Home | Take from Home | Buy from Takeaway Outlet - Cafeteria, Cafe / Restaurant | Do not Eat | Comments |
|---|---|---|---|---|---|---|
| Morning meal (Breakfast) | | | | | | |
| Morning snack | | | | | | |
| Noon meal (Lunch) | | | | | | |
| Afternoon snack | | | | | | |
| Evening meal (Dinner) | | | | | | |
| Evening snack (Supper) | | | | | | |
| Additional snack | | | | | | |

14. Who prepares most of your home-cooked meals?

| Self | Parent | Spouse | Other Household Member |
|---|---|---|---|
| ☐ | ☐ | ☐ | ☐ |

Please estimate how often you eat the following foods by ticking the appropriate box. Include diet foods and other special products in the general food categories. For example include low calorie beer with beer. If they are diet/special products please indicate this in the comments section. You may also use the Comments Section for details such as seasonal variation or the brand/product name. Feel free to use the bottom of each page for any additional comments.

| **BEVERAGES** | **Daily** | **4-6 times a week** | **1-3 times a week** | **1-3 times a month** | **1-3 times a year or never** | **Comments eg seasonal variation, low fat, product name etc...** |
|---|---|---|---|---|---|---|
| Coffee-regular or decaffeinated | | | | | | |
| Coffee substitute (eg Ecco, Caro) | | | | | | |
| Tea-regular, decaf, herbal | | | | | | |
| Drinking chocolate, Milo, Ovaltine etc | | | | | | |
| Beer, ale | | | | | | |
| Spirits, cocktails | | | | | | |
| Liqueur, Port, Brandy | | | | | | |
| Wine, dry or sweet | | | | | | |
| Soft drinks- cola and non-cola | | | | | | |
| Diet soft drinks-cola and non-cola | | | | | | |
| Cordial (regular or low joule) | | | | | | |
| **DAIRY PRODUCTS** | | | | | | |
| Milk-whole, skim, reduced fat, powdered UHT, buttermilk, etc | | | | | | |
| Cottage/ricotta cheese | | | | | | |
| Cheese- block, slice, cheese spread | | | | | | |
| Yoghurt, plain | | | | | | |
| Yoghurt, sweetened | | | | | | |
| Sour cream, dips | | | | | | |
| Ice cream regular | | | | | | |
| Ice confectionary/low calorie ice cream | | | | | | |

| **DAIRY PRODUCTS (continued)** | **Daily** | **4-6 times a week** | **1-3 times a week** | **1-3 times a month** | **1-3 times a year or never** | **Comments eg seasonal variation, low fat, product name etc...** |
|---|---|---|---|---|---|---|
| Milk shakes, smoothies | | | | | | |
| Eggs | | | | | | |
| Egg substitutes (eg Scramblers) | | | | | | |
| **BREADS & CEREALS** | | | | | | |
| Bread and rolls-white | | | | | | |
| Bread and rolls-wholemeal, mixed grain | | | | | | |
| Fruit loaf/raisin bread | | | | | | |
| Plain Sweet Biscuits | | | | | | |
| Fancy Biscuits (eg cream, choc-coated etc) | | | | | | |
| Bagels, English muffins, crumpets | | | | | | |
| Sweet bun, Danish, doughnuts | | | | | | |
| Pancakes, pikelets, waffles | | | | | | |
| Cereals-Porridge/Oatmeal | | | | | | |
| Muesli | | | | | | |
| Other Breakfast Cereals | | | | | | |
| Pasta, Noodles | | | | | | |
| Rice-brown, white, rice mixes | | | | | | |
| Crackers/Crispbreads | | | | | | |
| Popcorn | | | | | | |
| Chips-potato, corn etc | | | | | | |
| Muesli/Health bars | | | | | | |
| **DESSERTS** | | | | | | |
| Puddings, custards | | | | | | |
| Bars, slices | | | | | | |
| Cakes | | | | | | |

| **DESSERTS (continued)** | **Daily** | **4-6 times a week** | **1-3 times a week** | **1-3 times a month** | **1-3 times a year or never** | **Comments e.g seasonal variation, low fat, product name etc...** |
|---|---|---|---|---|---|---|
| Pies, fruit crumbles | | | | | | |
| Gelatine desserts - Jelly etc | | | | | | |
| Other, specify: | | | | | | |
| | | | | | | |
| **MEAT, POULTRY, FISH** | | | | | | |
| Pork | | | | | | |
| Lamb, Veal | | | | | | |
| Beef | | | | | | |
| Sausages / Continental Sausages | | | | | | |
| Bacon | | | | | | |
| Frankfurts, Saveloys | | | | | | |
| Luncheon meats- ham, devon, salami , corned beef etc | | | | | | |
| Variety / Organ meats- liver, tongue, kidney etc | | | | | | |
| Chicken, turkey | | | | | | |
| Duck, quail | | | | | | |
| Fish, fresh or frozen- perch, salmon, hake, cod, sole etc | | | | | | |
| Shellfish, fresh or canned - lobster, prawn, crab, mussels, scallops etc | | | | | | |
| **MEAT SUBSTITUTES** | | | | | | |
| Peanut butter | | | | | | |
| Nuts or seeds | | | | | | |
| Canned or dried beans, lentils, split peas, lima beans, baked beans | | | | | | |
| Soy protein foods such as tofu | | | | | | |

| MIXED DISHES, SOUPS | Daily | 4-6 times a week | 1-3 times a week | 1-3 times a month | 1-3 times a year or never | Comments e.g seasonal variation, low fat, product name etc... |
|---|---|---|---|---|---|---|
| Pizza, lasagne, macaroni & cheese, ravioli, spaghetti bolognaise etc | | | | | | |
| Tacos, enchiladas, burritos, chilli etc | | | | | | |
| Hamburger | | | | | | |
| Stews/Casseroles/ Curry/Goulash | | | | | | |
| Meat Loaf | | | | | | |
| Quiche, souffle | | | | | | |
| Stir fry meat and vegetable dishes | | | | | | |
| TV/frozen dinners eg McCain, Findus | | | | | | |
| Soups, including cream soups, chowders | | | | | | |
| Sausage Roll, Pastie, Meat Pie | | | | | | |
| Canned meals eg Heinz, Kraft beef and vegetables | | | | | | |
| Other mixed dishes commonly eaten Specify: | | | | | | |
| | | | | | | |
| VEGETABLES | | | | | | |
| Potatoes-baked, boiled, mashed, hot chips etc | | | | | | |
| Sweet potatoes | | | | | | |
| Green vegetables-peas, broccoli, spinach, beans, cabbage etc | | | | | | |
| Other cooked vegetables-pumpkin, , carrots, corn etc | | | | | | |

| **VEGETABLES (continued)** | **Daily** | **4-6 times a week** | **1-3 times a week** | **1-3 times a month** | **1-3 times a year or never** | **Comments e.g seasonal variation, low fat, product name etc...** |
|---|---|---|---|---|---|---|
| Salads, raw vegetables | | | | | | |
| Vegetable juices-V8, tomato juice | | | | | | |
| **FRUIT AND FRUIT JUICES** | | | | | | |
| Fruit juice | | | | | | |
| Fruit-flavoured drinks-Tang etc | | | | | | |
| Citrus fruits-oranges, grapefruits | | | | | | |
| Canned fruits in natural juice/water | | | | | | |
| Dried fruits-raisins, dates, prunes, apricots etc | | | | | | |
| Avocado | | | | | | |
| **SUGAR-FREE PRODUCTS** | | | | | | |
| Artificial sweeteners | | | | | | |
| Lollies, chewing gum | | | | | | |
| Chocolate | | | | | | |
| Syrups, jams | | | | | | |
| Ice cream | | | | | | |
| Biscuits, cake | | | | | | |
| Jelly | | | | | | |
| Puddings, custards | | | | | | |
| **MISCELLANEOUS** | | | | | | |
| Soy milk | | | | | | |
| Vegemite/marmite | | | | | | |
| Fish paste | | | | | | |
| Pickles, relish, chutneys | | | | | | |
| Olives | | | | | | |

| **MISCELLANEOUS (continued)** | **Daily** | **4-6 times a week** | **1-3 times a week** | **1-3 times a month** | **1-3 times a year or never** | **Comments e.g seasonal variation, low fat, product name etc...** |
|---|---|---|---|---|---|---|
| Steak sauces, mustard | | | | | | |
| Tomato sauce, chilli sauce | | | | | | |
| Soy sauce, teriyaki sauce | | \ | | | | |
| Confectionary, gum, cough lozenges | | | | | | |
| Spreads- jam, honey, syrup, marmalade | | | | | | |
| Chocolate bars | | | | | | |
| **DIETARY SUPPLEMENTS** | | | | | | |
| Vitamins and/or minerals | | | | | | |
| Bran | | | | | | |
| Wheat germ | | | | | | |
| Malt | | | | | | |
| Other supplements Specify: | | | | | | |
| | | | | | | |
| **OTHER COMMONLY CONSUMED FOODS OR BEVERAGES NOT INCLUDED IN PREVIOUS GROUPS** Specify: | | | | | | |
| | | | | | | |
| | | | | | | |

**Prepared by: Effie Tsivis, Dietitian, July 1995.**

*ADAPTED FROM THE FOOD PATTERN QUESTIONNAIRE
DEVELOPED BY:
The Nutrition Coordinating Centre
2829 University Avenue SW
MINNEAPOLIS, MN 55414

# FOOD PREPARATION QUESTIONNAIRE JULY 1995

**Diabetes Control and Complications Trial (DCCT) - Australian Version***
This questionnaire is to be completed by the person who usually prepares the food in your home. If this is not possible then please fill the form out as best you can.
This questionnaire is important for analysis of the dietary component of the study. If you have any questions about the form you can ask the researcher

Thank you for your co-operation

1. What relationship are you to the participant?

☐ self ☐ parent ☐ spouse ☐ other, specify _____

2. If the following foods are prepared at home, tick the type of sweetener usually used:

| | Sugar Added | Artificial Sweetener Added (give name) | Other eg honey | None Added |
|---|---|---|---|---|
| Fruit juices | | | | |
| Fresh fruit | | | | |
| Canned fruit | | | | |
| Beverages (tea, coffee, milk drinks etc | | | | |
| Baked goods (cakes, biscuits etc) | | | | |
| Other (specify) | | | | |
| | | | | |
| | | | | |
| | | | | |

3. Tick whether salt or a salt substitute is usually added in preparing the following foods:

| | salt | salt substitute | seasoning salts | none added |
|---|---|---|---|---|
| Pasta, such as noodles, spaghetti, etc | ☐ | ☐ | ☐ | ☐ |
| Rice | ☐ | ☐ | ☐ | ☐ |
| Potatoes, hot chips | ☐ | ☐ | ☐ | ☐ |
| Other Vegetables | ☐ | ☐ | ☐ | ☐ |
| Meat | ☐ | ☐ | ☐ | ☐ |
| Eggs | ☐ | ☐ | ☐ | ☐ |
| Other: Specify _____ | ☐ | ☐ | ☐ | ☐ |
| _____ | ☐ | ☐ | ☐ | ☐ |

If salt substitute, specify kind/brand: .....

4. Are the following cooking oils/fats and spreads used (please tick):

a) Butter ☐ yes Specify: ☐ regular
☐ salt reduced
☐ no
☐ diet/reduced fat

b) Margarine ☐ yes Specify: ☐ regular
☐ salt reduced
☐ no
☐ diet/reduced fat

Specify the brand(s) of butter/
margarine used:

_____

_____

_____

c) Vegetable oil (such as olive, canola, safflower, sunflower etc)

☐ yes — Specify types and/or brands used:

☐ no _____

_____

d) Oil Sprays (eg Pure and Simple, Golden Canola etc)

☐ yes — Specify brand:

☐ no _____

_____

e) Solid oils/fats (eg Frymaster, Fairy, Copha, Tulip etc)

☐ yes — Specify types and/or brands used:

☐ no _____

_____

f) Other cooking fats (such as lard, ghee, beef dripping etc)

☐ yes — Specify types and/or brands used:

☐ no _____

_____

5. Tick the type of oil/fat most often used in preparing each of the following foods:

| | BUTTER | MARGARINE | OIL SPRAYS | VEGETABLE OIL | SOLID VEGETABLE FATS eg tulip, frymaster | SOLID ANIMAL FATS eg Ghee, Lard etc | NONE ADDED | DON'T COOK/EAT |
|---|---|---|---|---|---|---|---|---|
| Eggs, fried | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Eggs, scrambled | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Toast/Sandwiches | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Potatoes, mashed | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Potatoes, baked | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Hot Chips | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Green Vegetables | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Other Vegetables | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Beans, lentils | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Gravy | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Sauces eg white, mushroom | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| Pastry | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

6. Indicate the most usual method of preparing each of the following. If you fry any of them, comment on whether the item is dipped in flour or batter or crumbed before frying and what oil/fat is used for frying. Also tick whether gravy or sauce is prepared. If sauce is prepared state what type it is (e.g Maggi satay packet mix, Masterfoods chilli sauce, homemade etc...)

| ITEM | METHOD OF COOKING (eg baking, grilling, pan fry, deep fry etc) | KIND OF FAT USED (if any) | GRAVY or SAUCE | | IF SAUCE, SPECIFY TYPE |
|---|---|---|---|---|---|
| | | | YES | NO | |
| Hamburger | | | | | |
| Steaks | | | | | |
| Chops | | | | | |
| Chicken | | | | | |
| Fish | | | | | |
| Shellfish (prawn, lobster etc) | | | | | |
| Liver, kidney etc | | | | | |
| Other, specify | | | | | |
| | | | | | |
| | | | | | |
| | | | | | |

7. If you prepare gravies, do you usually use:

☐ cornflour ☐ flour ☐ packet mix eg Gravox

Is the liquid usually:

☐ milk ☐ water ☐ other, specify _____

8. Tick how much fat is usually trimmed from the meat before cooking or eating:

☐ trim most or all ☐ trim some ☐ usually don't trim

9. Tick the type of salad dressing <u>most often</u> used with the following salads (specify brand(s) where possible):

| | **Regular mayonnaise such as Praise, Kraft** | **Reduced fat mayonnaise such as Kraft Light, Weight Watchers** | **Oil free dressing such as Praise No Oil, Fountain No Oil, Kraft Free** | **Other- specify type e.g , Italian, Thousand Island etc.- OR Homemade (list ingredients)** | **Don't use salad dressing** |
|---|---|---|---|---|---|
| Potato salad | | | | | |
| Coleslaw | | | | | |
| Tossed salad | | | | | |
| Pasta salad | | | | | |
| Other, specify | | | | | |
| | | | | | |
| | | | | | |
| | | | | | |

**Prepared by Effie Tsivis, Dietitian, July 1995.**

ADAPTED FROM THE FOOD PREPARATION QUESTIONNAIRE
DEVELOPED BY:
The Nutrition Coordinating Centre
2829 University Avenue SW
MINNEAPOLIS, MN 55414

## Appendix 15: Practical Aspects of Type 1 Diabetes

For the following questions please tick the response that best applies to yourself

DIETARY ADHERENCE

In Questions 1 - 3, we want to find out about your adherence to a diabetic diet, and the difficulties that you may experience keeping to a diabetic diet.

Office use only

1. In general, how often do you routinely follow a carbohydrate portion plan on a typical day ? For example do you have a pattern of carbohydrate "portions" you follow over the day, such as 3 portions for breakfast, 2 portions for morning tea, 4 for lunch, etc.

287 ☐

I follow my carbohydrate portion plan:

| | | |
|---|---|---|
| Always | (7 days a week) | 1☐ |
| Usually | (5-6 days a week) | 2☐ |
| Sometimes | (3-4 days a week) | 3☐ |
| Not very often | (1-2 days a week) | 4☐ |
| No | (0 days a week) | 5☐ |
| Don't Know | | 6☐ |

2. We would like to know what specific factors prevent you from routinely following a carbohydrate "portion" meal plan or from following it as often as you might otherwise. You may tick more than one response or write your own down on the space provided.

288 ☐

It didn't give me good blood sugar control when

| | |
|---|---|
| I tried it before | 1 ☐ |
| I am tired of following a set plan | 2 ☐ |
| My work is too hectic | 3 ☐ |
| My family life makes it difficult | 4 ☐ |
| Family/friends are not supportive enough | 5 ☐ |
| I crave food I shouldn't eat | 6 ☐ |
| Other. Please Specify: | 7 ☐ |

_____

_____

3. I generally find it....

289 ☐

| | |
|---|---|
| Very difficult | 1 ☐ |
| Moderately difficult | 2 ☐ |
| Neither difficult or easy | 3 ☐ |
| Moderately easy | 4 ☐ |
| Very easy | 5 ☐ |

to adhere to my diabetic diet

WEIGHT CONTROL

In Questions 4 - 7 we want to find out about your weight maintenance

Office use only

4. Are you currently trying to reduce your weight (please indicate) 290 ☐
   No 1 ☐
   Yes 2 ☐

   If yes what measures are you taking?

   _____

   _____

5. Are you trying to maintain your current weight? (please indicate) 291 ☐
   No 1 ☐
   Yes 2 ☐

   If yes what measures are you taking?

   _____

   _____

6. Are you currently trying to increase your weight? (please indicate) 292 ☐
   No 1 ☐
   Yes 2 ☐

   If yes what measures are you taking?

   _____

   _____

7. Please indicate what you think is your ideal goal weight: _____kg 293 ☐

## ALCOHOL INTAKE

In Questions 8-9 we want to find out about the amount of alcohol you drink

Office use only

8. How often do you usually drink alcohol?

| | |
|---|---|
| I don't drink alcohol | ☐ |
| Less than once a week | ☐ |
| On 1 or 2 days a week | ☐ |
| On 3 or 4 days a week | ☐ |
| On 5 or 6 days a week | ☐ |
| Every day | ☐ |

294 ☐

9. On a day when you drink alcohol, how many drinks do you usually have?

| | |
|---|---|
| 1 or 2 drinks | ☐ |
| 3 or 4 drinks | ☐ |
| 5 to 8 drinks | ☐ |
| 9 to 12 drinks | ☐ |
| 13 to 20 drinks | ☐ |
| more than 20 drinks | ☐ |

295 ☐

EXERCISE

In questions 9-12, we want to find out about the exercise you had during the PAST 2 WEEKS

* For recreation, sport or health-fitness purposes
* As part of your tasks at work and around the house

Please distinguish between vigorous and exercise which made you breathe harder or puff and pant, and less vigorous exercise

Office use only _____

## RECREATION, SPORT OR HEALTH-FITNESS

9. In the PAST 2 WEEKS, did you engage in vigorous exercise - exercise which makes you breathe harder or puff or pant? (eg vigorous sports such as football, netball, tennis, squash, athletics: jogging or running: keep fit exercises: vigorous swimming: etc.) 296 ☐ 297 ☐

| | |
|---|---|
| No | 1 ☐ |
| Yes | 2 ☐ |

298 ☐

If yes, how many sessions of vigorous exercise did you have over the 2 week period? _____

Please estimate the TOTAL TIME spent exercising vigorously during the PAST 2 WEEKS. 299 ☐

_____ hours _____ minutes

10. In the PAST 2 WEEKS, did you engage in less vigorous exercise for recreation, sport or health-fitness purposes which did not make you breathe harder or puff and pant? 300 ☐

| | |
|---|---|
| No | 1 ☐ |
| Yes | 2 ☐ |

301 ☐

If yes, how many sessions of less vigorous exercise did you have over the 2 week period? _____

Please estimate the TOTAL TIME spent exercising less vigorously each week. 302 ☐

_____ hours _____ minutes

303 ☐

11. In the PAST 2 WEEKS, did you walk for recreation or exercise for periods of 20 minutes or longer?

| | |
|---|---|
| No | 1 ☐ |
| Yes | 2 ☐ |

If yes, how many times? _____

## VIGOROUS TASKS AT WORK AND AROUND THE HOUSE (paid or unpaid work)

Office use only

12. In the PAST 2 WEEKS, did you engage in vigorous activity, apart from exercise, which makes you breathe harder or puff and pant? (eg carrying loads, heavy gardening, chopping wood, labouring - at home, during employment or anywhere else).

304 ☐

| | |
|---|---|
| No | 1 ☐ |
| Yes | 2 ☐ |

305 ☐

If yes, how many sessions of these types of vigorous activity did you have over the 2 week period?_____

306 ☐

Please estimate the TOTAL TIME spent in these types of vigorous activity during the past 2 weeks: _____ hours_____minutes

Thank you for taking time to complete these questions ☺

## Diabetes Quality of Life Measure

Please read each statement carefully. Please indicate how satisfied or dissatisfied you currently are with the aspect of your life described in the statement. Circle the number that best describes how you feel. There are no right or wrong answers to these questions. We are interested in your opinion.

| | Very Satisfied | Moderately Satisfied | Neither | Moderately Dissatisfied | Very Dissatisfied |
|---|---|---|---|---|---|
| A1. How satisfied are you with the amount of time it takes to manage your diabetes? | 1 | 2 | 3 | 4 | 5 |
| A2. How satisfied are you with the amount of time you spend getting checkups? | 1 | 2 | 3 | 4 | 5 |
| A3. How satisfied are you with the time it takes to determine your sugar level? | 1 | 2 | 3 | 4 | 5 |
| A4. How satisfied are you with your current treatment? | 1 | 2 | 3 | 4 | 5 |
| A5. How satisfied are you with the flexibility you have in your diet? | 1 | 2 | 3 | 4 | 5 |
| A6. How satisfied are you with the burden your diabetes is placing on your family? | 1 | 2 | 3 | 4 | 5 |
| A7. How satisfied are you with your knowledge about your diabetes? | 1 | 2 | 3 | 4 | 5 |
| A8. How satisfied are you with your sleep? | 1 | 2 | 3 | 4 | 5 |
| A9.How satisfied are you with your social relationships and friendships? | 1 | 2 | 3 | 4 | 5 |
| A10. How satisfied are you with your sex life? | 1 | 2 | 3 | 4 | 5 |

| | Very Satisfied | Moderately Satisfied | Neither | Moderately Dissatisfied | Very Dissatisfied |
|---|---|---|---|---|---|
| A11. How satisfied are you with your work, school, and household activities? | 1 | 2 | 3 | 4 | 5 |
| A12. How satisfied are you with the appearance of your body? | 1 | 2 | 3 | 4 | 5 |
| A13. How satisfied are you with the time you spend exercising? | 1 | 2 | 3 | 4 | 5 |
| A14. How satisfied are you with your leisure time? | 1 | 2 | 3 | 4 | 5 |
| A15. How satisfied are you with with life in general? | 1 | 2 | 3 | 4 | 5 |

Please indicate how often the following events happen to you. Circle the appropriate number.

| | Never | Very Seldom | Sometimes | Often | All the Time |
|---|---|---|---|---|---|
| B1. How often do you feel pain associated with the treatment for your diabetes? | 1 | 2 | 3 | 4 | 5 |
| B2. How often are you embarrassed by having to deal with your diabetes in public? | 1 | 2 | 3 | 4 | 5 |
| B3. How often do you have low blood sugar? | 1 | 2 | 3 | 4 | 5 |
| B4. How often do you feel physically ill? | 1 | 2 | 3 | 4 | 5 |
| B5. How often does your diabetes interfere with your family life? | 1 | 2 | 3 | 4 | 5 |

| | Never | Very Seldom | Sometimes | Often | All the Time |
|---|---|---|---|---|---|
| B6. How often do you have a bad night's sleep? | 1 | 2 | 3 | 4 | 5 |
| B7. How often do you find your diabetes limiting your social relationships and friendships? | 1 | 2 | 3 | 4 | 5 |
| B8. How often do you feel good about yourself? | 1 | 2 | 3 | 4 | 5 |
| B9. How often do you feel restricted by your diet? | 1 | 2 | 3 | 4 | 5 |
| B10. How often does your diabetes interfere with your sex life? | 1 | 2 | 3 | 4 | 5 |
| B11. How often does your diabetes keep you from driving a car or using a machine (e.g. a typewriter)? | 1 | 2 | 3 | 4 | 5 |
| B12. How often does your diabetes interfere with your exercising? | 1 | 2 | 3 | 4 | 5 |
| B13. How often do you miss work, school, or household duties because of your diabetes? | 1 | 2 | 3 | 4 | 5 |
| B14. How often do you find yourself explaining what it means to have diabetes? | 1 | 2 | 3 | 4 | 5 |
| B15. How often do you find that your diabetes interrupts your leisure-time activities? | 1 | 2 | 3 | 4 | 5 |
| B16. How often do you tell others about your diabetes? | 1 | 2 | 3 | 4 | 5 |

| | Never | Very Seldom | Sometimes | Often | All the Time |
|---|---|---|---|---|---|
| B17. How often are you teased because you have diabetes? | 1 | 2 | 3 | 4 | 5 |
| B18. How often do you feel that because of your diabetes you go to the bathroom more than others? | 1 | 2 | 3 | 4 | 5 |
| B19. How often do you find that you eat something you shouldn't rather than tell someone that you have diabetes? | 1 | 2 | 3 | 4 | 5 |
| B20. How often do you hide from others the fact that you are having an insulin reaction? | 1 | 2 | 3 | 4 | 5 |

Please indicate how often the following events happen to you. Please circle the number that best describes your feelings. If the question is not relevant to you, circle non-applicable.

| | Never | Very Seldom | Sometimes | Often | All the Time | Does not Apply |
|---|---|---|---|---|---|---|
| C1. How often do you worry about whether you will get married? | 1 | 2 | 3 | 4 | 5 | 0 |
| C2. How often do you worry about whether you will have children? | 1 | 2 | 3 | 4 | 5 | 0 |
| C3. How often do you worry about whether you will not get a job you want? | 1 | 2 | 3 | 4 | 5 | 0 |
| C4. How often do you worry about whether you will be denied insurance? | 1 | 2 | 3 | 4 | 5 | 0 |

| | Never | Very Seldom | Sometimes | Often | All the Time | Does not Apply |
|---|---|---|---|---|---|---|
| C5. How often do you worry about whether you will be able to complete your education? | 1 | 2 | 3 | 4 | 5 | 0 |
| C6. How often do you worry about whether you will miss work? | 1 | 2 | 3 | 4 | 5 | 0 |
| C7. How often do you worry about whether you will be able to take a vacation or a trip? | 1 | 2 | 3 | 4 | 5 | 0 |
| D1. How often do you worry about whether you will pass out? | 1 | 2 | 3 | 4 | 5 | 0 |
| D2. How often do you worry that your body looks differently because you have diabetes? | 1 | 2 | 3 | 4 | 5 | 0 |
| D3. How often do you worry that you will get complications from your diabetes? | 1 | 2 | 3 | 4 | 5 | 0 |
| D4. How often do you worry about whether someone will not go out with you because you have diabetes? | 1 | 2 | 3 | 4 | 5 | 0 |

E1. Compared to other people your age, would you say your health is: (Circle One)

1. Excellent
2. Good
3. Fair
4. Poor

# SF-36 HEALTH SURVEY

**INSTRUCTIONS:** This questionnaire asks for your views about your health, how you feel and how well you are able to do your usual activities.

Answer every question by marking the answer as indicated. If you are unsure about how to answer a question, please give the best answer you can.

1. In general, would you say your health is:

(circle one)

Excellent . . . . . 1

Very good . . . . . 2

Good . . . . . 3

Fair . . . . . 4

Poor . . . . . 5

2. Compared to one year ago, how would you rate your health in general now?

(circle one)

Much better now than one year ago . . . . . 1

Somewhat better now than one year ago . . . . . 2

About the same as one year ago . . . . . 3

Somewhat worse now than one year ago . . . . . 4

Much worse now than one year ago . . . . . 5

1 (For further information, write to: Medical Outcomes Trust, PO Box 1917, Boston MA 02205-8516, USA.)

3. The following questions are about activities you might do during a typical day. Does your health now limit you in these activities? If so, how much?

(circle one number on each line)

| ACTIVITIES | Yes, Limited A Lot | Yes, Limited A Little | No, Not Limited At All |
|---|---|---|---|
| a. Vigorous activities, such as running, lifting heavy objects, participating in strenuous sports | 1 | 2 | 3 |
| b. Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf | 1 | 2 | 3 |
| c. Lifting or carrying groceries | 1 | 2 | 3 |
| d. Climbing several flights of stairs | 1 | 2 | 3 |
| e. Climbing one flight of stairs | 1 | 2 | 3 |
| f. Bending, kneeling or stooping | 1 | 2 | 3 |
| g. Walking more than one kilometre | 1 | 2 | 3 |
| h. Walking half a kilometre | 1 | 2 | 3 |
| i. Walking 100 metres | 1 | 2 | 3 |
| j. Bathing or dressing yourself | 1 | 2 | 3 |

4. During the past 4 weeks, have you had any of the following problems with your work or other regular daily activities as a result of your physical health?

(circle one number on each line)

| | YES | NO |
|---|---|---|
| a. Cut down on the amount of time you spent on work or other activities | 1 | 2 |
| b. Accomplished less than you would like | 1 | 2 |
| c. Were limited in the kind of work or other activities | 1 | 2 |
| d. Had difficulty performing the work or other activities (for example, it took extra effort) | 1 | 2 |

2 (For further information, write to: Medical Outcomes Trust, PO Box 1917, Boston MA 02205-8516, USA.)

5. During the past 4 weeks, have you had any of the following problems with your work or other regular daily activities as a result of any emotional problems (such as feeling depressed or anxious)?

(circle one number on each line)

| | YES | NO |
|---|---|---|
| a. Cut down on the amount of time you spent on work or other activities | 1 | 2 |
| b. Accomplished less than you would like | 1 | 2 |
| c. Didn't do work or other activities as carefully as usual | 1 | 2 |

6. During the past 4 weeks, to what extent has your physical health or emotional problems interfered with your normal social activities with family, friends, neighbours, or groups?

(circle one)

Not at all . . . . . 1

Slightly . . . . . 2

Moderately . . . . . 3

Quite a bit . . . . . 4

Extremely . . . . . 5

7. How much bodily pain have you had during the past 4 weeks?

(circle one)

No bodily pain . . . . . 1

Very mild . . . . . 2

Mild . . . . . 3

Moderate . . . . . 4

Severe . . . . . 5

Very severe . . . . . 6



(For further information, write to: Medical Outcomes Trust, PO Box 1917, Boston MA 02205-8516, USA.)

8. During the past 4 weeks, how much did pain interfere with your normal work (including both work outside the home and housework)?

(circle one)

Not at all ..... 1

A little bit ..... 2

Moderately ..... 3

Quite a bit ..... 4

Extremely ..... 5

9. These questions are about how you feel and how things have been with you during the past 4 weeks. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the time during the past 4 weeks -

(circle one number on each line)

| | All of the Time | Most of the Time | A Good Bit of the Time | Some of the Time | A Little of the Time | None of the Time |
|---|---|---|---|---|---|---|
| a. Did you feel full of life? | 1 | 2 | 3 | 4 | 5 | 6 |
| b. Have you been a very nervous person? | 1 | 2 | 3 | 4 | 5 | 6 |
| c. Have you felt so down in the dumps that nothing could cheer you up? | 1 | 2 | 3 | 4 | 5 | 6 |
| d. Have you felt calm and peaceful? | 1 | 2 | 3 | 4 | 5 | 6 |
| e. Did you have a lot of energy? | 1 | 2 | 3 | 4 | 5 | 6 |
| f. Have you felt down? | 1 | 2 | 3 | 4 | 5 | 6 |
| g. Did you feel worn out? | 1 | 2 | 3 | 4 | 5 | 6 |
| h. Have you been a happy person? | 1 | 2 | 3 | 4 | 5 | 6 |
| i. Did you feel tired? | 1 | 2 | 3 | 4 | 5 | 6 |

10. During the past 4 weeks, how much of the time has your physical health or emotional problems interfered with your social activities (like visiting with friends, relatives, etc.)?

(circle one)

All of the time ..... 1

Most of the time ..... 2

Some of the time ..... 3

A little of the time ..... 4

None of the time ..... 5

11. How TRUE or FALSE is each of the following statements for you?

(circle one number on each line)

| | Definitely True | Mostly True | Don't Know | Mostly False | Definitely False |
|---|---|---|---|---|---|
| a. I seem to get sick a little easier than other people | 1 | 2 | 3 | 4 | 5 |
| b. I am as healthy as anybody I know | 1 | 2 | 3 | 4 | 5 |
| c. I expect my health to get worse | 1 | 2 | 3 | 4 | 5 |
| d. My health is excellent | 1 | 2 | 3 | 4 | 5 |

5 (For further information, write to: Medical Outcomes Trust, PO Box 1917, Boston MA 02205-8516, USA.)

**Anthropometric Measurements:**

Office use only

Weight

1.---------- kg 2. ---------- kg av: ---------- ☐ 54

Height

1. ----------cm 2. ---------- cm av: ---------- ☐ 55

BMI: ---------- kg/m2 ☐ 56

Skinfold thickness:

biceps (mm):

1. _ _ ._ 2. _ _._ av: _ _._ ☐ 57 A

triceps (mm):

1. _ _ ._ 2. _ _._ av: _ _._ ☐ 57 B

subscapular (mm):

1. _ _ ._ 2. _ _._ av: _ _._ ☐ 57 C

suprailiac (mm):

1. _ _ ._ 2. _ _._ av: _ _._

Sum of skinfold measurements: _ _._

Circumference Measurements:

Waist: ---------- cm ☐ 58

Hip circumference: ---------- cm ☐ 59

Waist/Hip: ---------- ☐ 60

**Blood Pressure:**

Systolic (mm Hg):

1. ---------- 2.---------- av: ---------- ☐ 61 A

Diastolic (mm Hg):

1. ---------- 2. ---------- av: ---------- ☐ 61 B

Mean Blood Pressure: ---------- (mm Hg) ☐ 61 C

# DOCUMENTATION CHECKLIST - AUSTRALIAN VERSION

Prepared by Effie Tsivis, Dietitian July 1995

Record portion sizes in the following standard measurements:

Weight in grams
Volume in fluid millilitres, cups, tablespoons or teaspoons
Fraction of the whole (eg 1/8 of 9" pie)
Comparison to approved food model
Dimensions for the following shapes:

| Shape | Measurement Needed | Example |
|---|---|---|
| Sphere | Diameter | Orange |
| Cylinder or disk | Diameter x thickness | Meat patty |
| Rectangle or cube | Length x height x width | Lasagne |
| Wedge | Length x height x width of arc | Pie |

| Food Group | Did You Specify: | Did You Probe for Additions and Amounts of: |
|---|---|---|
| **Beverages** | | |
| Coffee, Tea | Brewed, instant, decaf, herbal, coffee substitute eg (Caro, Ecco) | Sweetener, whitener, cream (type) |
| Cocoa, Drinking Chocolate | Mix (brand; regular, sugar-free or low-cal)<br>Mix (% fat) | Marshmallows Whipped topping (dairy or non-dairy) |
| Beer | Regular, light or low alcohol | |
| Liquor,Mixed Drinks, Liqueur | Name of mixed drink, liqueur<br>Proportion of ice | Mix (juice, other non-alcoholic beverage)<br>Cherry, olive, etc |
| Wine | Dinner or dessert | |
| Carbonated Beverages | Cola or non-cola, caffeine-free, diet, sodium-free<br>Proportion of ice | |
| **Dairy/Non-Dairy Products** | | |
| Milk, Cream, Toppings | % fat, dairy or non-dairy (brand)<br>If non-dairy: powder, liquid | Sweetener, cocoa mixes, etc |

| Food Group | Did You Specify: | Did You Probe for Additions and Amounts of: |
|---|---|---|
| **Dairy/Non-Dairy Products (continued)** | | |
| Cheese | Natural or processed kind (Cheddar, Swiss etc)<br>If low fat: brand or % fat<br>Low sodium | |
| Yoghurt | % fat, plain or flavoured, Brand | Fruit, nuts, etc |
| Ice Cream | Flavour: Rich/average fat/reduced fat | Topping |
| Milkshakes, Smoothies | Homemade or bought<br>Flavour - malt added<br>Icecream (Regular or low fat) or Yoghurt (Regular or low fat) | |
| Egg, Egg Substitutes | Method of preparation<br>Brand of substitute<br>Milk (% fat) if scrambled<br>Fat in preparation (kind) | Cheese, vegetables, meat etc |
| **Desserts, Baked Goods** | | |
| Puddings, Custards | Kind<br>Mix or homemade<br>Low-cal or regular<br>Milk (% fat)<br>With or without egg | Topping |
| Biscuits | Kind, brand<br>Mix, homemade or commercial | |
| Cakes | Kind<br>Mix, homemade or commercial<br>Cupcake, slice (large/small)<br>Ingredient fat<br>Additional oil, egg | Frosting, filling, topping |
| Pies | Kind (filling)<br>Mix, homemade or commercial<br>Single or double pastry<br>Ingredient fat for filling and pastry | Topping |
| Gelatine Desserts | Low-cal or regular | Topping, other additions (fruit etc) |

| Food Group | Did You Specify: | Did You Probe for Additions and Amounts of: |
|---|---|---|
| **Fats** | | |
| Oil, (solid and liquid) | Brand and/or type of fat | |
| Salad Dressing | Brand, type<br>Ingredient oil, if homemade<br>Creamy or clear<br>Low-cal or low sodium | |
| Margarine, Butter | Brand and major oil<br>Form (block, tub, diet, reduced fat<br>Salt free, salt reduced | |
| **Fruits/Fruit Juices** | Fresh, canned or dried<br>Cooked or uncooked<br>Sweetened or unsweetened<br>With or without peel | |
| **Grain Products** | | |
| Bread, Rolls | Kind; (white, wholemeal), rye etc) | Butter, margarine, other spread |
| Sweet Rolls, Doughnuts | Yeast or cake-type<br>Mix, homemade or commercial<br>Ingredient fat | Frosting, glaze, nuts, preserve |
| Pancakes, Waffles, Biscuits, Muffins | Kind; (wholemeal, buckwheat, bran etc) | Butter, margarine, syrup etc |
| Breakfast Cereals | Kind, brand | Milk (% fat)<br>Sweetener, fat, fruit, etc |
| Pasta, Rice | Kind; (spaghetti, brown rice, egg noodles etc)<br>Salt in preparation | Fat (kind), sauce, cheese etc |
| Crackers | Kind, brand | Spread |
| **Gravies, Sauces** | Packet Mix or homemade<br>Milk (% fat) or water<br>Fat (kind)<br>Salt in preparation | |

| Food Group | Did You Specify: | Did You Probe for Additions and Amounts of: |
|---|---|---|
| **Meat, Poultry, Fish** | | |
| Meat | Kind, cut<br>Trimmed or untrimmed, % fat of hamburger or type of mince<br>Fat in preparation<br>Salt in preparation<br>Cooked or raw weight<br>With or without bone | Sauce, gravy etc |
| Meatloaf | Kind, % fat or type of meat | Sauce, gravy, etc |
| Poultry | Light or dark meat (or name of part)<br>Prepared with or without skin<br>Skin eaten or not<br>Crumbed or battered and kind<br>Fat in preparation (kind)<br>Salt in preparation<br>Cooked or raw weight<br>With or without bone | Sauce, gravy, etc |
| Fish | Kind<br>Crumbed or battered and fried<br>Fat in preparation (kind)<br>Salt in preparation<br>Cooked or raw weight<br>Fresh or canned<br>If canned, water or oil pack, drained, undrained, low sodium | |
| Cold Cuts, Luncheon Meats | Kind, % fat, brand | |
| **Mixed Dishes** | Mix, homemade or commercial<br>Fat in preparation (kind)<br>Salt in preparation<br>Meat, kind and % fat<br>Sauce or gravy<br>Milk or cheese (% fat or kind)<br>Pasta or vegetables | Topping (eg croutons, crackers, cheese etc) |

| Food Group | Did You Specify: | Did You Probe for Additions and Amounts of: |
|---|---|---|
| Pizza | Thick or thin crust | Topping |
| Restaurant Meals | Type of restaurant | Usual dishes chosen |
| Seasonings/ Condiments | Salt or seasonings (eg celery salt, garlic salt, MSG) added in prep or at table | Pickle, relish, tomato sauce, mustard, BBQ sauce etc |
| Snacks/Candy | Kind, brand | |
| Soups | Kind; homemade or commercial<br>Ready to serve,<br>Milk (% fat) or cream added<br>Chunky or regular<br>Low sodium or low cal | Croutons, crackers, cheese etc |
| Vegetables | Cooked or raw<br>Fresh, frozen or canned<br>Low sodium<br>Salt in preparation | Fat (kind), cheese, sauce, nuts, dip etc |
| Salads | Kind (major vegetables) | Dressing, kind and/or brand<br>Croutons, seeds etc |
| Baked Potato | Skin eaten or not | Butter, sour cream, etc |
| Hot chips | Frozen, homemade,<br>Fat in preparation (kind) | Tomato sauce<br>Salt added |
| Miscellaneous<br>Medications containing nutrients such as sodium and/or caffeine | Type (eg analgesics, antacids, decongestants)<br>Brand | |
| Dietary Supplements | Kind, brand, amount of each nutrient (IU, mg, gm, mcg) on the Dietary Supplement Information Form<br>Number of tablets | |

## APPROVED ABBREVIATIONS

Use these and other standard abbreviations when documenting food intake on Dietary Intake Records.

| | | |
|---|---|---|
| approx - approximate | fl ml - fluid millilitres | pc - piece |
| avg - average | gm - gram | sl - slice |
| c - with | gr - ground | sm - small |
| cnd - canned | hyd - hydrogenated | med - medium |
| choc - chocolate | swt - sweetened | lg - large |
| chpd - chopped | mayo - mayonnaise | tb - tablespoon |
| comm - commercial | br - bread | ts - teaspoon |
| ckd - cooked | misc - miscellaneous | TVP - textured vegetable protein |
| crax - cracker | prep - preparation | unkn - unknown |
| cp - cup | pkg - package | veg - vegetable |
| diam - diameter | sat - saturated | w - with |
| fg - few grains | poly - polyunsaturated | w/o - without |
| | mono - monounsaturated | |

ADAPTED FROM THE DOCUMENTATION CHECKLIST
DEVELOPED BY:
The Nutrition Coordinating Centre
2829 University Avenue SW
MINNEAPOLIS, MN 55414

**Appendix 23: Diet history recording form**

**CLIENT CODE:** _____ **INTERVIEWER:** _____

**********

1. AGE:_____ 2. SEX: M/F 3. PREGNANT Yes/No

4. HEIGHT (cm): _____ 5. WEIGHT (kg): _____

6. ACTIVITY: (sedentary) / (light) / (light-mod) / (modrate) / (mod-heavy) / (heavy)

20 min sessions: nil /incidental/ 1-2/7 / 3-4/7 / 5-6/7 / >6/7

**********

**MORNING MEAL** **MIDDAY MEAL** **EVENING MEAL**

**MORNING TEA** **AFTERNOON TEA** **SUPPER**

**Appendix 24: Result letter to patients**

Date

<<name>>
<<address>>

Dear <<name>>

**Study on management of type 1 diabetes mellitus**

Thank you for taking part in this study. As you know this study was designed to assess management of type 1 diabetes in people aged 18-40. It is a study in which we try to find out as much as is useful about management in all people with type 1 diabetes in the Illawarra. We compared details (such as diet) with people without diabetes. Your participation was very important in ensuring the success of the study. Knowing how diabetes is currently managed in the "real world" helps us set standard for future management.

Attached are the results of some of measurements, details of your diet history and biochemical tests. The "normal" range of these biochemical tests is indicated in front of each value - but if you are outside this range it does not necessarily mean you have and undesirably low or high value. If you would like to discuss your results please call us (Professor Calvert or Mrs Farideh Tahbaz, phone 266 594), or discuss them when you next see your own doctor. We shall refer any queries which require knowledge of your background or management to your doctor.

We can not comment in this letter on the diet history. There is some disagreement among those who study diabetes on which aspects of diet are important, and the relationship between various aspects of diet and good diabetes management. One aim of this study is to clarify this relationship. However, we consider that individual dietary practices are best discussed in a clinical setting, and any dietary advice must take into account a host of factors that are not dealt with in this study.

If you have given your doctor's name to us, and said you would like your results forwarded to him or her, we will have sent them a copy of your results and a covering note.

Thank you again for taking part.

Yours sincerely

GD Calvert
Professor of Medicine and Public Health

Farideh Tahbaz
Research Student

**Appendix 25: Result letter to the doctors**

Date

Dear <<Dr name1>>

**Study on management of type 1 diabetes mellitus**

<<name 2>> <<DOB>>
<<address 2>>

Your patient <<name2>> has taken part in the Study on management of type 1 diabetes mellitus coordinated from the Medical Research Unit, Wollongong Hospital. This is primarily a study of dietary intake in adults with type 1 diabetes. A "usual consumption" diet history was taken, and a blood test and information relating to diabetic control, obesity, and demographic factors. As we noted in our initial letter to you, individual patients or practices will not be identified in any reports arising from this study. We expect to find a "gap" between what patients say they eat and what they think they are expected to eat. The hypothesis is that this "gap" is not related to poor diabetic control (high HbA1c%, dyslipidemia).

We agreed to give some results of measurements and details of the diet history to the patients who took part. Our advice to them is to discuss them with you next time they consult you. An individual dietary record is not easy to interpret on its own, outside a full consultation, and the record will only be of limited use. The reference ranges of biochemical tests is indicated. We have explained to the patient that values outside this range are not necessarily undesirable, and any queries of substance will be referred to you.

As you know, the relationship between various aspects of diet and good diabetes management is not clear. We have not discussed the diet history or offered any dietary advice, or in fact any advice on management of their diabetes. We consider that individual dietary practice is best discussed in a clinical setting, and any dietary advice must take into account a host of factors that are not dealt with in this study.

We believe that patients are best cared for by their own doctor, therefore we have suggested that they visit you if any of our findings seem to need review or follow-up.

Yours sincerely

GD Calvert
Professor of Medicine and Public Health

Farideh Tahbaz
Research Student

**Appendix 26: Result letter to controls**

Date

Dear << name>>

**Study on management of type 1 diabetes mellitus**

We are grateful for your contribution to our Study on management of type 1 diabetes mellitus undertaken at the Medical Research Unit, Wollongong Hospital.

Attached are the results of some measurements, details of diet history and biochemical tests. The "normal" range of these biochemical tests are indicated under the heading "Desirable" Range, but if you are outside this range it does not necessarily mean you have an undesirably low or high value.

as you see on the result sheet, the urine tests are screening tests only, and we will contact you again to ask if we can check kidney function with a more accurate estimation based on urine collected early morning.

If you would like to discuss your results please call us (Professor Calvert or Mrs Farideh Tahbaz, phone 266 594), or discuss them when you see your own doctor.

Yours sincerely

GD Calvert
Professor of Medicine and Public Health

Farideh Tahbaz
Research Student

**Appendix 27: Results Form**

## RESULTS

**NAME: <<name>>**

| **Body Measurements:** | **Estimated** | **"Desirable Range"** |
|---|---|---|
| Body Mass Index | <<BMI>> kg per meter$^2$ | 19-26 for men and women |
| Body fat | <<BF%>> | 15-20% for men<br>20-28% for women |
| Waist Hip Ratio | <<WHR>> | <0.90 for men<br><0.80 for women |
| **Blood Results:** | | |
| HbA1c% | <<HbA1c%>> | 4.2-5.9 |
| Plasma cholesterol | <<Chol>> mmol/l | <5.2 mmol/l |
| Serum creatinine | <<SCre>> µmol/l | 60-100 µmol/l |
| Serum albumin | <<SAlb>> g/l | 36-52 g/l |
| Plasma fibrinogen | <<PFib>> g/l | 2.0-4.0 g/l |
| **URINE RESULTS*:** | | |
| Albumin | <<Alb>> mg/l | |
| Creatinine | <<Creat>> mmol/l | |

* These urine tests are a screen for protein (albumin) in urine. We will contact you to request a further urine sample taken early morning which will give more accurate measurements of protein in urine and of kidney function.

Details:
Male, 36 years, 73 kg
Heavy activity

Body Mass Index (BMI): 23

Nutrients (Mean all Days)

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Weight: | 4052.20 g | Carbohydrate: | 441.43 g | Sat. Fat: | 45.69 g | Potassium: | 5236.67 mg |
| Energy: | 15229.59 kJ | Fibre: | 37.66 g | Mono. Fat: | 53.04 g | Calcium: | 2104.56 mg |
| Energy: | 3638.22 kcal | Sugars: | 132.19 g | Poly. Fat: | 19.67 g | Phosphorus: | 2451.77 mg |
| Protein: | 160.33 g | Starch: | 307.90 g | Retinol Eq: | 724.07 ug | Iron: | 20.03 mg |
| Fat: | 130.88 g | Cholesterol: | 248.86 mg | Sodium: | 6321.83 mg | Zinc: | 20.54 mg |

Energy Ratios (Mean all Days)
Protein: 18% Fat: 33% Carbohydrate: 48% Alcohol: 1%

Fat Ratios (Mean all Days)
Poly: 17% Mono: 45% Saturated: 39%

Recommended Dietary Intakes:

| | Mean | Rdi | % |
|---|---|---|---|
| Protein (g) | 160.3 | 55.0 | 292 |
| Thiamin (mg) | 2.4 | 1.5 | 162 |
| Riboflavin (mg) | 3.2 | 2.3 | 143 |
| Niacin Eq (mg) | 57.7 | 24.0 | 240 |
| Retinol Eq (ug) | 724.1 | 750.0 | 97 |
| Vitamin C (mg) | 138.4 | 40.0 | 346 |
| Iron (mg) | 20.0 | 7.0 | 286 |
| Zinc (mg) | 20.5 | 12.0 | 171 |
| Potassium (mg) | 5236.7 | 1950.0 | 269 |
| Calcium (mg) | 2104.6 | 800.0 | 263 |
| Phosphorus (mg) | 2451.8 | 1000.0 | 245 |
| Magnesium (mg) | 527.1 | 320.0 | 165 |

0% 50% 100% 150% 200%

Energy Requirements:

| | Kjoule |
|---|---|
| Basal Metabolic Rate: | 7157 |
| Bed Rest: | 8588 |
| Average Requirement: | 15030 |
| Estimated Intake: | 15230 |
| Expected Individual Range: | 12024-18036 |

**Appendix 29- Descriptive data on different continuous variables in diabetic subjects**

| variable | mean | SD | range | percentile 75th | 50th | 25th |
|---|---|---|---|---|---|---|
| age (yr) | 31.2 | 6.1 | 18-40 | 36 | 32 | 26 |
| diabetes duration (yr) | 6.4 | 3.3 | 1-12 | 10 | 6 | 4 |
| Insulin dose (unit/day) | 51 | 22.5 | 8-108 | 65 | 48 | 34 |
| Insulin dose (bwt (kg)/day) | 0.68 | 0.29 | 0.08-1.47 | 0.83 | 0.66 | 0.50 |
| insulin injection (no) | 2.49 | 0.67 | 2-5 | 3.0 | 2.0 | 2.0 |
| BMI[a] ($kg/m^2$) | 25.8 | 3.8 | 20.5-41.8 | 27.3 | 24.9 | 23.2 |
| Body fat% | 22.0 | 6.8 | 10.5-35.7 | 29.0 | 20.7 | 11.1 |
| WHR[b] | 0.84 | 0.08 | 0.60-1.02 | 0.89 | 0.84 | 0.79 |
| sBP[c] (mmHg) | 121.8 | 15.5 | 100.0-165.0 | 130.0 | 120.0 | 110.0 |
| dBP[d] (mmHg) | 74.1 | 13.9 | 50.0-110.0 | 80.0 | 70.0 | 60.0 |
| HbA1c% | 8.6 | 2.0 | 4.6-13.8 | 10.0 | 8.3 | 7.1 |
| Glucose mmol/l) | 11.1 | 6.3 | 1.9-27.3 | 14.2 | 11.3 | 6.4 |
| Cholesterol (mmol/l) | 4.7 | 1.1 | 3.2-8.2 | 5.3 | 4.4 | 4.0 |
| HDL cholesterol (mmol/l) | 1.43 | 0.53 | 0.42-3.64 | 1.70 | 1.30 | 1.14 |
| LDL cholesterol (mmol/l) | 2.71 | 0.88 | 1.25-5.73 | 3.14 | 2.47 | 1.33 |
| Triglyceride (mmol/l) | 1.18 | 0.74 | 0.32-3.99 | 1.33 | 0.98 | 0.67 |
| Creatinine (µmol/l) | 76.14 | 11.13 | 51.0-100.0 | 83.0 | 76.0 | 65.0 |
| Albumin (g/l) | 41.82 | 2.97 | 35.0-47.0 | 44.0 | 42.0 | 39.0 |
| Fibrinogen (g/l) | 2.64 | 0.78 | 1.71-6.40 | 2.84 | 2.56 | 2.05 |
| Urine albumin (mg/l) (1)[e] | 11.76 | 19.06 | 2.0-104.0 | 10.30 | 6.28 | 3.18 |
| Urine creatinine (mmol/l)(1) | 10465.2 | 6462.6 | 2074.0-25549.0 | 16522.0 | 8534.0 | 4160.0 |
| Alb (mg/l)/creat (mmol/l)(1) | 0.79 | 2.04 | 0.29-16.22 | 1.01 | 0.69 | 0.50 |
| Urine albumin (mg/l) (2)[f] | 9.22 | 8.29 | 2.0-40.70 | 12.30 | 6.62 | 3.72 |
| Urine creat (µmmol/l) (2) | 12629.1 | 5802.9 | 3178.0-22904.0 | 17566.0 | 13687.0 | 6634.0 |
| Alb (mg/l)/creat (mmol/l)(2) | 0.62 | 2.09 | 0.23-7.94 | 0.78 | 0.54 | 0.36 |
| Protein (g) | 129.8 | 37.8 | 63-219 | 158.5 | 124.5 | 98.7 |
| Prot (g)/bwt (kg) | 1.75 | 0.55 | 0.88-3.10 | 2.06 | 1.62 | 1.40 |
| Total fat (g) | 85.3 | 37.3 | 75.1-95.4 | 113.2 | 78.5 | 55.0 |
| Carbohydrate (g) | 302.5 | 90.1 | 153.0-521.0 | 348.0 | 293.5 | 242.0 |
| Starch (g) | 180.9 | 65.2 | 85.0-377.0 | 210.5 | 173.5 | 128.5 |
| Sugar (g) | 118.9 | 45.7 | 27.0-252.0 | 141.2 | 113.0 | 92.0 |
| Dietary fibre (g) | 38.2 | 11.3 | 15.0-66.0 | 44.5 | 37.5 | 29.7 |
| Alcohol (g) | 6.8 | 13.8 | 0.0-85.2 | 9.1 | 0.0 | 0.0 |
| Energy (kJ) | 10621 | 2990 | 4769-17211 | 13163 | 10462 | 8451 |
| Energy (kcal) | 2537 | 714 | 1139-4112 | 3145 | 2499 | 2019 |
| Fat (energy%) | 30.3 | 7.9 | 13.0-56.0 | 34.5 | 30.0 | 14.1 |
| Carbohydrate (energy%) | 46.0 | 10.0 | 34.0-64.0 | 52.2 | 46.0 | 42.7 |
| Protein (energy%) | 21.3 | 4.1 | 13.0-33.0 | 24.0 | 21.0 | 18.0 |
| Alcohol (energy%) | 2.1 | 4.1 | 0.0-22.0 | 3.0 | 0.0 | 0.0 |
| PUFA (g)[g] | 14.6 | 10.7 | 2.0-68.0 | 18.2 | 11.5 | 7.7 |
| PUFA (energy%) | 5.9 | 3.1 | 1.7-18.5 | 6.8 | 5.5 | 3.8 |
| MUFA (g)[g] | 28.3 | 13.9 | 5.0-70.0 | 41.2 | 24.5 | 17.7 |
| MUFA (energy%) | 11.6 | 4.0 | 4.5-29.1 | 14.0 | 10.8 | 8.9 |

[a] Body Mass Index. [b] Waist to Hip Ratio. [c] Systolic blood pressure. [d] Diastolic blood pressure. [e] (1) On spot urine sample. [f] (2) On early morning urine sample. [g]Polyunsaturated fatty acids. [g]Monounsaturated fatty acids.

**Descriptive data on different continuous variables in diabetic subjects (continued)**

| variable | mean | SD | range | percentile 75th | 50th | 25th |
|---|---|---|---|---|---|---|
| SAFA (g)[i] | 31.5 | 13.8 | 8.0-59.0 | 44.2 | 28.0 | 20.0 |
| SAFA (energy%) | 13.1 | 4.1 | 6.2-29.1 | 15.5 | 12.4 | 9.7 |
| P:S[j] | 0.48 | 0.26 | 0.17-1.48 | 0.59 | 0.44 | 0.33 |
| M:S[k] | 0.90 | 0.21 | 0.3-1.61 | 1.00 | 0.85 | 0.75 |
| Cholesterol (mg) | 261.2 | 131.7 | 88.9-695.0 | 322.6 | 235.8 | 172.3 |
| Retinol Eq (µg) | 1751.7 | 960.9 | 35.4-5383.2 | 2361.6 | 1617.2 | 995.9 |
| Vitamin C (mg) | 209.5 | 136.7 | 29.9-621.3 | 279.5 | 179.8 | 107.9 |
| Thiamin (mg) | 2.3 | 1.2 | 1.0-6.2 | 2.6 | 2.2 | 1.5 |
| Riboflavin (mg) | 3.3 | 1.5 | 1.2-7.8 | 3.8 | 3.1 | 2.2 |
| Niacin Eq (mg) | 54.1 | 17.3 | 25.2-103.9 | 59.6 | 53.7 | 42.5 |
| Sodium (mg) | 3533.8 | 1582.2 | 1027.0-9281.1 | 4140.2 | 3288.7 | 2504.3 |
| Potassium (mg) | 4723.7 | 1200.9 | 2666.1-7751.4 | 5355.0 | 4745.8 | 3824.3 |
| Calcium (mg) | 1184.9 | 486.8 | 484.6-3169.1 | 1452.0 | 1073.0 | 836.5 |
| Iron (mg) | 18.8 | 6.4 | 8.0-37.0 | 23.5 | 17.5 | 15.0 |
| Zinc (mg) | 16.5 | 6.4 | 6.0-32.0 | 21.0 | 15.8 | 11.4 |
| Diet score | 15.7 | 1.8 | 11.0-19.0 | 17.0 | 16.0 | 14.0 |
| Satisfaction | 73.9 | 15.5 | 35.0-98.3 | 85.0 | 76.7 | 63.3 |
| Impact | 73.9 | 11.4 | 33.7-91.2 | 82.8 | 75.0 | 68.7 |
| Social worry | 81.4 | 15.2 | 50.00-100.0 | 93.1 | 86.6 | 70.8 |
| Diabetes worry | 74.2 | 17.1 | 37.5-100.0 | 87.5 | 75.0 | 58.3 |
| Total | 75.6 | 10.6 | 72.5-78.6 | 83.5 | 75.6 | 70.8 |
| Physical function | 92.2 | 18.3 | 10.0-100.0 | 100.0 | 95.0 | 90.0 |
| Role-physical | 86.7 | 29.3 | 0.0-100.0 | 100.0 | 100.0 | 100.0 |
| Bodily pain | 79.2 | 14.5 | 20.0-90.0 | 90.0 | 80.0 | 72.5 |
| General health | 67.2 | 19.0 | 10.0-100.0 | 80.0 | 67.5 | 55.0 |
| Vitality | 66.4 | 18.0 | 20.0-100.0 | 80.0 | 70.0 | 55.0 |
| Social function | 86.1 | 20.6 | 12.5-100.0 | 100.0 | 100.0 | 75.0 |
| Role-emotional | 85.6 | 29.2 | 0.0-100.0 | 100.0 | 100.0 | 100.0 |
| Mental health | 76.9 | 15.9 | 40.0-100.0 | 92.0 | 82.0 | 64.0 |

[i] Saturated fatty acids. [j] Polyunsaturated to saturated fatty acids. [j] Monounsaturated to saturated fatty acids.

**Appendix 30- Descriptive data on different continuous variables in control subjects**

| variable | mean | SD | range | percentile 75th | 50th | 25th |
|---|---|---|---|---|---|---|
| Age (yr) | 27.7 | 5.1 | 18-40 | 31 | 27 | 24 |
| BMI (kg/m$^2$)[a] | 25.3 | 4.0 | 17.7-34.9 | 27.7 | 25.1 | 22.1 |
| Body fat% | 21.5 | 6.6 | 10.6-37.6 | 25.9 | 20.8 | 15.4 |
| WHR[b] | 0.84 | 0.1 | 0.68-1.10 | 0.91 | 0.85 | 0.74 |
| sBP (mmHg)[c] | 121.9 | 11.8 | 98.0-160.0 | 125.0 | 120.0 | 120.0 |
| dBP (mmHg)[d] | 72.7 | 8.1 | 60.0-95.0 | 80.0 | 70.0 | 70.0 |
| HbA1c% | 5.3 | 0.6 | 4.1-6.4 | 5.7 | 5.4 | 4.9 |
| Glucose mmol/l) | 5.1 | 0.7 | 3.9-7.3 | 5.5 | 5.0 | 4.6 |
| Cholesterol (mmol/l) | 5.0 | 1.1 | 8.0-7.6 | 5.5 | 4.8 | 4.2 |
| HDL cholesterol (mmol/l) | 1.2 | 0.4 | 0.4-2.1 | 1.5 | 1.2 | 0.9 |
| LDL cholesterol (mmol/l) | 3.0 | 1.0 | 1.2-5.7 | 3.4 | 2.9 | 2.4 |
| Triglyceride (mmol/l) | 1.5 | 1.0 | 0.44-5.2 | 1.9 | 1.3 | 0.9 |
| Creatinine (µmol/l) | 86.6 | 15.0 | 54.0-126.0 | 97.0 | 84.0 | 77.0 |
| Albumin (g/l) | 43.3 | 2.6 | 37.0-49.0 | 45.0 | 43.0 | 41.0 |
| Fibrinogen (g/l) | 2.2 | 0.6 | 1.0-4.0 | 2.7 | 2.2 | 1.7 |
| Urine albumin (mg/l) (1)[e] | 7.38 | 4.36 | 2.0-21.6 | 9.25 | 6.7 | 4.3 |
| Urine creatinine (mmol/l)(1) | 14012.5 | 6550.5 | 1848.0-29350.0 | 18336.0 | 14038.0 | 9596.0 |
| Alb (mg/l)/creat (mmol/l)(1) | 0.51 | 1.62 | 0.19-1.58 | 0.67 | 0.48 | 0.35 |
| Urine albumin (mg/l) (2)[f] | 6.41 | 3.92 | 2.0-18.0 | 8.92 | 5.22 | 3.42 |
| Urine creat (µmmol/l) (2) | 15271.1 | 5985.6 | 5795.0-30056.0 | 19040.0 | 15290.0 | 10858.0 |
| Alb (mg/l)/creat (mmol/l)(2) | 0.39 | 1.38 | 0.23-1.01 | 0.46 | 0.37 | 0.31 |
| Protein (g) | 115.3 | 34.7 | 66.0-209.0 | 136.5 | 113.0 | 86.6 |
| Prot (g)/bwt (kg) | 1.7 | 0.8 | 0.7-6.3 | 1.9 | 1.5 | 1.2 |
| Total fat (g) | 90.6 | 35.5 | 15.0-163.0 | 118.0 | 87.0 | 61.0 |
| Carbohydrate (g) | 267.6 | 79.7 | 131.0-493.0 | 297.0 | 263.0 | 197.0 |
| Starch (g) | 134.3 | 51.5 | 41.0-265.0 | 173.0 | 130.0 | 95.0 |
| Sugar (g) | 130.6 | 53.0 | 165.0-123.0 | 93.0 | 123.0 | 93.0 |
| Dietary fibre (g) | 31.6 | 10.4 | 12.0-57.0 | 39.0 | 30.0 | 25.0 |
| Alcohol (g) | 8.8 | 13.5 | 0.0-62.7 | 12.6 | 3.4 | 0.0 |
| Energy (kJ) | 10057 | 2633 | 5193-15408 | 12146 | 10185 | 8024 |
| Energy (kcal) | 2404 | 629 | 1241-3682 | 2901 | 2437 | 1917 |
| Fat (energy%) | 32.7 | 8.2 | 11.0-56.0 | 37.0 | 34.0 | 29.0 |
| Carbohydrate (energy%) | 43.7 | 7.3 | 31.0-63.0 | 51.0 | 43.0 | 38.0 |
| Protein (energy%) | 20.7 | 4.9 | 11.0-40.0 | 23.0 | 20.0 | 18.0 |
| Alcohol (energy%) | 2.6 | 4.4 | 0.0-23.0 | 3.0 | 1.0 | 0.0 |
| PUFA (g)[g] | 12.2 | 7.4 | 3.0-39.0 | 15.0 | 11.0 | 7.0 |
| PUFA (energy%) | 5.0 | 2.5 | 1.3-12.9 | 6.1 | 5.0 | 3.4 |
| MUFA (g)[h] | 31.0 | 14.3 | 5.0-69.0 | 40.0 | 31.0 | 19.0 |
| MUFA (energy%) | 12.5 | 3.8 | 4.2-22.4 | 14.4 | 12.9 | 10.5 |
| SAFA (g)[i] | 37.1 | 15.7 | 5.0-69.0 | 49.0 | 36.0 | 25.0 |
| SAFA (energy%) | 15.1 | 4.3 | 4.3-24.0 | 18.4 | 15.4 | 12.6 |
| P:S[j] | 0.36 | 0.21 | 0.10-1.00 | 0.44 | 0.32 | 0.21 |

[a] Body Mass Index. [b] Waist to Hip Ratio. [c] Systolic blood pressure. [d] Diastolic blood pressure. [e] (1) On spot urine sample. [f] (2) On early morning urine sample. [g]Polyunsaturated fatty acids. [g]Monounsaturated fatty acids. [i] Saturated fatty acids. [j]Polyunsaturated fatty acids to saturated fatty acids ratio.

**Descriptive data on different continuous variables in control subjects (continued)**

| variable | mean | SD | range | percentile 75th | 50th | 25th |
|---|---|---|---|---|---|---|
| M:S [k] | 0.85 | 0.22 | 0.52-1.44 | 0.97 | 0.83 | 0.70 |
| Cholesterol (mg) | 296.4 | 159.6 | 70.5-904.2 | 365.5 | 275.4 | 192.2 |
| Retinol Eq (µg) | 1904.2 | 1074.1 | 252.3-6331.5 | 2374.2 | 1872.0 | 1129.4 |
| Vitamin C (mg) | 257.2 | 326.2 | 19.6-2039.6 | 283.1 | 190.5 | 124.5 |
| Thiamin (mg) | 1.9 | 0.7 | 0.7-4.4 | 2.3 | 1.9 | 1.3 |
| Riboflavin (mg) | 2.8 | 1.2 | 1.0-5.8 | 3.4 | 2.5 | 2.1 |
| Niacin Eq (mg) | 43.1 | 15.8 | 11.3-79.6 | 54.0 | 40.4 | 33.0 |
| Sodium (mg) | 2718.9 | 1146.6 | 1154.9-5805.0 | 3340.7 | 2558.5 | 1944.9 |
| Potassium (mg) | 4204.9 | 1245.6 | 2265.0-8588.6 | 4876.0 | 4038.2 | 3308.2 |
| Calcium (mg) | 1097.9 | 412.9 | 299.4-2726.4 | 1297.3 | 1031.0 | 889.8 |
| Iron (mg) | 18.1 | 6.4 | 6.1-42.5 | 21.9 | 17.3 | 13.8 |
| Zinc (mg) | 15.2 | 6.4 | 6.1-31.0 | 18.9 | 14.0 | 9.6 |
| Diet score | 15.3 | 2.4 | 12.0-20.0 | 17.0 | 15.0 | 13.0 |
| Physical function | 92.7 | 12.4 | 89.1-96.4 | 100.0 | 95.0 | 90.0 |
| Role-physical | 95.2 | 13.4 | 50.0-100.0 | 100.0 | 100.0 | 100.0 |
| Bodily pain | 80.4 | 13.7 | 30.0-90.0 | 90.0 | 90.0 | 80.0 |
| General health | 72.2 | 17.4 | 30.0-100.0 | 85.0 | 80.0 | 60.0 |
| Vitality | 63.3 | 20.8 | 15.0-90.0 | 80.0 | 70.0 | 50.0 |
| Social function | 89.9 | 15.5 | 37.5-100.0 | 100.0 | 100.0 | 87.0 |
| Role-emotional | 87.2 | 26.5 | 0.0-100.0 | 100.0 | 100.0 | 100.0 |
| Mental health | 78.4 | 14.3 | 44.0-100.0 | 88.0 | 84.0 | 71.0 |

[k] Monounsaturated fatty acids to saturated fatty acids ratio.

## Appendix 31: Correlation coefficient of different variables in patients with type 1 diabetes*

| Variable | Age | dia.yr. | ins/bwt | BMI | BF% | WHR |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| dia.yr. | -0.1767 | | | | | |
| ins/bwt | -0.5058 | 0.5634 | | | | |
| BMI | 0.1747 | 0.2459 | -0.1488 | | | |
| BF% | -0.0746 | 0.6442 | 0.3295 | 0.4483 | | |
| WHR | -0.1015 | -0.2845 | -0.2574 | 0.5150 | -0.2073 | |
| sBP | 0.0503 | -0.1155 | -0.2055 | 0.2608 | -0.0981 | 0.3820 |
| dBP | 0.2363 | -0.1720 | -0.1976 | 0.2310 | -0.0550 | 0.4064 |
| HbA1c% | -0.5379 | 0.2230 | 0.4873 | -0.1182 | -0.0606 | -0.0850 |
| C(mmol/l) | 0.0912 | -0.1470 | -0.1159 | -0.0630 | -0.0658 | 0.2944 |
| HDL C(mmol/l) | -0.0747 | 0.1512 | 0.6254 | -0.4605 | -0.0734 | -0.5095 |
| LDL C(mmol/l) | 0.0141 | -0.1472 | -0.2577 | -0.0251 | -0.1293 | 0.4009 |
| trig(mmol/l) | 0.3294 | -0.2149 | -0.4693 | 0.5333 | 0.2841 | 0.4134 |
| cre(umol/l) | 0.1650 | -0.4946 | -0.4750 | -0.0154 | -0.4111 | 0.3078 |
| alb(g/l) | -0.1437 | 0.0742 | -0.1076 | -0.1219 | -0.0164 | -0.0212 |
| fibr(g/l) | -0.2047 | 0.3418 | 0.3077 | 0.1972 | 0.5360 | -0.1360 |
| log alb/cre 1 | -0.0083 | -0.0447 | -0.2764 | 0.2128 | 0.4100 | 0.0886 |
| log alb/cre 2 | -0.1977 | 0.0768 | 0.0429 | 0.0698 | 0.3744 | 0.0652 |
| Prot(En%) | 0.1396 | 0.1483 | -0.1834 | -0.0764 | 0.1391 | -0.4366 |
| Fat(En%) | -0.3597 | -0.0126 | 0.1810 | 0.0708 | -0.1736 | 0.3966 |
| CHO(En%) | 0.1579 | 0.0900 | 0.1628 | -0.1446 | 0.1712 | -0.5018 |
| Alc (energy%) | 0.2196 | -0.2392 | -0.3940 | 0.1223 | -0.1062 | 0.4217 |
| PUFA(En%) | -0.3287 | -0.0297 | 0.1650 | -0.0556 | -0.2953 | 0.1561 |
| MUFA(En%) | -0.3277 | -0.0256 | 0.1766 | 0.0094 | -0.1133 | 0.2958 |
| SAFA(En%) | -0.1754 | 0.0231 | 0.0638 | 0.1848 | -0.0111 | 0.4323 |
| P/S | -0.2949 | -0.0880 | 0.1104 | -0.1949 | -0.3130 | -0.0343 |
| M/S | -0.2678 | -0.0657 | 0.1422 | -0.2307 | -0.1990 | -0.0952 |
| En (kJ) | -0.0524 | -0.0824 | -0.0153 | 0.0303 | -0.5679 | 0.4250 |
| Prot(g) | 0.0402 | -0.0253 | -0.1141 | -0.0243 | -0.5055 | 0.2134 |
| Prot/bwt | -0.0084 | 0.0018 | 0.0529 | -0.4545 | -0.4275 | -0.1715 |
| Fat(g) | -0.1957 | -0.0336 | 0.0730 | 0.0729 | -0.3869 | 0.4437 |
| CHO (g) | 0.0044 | -0.0365 | 0.0988 | -0.0597 | -0.5470 | 0.1580 |
| Alc(g) | 0.2263 | -0.2429 | -0.3998 | 0.1365 | -0.1175 | 0.4350 |
| Fibre(g) | 0.3209 | 0.0214 | -0.2002 | -0.0173 | -0.4258 | 0.0627 |
| Sugar(g) | 0.1830 | 0.2352 | 0.2603 | -0.0659 | -0.2128 | -0.0917 |
| Starch(g) | -0.0852 | -0.1675 | -0.0150 | -0.0199 | -0.5739 | 0.2545 |
| chol(mg) | 0.0550 | 0.1368 | -0.0007 | 0.0439 | -0.3012 | 0.2509 |
| Na (mg) | -0.2834 | -0.0101 | 0.1998 | -0.0130 | -0.3346 | 0.4412 |
| satisfaction | -0.0014 | 0.1125 | -0.1579 | -0.1768 | 0.0493 | -0.3260 |
| impact | 0.1442 | -0.1823 | -0.3937 | -0.0442 | -0.0362 | 0.0151 |
| soc wor | 0.3639 | -0.0401 | -0.3473 | 0.1879 | -0.1031 | 0.0782 |
| diab wor | 0.2646 | -0.3583 | -0.3203 | -0.3268 | -0.2969 | -0.1550 |
| total | 0.2602 | -0.1615 | -0.3920 | -0.1297 | -0.1414 | -0.1334 |
| phy fun | -0.0574 | -0.2357 | -0.2622 | 0.0521 | -0.2175 | 0.0585 |
| role-phy | 0.1183 | 0.0794 | -0.2203 | 0.0613 | 0.1568 | -0.2416 |
| body pain | 0.4323 | -0.1830 | -0.4234 | -0.1660 | -0.2577 | -0.2695 |
| gen hlth | 0.1949 | -0.1805 | -0.3479 | -0.0078 | -0.0948 | -0.1707 |
| vitality | 0.5035 | -0.1921 | -0.5034 | 0.1323 | -0.2786 | 0.1121 |
| soc func | 0.2994 | -0.0407 | -0.4102 | 0.1114 | -0.1688 | -0.0405 |
| role-emo | 0.5277 | 0.1250 | -0.1709 | 0.0956 | 0.1185 | -0.2725 |
| ment hlth | 0.4969 | 0.0076 | -0.3214 | 0.1598 | 0.0741 | -0.2014 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by age, diabetes duration (yr.), units of insulin/body weight (kg), body mass index, body fat% and waist to hip ratio respectively.

## Correlation coefficient of different variables in patients with type 1 diabetes* (continued)

| Variable | sBP | dBP | HbA1c% | C(mmol/l) | HDL C(mmol/l) | LDL C(mmol/l) |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| diab.yr | | | | | | |
| ins/bwt | | | | | | |
| BMI | | | | | | |
| B F% | | | | | | |
| sBP | | | | | | |
| dBP | | | | | | |
| HbA1c% | 0.7636 | | | | | |
| C (mmol/l) | 0.0935 | -0.0651 | | | | |
| HDL C (mmol/l) | 0.0359 | 0.0478 | 0.0800 | | | |
| LDL C (mmol/l) | -0.2953 | -0.4248 | 0.3498 | -0.0226 | | |
| trig (mmol/l) | 0.1896 | 0.1856 | 0.0612 | 0.9307 | -0.2857 | |
| cre(umol/l) | -0.0291 | 0.1983 | -0.4329 | 0.2581 | -0.6409 | 0.2247 |
| alb (g/l) | -0.1346 | -0.0152 | -0.5582 | 0.0616 | -0.3168 | 0.1115 |
| fibr (g/l) | -0.1527 | -0.2283 | -0.0431 | 0.3901 | -0.0854 | 0.3706 |
| log alb/cre1 | -0.2641 | -0.1637 | 0.0650 | 0.1220 | -0.0994 | 0.0315 |
| log alb/cre2 | -0.0988 | -0.1094 | -0.2341 | -0.1123 | -0.2633 | -0.1335 |
| Prot (En%) | 0.2322 | 0.2249 | 0.0734 | -0.1209 | -0.1957 | -0.0688 |
| Fat (En %) | -0.0962 | -0.2295 | -0.2545 | -0.0786 | -0.1266 | -0.0222 |
| CHO (En %) | -0.0961 | -0.2201 | 0.3223 | 0.2780 | -0.0060 | 0.3048 |
| Alc (En %) | 0.0723 | 0.2394 | 0.0670 | -0.5737 | 0.2085 | -0.6256 |
| PUFA(En%) | 0.0996 | 0.1523 | -0.4174 | 0.4599 | -0.2112 | 0.4534 |
| MUFA(En%) | -0.1167 | -0.2284 | 0.2587 | 0.1802 | 0.0123 | 0.2253 |
| SAFA(En%) | -0.0026 | -0.1809 | 0.3133 | 0.3396 | 0.0527 | 0.3450 |
| P/S | -0.0945 | -0.0964 | 0.1671 | 0.1111 | -0.0792 | 0.1282 |
| M/S | -0.0302 | -0.1817 | 0.1395 | 0.0549 | 0.0603 | 0.0975 |
| En (kJ) | 0.1030 | -0.1677 | 0.2044 | 0.2746 | 0.1661 | 0.2689 |
| Prot (g) | 0.2036 | 0.0422 | 0.2795 | 0.3114 | 0.0658 | 0.3523 |
| Prot/bwt | 0.1357 | -0.1248 | 0.1007 | 0.3141 | 0.0141 | 0.3695 |
| Fat(g) | -0.1183 | -0.3026 | 0.1099 | 0.3467 | 0.2819 | 0.3207 |
| CHO (g) | 0.0746 | -0.0713 | 0.3211 | 0.3448 | -0.0090 | 0.3905 |
| Alc (g) | 0.2670 | 0.1703 | 0.3685 | -0.0510 | 0.2496 | -0.0400 |
| Fibre(g) | 0.0936 | 0.1379 | -0.4152 | 0.4787 | -0.2124 | 0.4720 |
| Sugar (g) | 0.0653 | 0.0468 | -0.0310 | -0.1327 | -0.0755 | -0.0134 |
| Starch (g) | -0.1295 | -0.0162 | 0.1905 | -0.1967 | 0.3651 | -0.3089 |
| chol (mg) | 0.4022 | 0.2271 | 0.3708 | 0.0353 | 0.1090 | 0.1084 |
| Na (mg) | 0.0052 | -0.2651 | 0.0912 | 0.4479 | 0.0762 | 0.4285 |
| satisfaction | 0.1650 | 0.0490 | 0.3817 | 0.2932 | 0.1539 | 0.2943 |
| impact | 0.0431 | -0.0715 | 0.0488 | -0.1225 | -0.1421 | -0.0520 |
| soc wor | 0.2297 | 0.0553 | -0.1460 | 0.2634 | -0.2632 | 0.3287 |
| diab wor | 0.1349 | -0.0691 | -0.2970 | 0.1667 | -0.1603 | 0.1534 |
| total | 0.1279 | -0.0161 | -0.3828 | 0.3843 | 0.0101 | 0.4007 |
| phys func | 0.1678 | -0.0378 | -0.2680 | 0.2319 | -0.1642 | 0.2733 |
| rol-phys | -0.0229 | -0.1143 | -0.0633 | 0.0301 | -0.1975 | 0.1331 |
| body pain | 0.1172 | -0.0724 | -0.1644 | -0.0506 | -0.1981 | -0.0024 |
| gen hlth | 0.1608 | 0.0471 | -0.1773 | 0.0608 | -0.1194 | 0.1536 |
| vitality | 0.0015 | -0.0527 | -0.1787 | 0.0575 | -0.2308 | 0.1154 |
| soc funct | 0.2568 | 0.1791 | -0.1059 | 0.2421 | -0.3406 | 0.3184 |
| rol-emot | 0.3963 | 0.2340 | -0.2233 | -0.1912 | -0.3215 | -0.0468 |
| ment hlth | 0.2175 | 0.0383 | -0.2911 | 0.0302 | 0.0977 | -0.0338 |
| | 0.0309 | 0.0362 | -0.1785 | 0.1095 | -0.2159 | 0.1258 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficient are shown for each of variables by systolic blood pressure, diastolic blood pressure, HbA1c%, total cholesterol, HDL cholesterol and LDL cholesterol respectively.

## Correlation coefficient of different variables in patients with type 1 diabetes* (continued)

| Variable | trig(mmol/l) | cre(umol/l) | alb(g/l) | fibr(g/l) | log alb/cre 1 | log alb/cre 2 |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| diab.yr | | | | | | |
| ins/bwt | | | | | | |
| BMI | | | | | | |
| B F% | | | | | | |
| sBP | | | | | | |
| dBP | | | | | | |
| HbA1c% | | | | | | |
| C (mmol/l) | | | | | | |
| HDL C (mmol/l) | | | | | | |
| LDL C (mmol/l) | | | | | | |
| trig (mmol/l) | | | | | | |
| cre(umol/l) | | | | | | |
| alb (g/l) | 0.3011 | | | | | |
| fibr (g/l) | 0.1859 | 0.2789 | | | | |
| log alb/cre1 | 0.4035 | -0.2046 | 0.3411 | | | |
| log alb/cre2 | 0.4267 | -0.1142 | 0.0600 | 0.2254 | | |
| Prot (En%) | 0.1203 | -0.2946 | -0.3086 | 0.2945 | 0.5414 | |
| Fat (En %) | 0.0124 | -0.1740 | 0.1571 | 0.2568 | 0.1399 | -0.0820 |
| CHO (En %) | -0.0615 | 0.2197 | 0.1605 | -0.1091 | -0.2925 | -0.1980 |
| Alc (En %) | -0.1571 | -0.4346 | -0.4642 | 0.0581 | 0.0623 | 0.2138 |
| PUFA(En%) | 0.3263 | 0.4297 | 0.3298 | -0.1027 | 0.2466 | 0.0417 |
| MUFA(En%) | -0.1424 | 0.1236 | 0.3579 | 0.1526 | -0.4355 | -0.3398 |
| SAFA(En%) | -0.0804 | 0.2190 | 0.1017 | -0.1548 | -0.3247 | -0.1120 |
| P/S | 0.0643 | 0.1645 | -0.0579 | -0.2360 | 0.0620 | -0.0248 |
| M/S | -0.2059 | 0.0836 | 0.3818 | 0.1720 | -0.3733 | -0.3048 |
| En (kJ) | -0.2088 | 0.1336 | 0.2948 | 0.0257 | -0.4845 | -0.2100 |
| Prot (g) | -0.2016 | 0.2203 | 0.1379 | -0.2805 | -0.5557 | -0.3650 |
| Prot/bwt | -0.1713 | 0.1253 | 0.2306 | -0.1663 | -0.4881 | -0.4476 |
| Fat(g) | -0.3103 | 0.0057 | 0.2708 | -0.1302 | -0.4051 | -0.4004 |
| CHO (g) | -0.1100 | 0.2196 | 0.1896 | -0.1264 | -0.4470 | -0.2370 |
| Alc (g) | -0.3823 | -0.0046 | -0.1493 | -0.3627 | -0.5945 | -0.3471 |
| Fibre(g) | 0.3292 | 0.4514 | 0.3353 | -0.1126 | 0.2232 | -0.0039 |
| Sugar (g) | -0.2419 | -0.0673 | -0.3659 | -0.4097 | -0.4247 | -0.3447 |
| Starch (g) | -0.1924 | -0.1858 | -0.1080 | 0.0430 | -0.3538 | -0.1760 |
| chol (mg) | -0.3625 | 0.0997 | -0.1242 | -0.4555 | -0.5612 | -0.3408 |
| Na (mg) | -0.0387 | 0.0111 | 0.3375 | 0.0684 | -0.3057 | -0.3505 |
| satisfaction | -0.2108 | 0.0220 | 0.0294 | -0.2279 | -0.1885 | -0.1290 |
| impact | -0.0075 | -0.2432 | 0.1821 | 0.1794 | -0.0542 | -0.0685 |
| soc wor | 0.1871 | -0.1038 | -0.0904 | 0.0430 | -0.0419 | 0.0999 |
| diab wor | 0.2671 | 0.0825 | -0.0323 | 0.1554 | -0.1607 | -0.0028 |
| total | -0.0511 | 0.2777 | 0.2176 | -0.0644 | -0.3259 | -0.2587 |
| phys func | 0.1166 | 0.0285 | 0.1069 | 0.0963 | -0.2065 | -0.0954 |
| rol-phys | -0.0200 | 0.0356 | -0.0190 | -0.1528 | -0.3020 | -0.4409 |
| body pain | 0.1367 | -0.0962 | 0.0212 | 0.0853 | -0.2186 | -0.2120 |
| gen hlth | -0.0990 | 0.0576 | -0.0325 | -0.2050 | -0.3929 | -0.2380 |
| vitality | 0.1574 | 0.1763 | 0.1198 | 0.1682 | -0.2737 | -0.2384 |
| soc funct | 0.2628 | -0.0232 | -0.0061 | 0.0030 | -0.1553 | -0.1086 |
| rol-emot | 0.0282 | -0.1131 | -0.0976 | -0.0680 | -0.0749 | -0.0042 |
| ment hlth | 0.0487 | -0.2061 | -0.0719 | -0.0142 | -0.2111 | -0.1174 |
| | 0.2579 | -0.1870 | -0.1020 | 0.2449 | -0.1125 | -0.0003 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by triglyceride, serum creatinine, serum albumin, fibrinogen, log of albumin: creatinine ratio in spot urine sample and log of albumin: creatinine ratio in early morning urine sample respectively.

## Correlation coefficient of different variables in patients with type 1 diabetes* (continued)

| Variable | Prot(En%) | Fat(En%) | CHO(En%) | Alc (energy%) | PUFA(En%) | MUFA(En%) |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| diab.yr | | | | | | |
| ins/bwt | | | | | | |
| BMI | | | | | | |
| B F% | | | | | | |
| sBP | | | | | | |
| dBP | | | | | | |
| HbA1c% | | | | | | |
| C (mmol/l) | | | | | | |
| HDL C (mmol/l) | | | | | | |
| LDL C (mmol/l) | | | | | | |
| trig (mmol/l) | | | | | | |
| cre(umol/l) | | | | | | |
| alb (g/l) | | | | | | |
| fibr (g/l) | | | | | | |
| log alb/cre1 | | | | | | |
| log alb/cre2 | | | | | | |
| Prot (En%) | | | | | | |
| Fat (En %) | | | | | | |
| CHO (En | -0.5081 | | | | | |
| Alc (En %) | 0.1671 | -0.7192 | | | | |
| PUFA(En%) | -0.1720 | -0.1325 | -0.4620 | | | |
| MUFA(En%) | 0.0768 | 0.5992 | -0.4418 | -0.3164 | | |
| SAFA(En%) | -0.5293 | 0.9473 | -0.6687 | -0.1092 | 0.4821 | |
| P/S | -0.6668 | 0.7324 | -0.5280 | 0.0980 | -0.0553 | 0.6738 |
| M/S | 0.3023 | 0.2195 | -0.1499 | -0.3100 | 0.8833 | 0.1459 |
| En (kJ) | 0.0714 | 0.4201 | -0.2868 | -0.2585 | 0.7286 | 0.5339 |
| Prot (g) | -0.3315 | 0.6099 | -0.4719 | -0.0091 | 0.6618 | 0.5718 |
| Prot/bwt | 0.2360 | 0.3557 | -0.4163 | -0.0999 | 0.7226 | 0.3058 |
| Fat(g) | 0.3328 | 0.1956 | -0.2264 | -0.1843 | 0.6129 | 0.2112 |
| CHO (g) | -0.3924 | 0.8703 | -0.6620 | -0.0941 | 0.7520 | 0.8135 |
| Alc (g) | -0.3145 | 0.2229 | 0.1097 | -0.2708 | 0.4009 | 0.2164 |
| Fibre(g) | -0.1930 | -0.0913 | -0.4935 | 0.9964 | -0.2944 | -0.0689 |
| Sugar (g) | -0.0145 | 0.1069 | 0.0778 | -0.2737 | 0.0576 | 0.0327 |
| Starch (g) | -0.3080 | 0.0030 | 0.2862 | -0.1851 | 0.1177 | -0.0674 |
| chol (mg) | -0.2349 | 0.2782 | -0.0152 | -0.2384 | 0.4378 | 0.3001 |
| Na (mg) | 0.1371 | 0.4251 | -0.5375 | 0.0177 | 0.4636 | 0.3393 |
| satisfaction | -0.3845 | 0.5213 | -0.3709 | 0.0181 | 0.4885 | 0.4920 |
| impact | 0.4654 | -0.1261 | 0.2214 | -0.4309 | 0.1753 | -0.1648 |
| soc wor | 0.1882 | -0.1176 | 0.0143 | 0.0443 | -0.1320 | -0.0273 |
| diab wor | 0.2725 | -0.1270 | 0.0031 | -0.0030 | 0.0898 | -0.0609 |
| total | 0.3233 | -0.1169 | -0.1961 | 0.2407 | 0.0425 | -0.0168 |
| phys func | 0.4130 | -0.1583 | 0.0010 | -0.0359 | 0.0681 | -0.0868 |
| rol-phys | 0.1263 | 0.0937 | -0.1212 | -0.0395 | 0.1995 | 0.0447 |
| body pain | 0.3443 | -0.1125 | 0.0856 | -0.1711 | -0.0529 | -0.0507 |
| gen hlth | 0.3057 | -0.0533 | -0.0124 | -0.1163 | -0.0150 | -0.0109 |
| vitality | 0.1988 | -0.0507 | 0.0345 | -0.1048 | 0.0342 | -0.0290 |
| soc funct | 0.3067 | -0.1513 | -0.0542 | 0.1096 | 0.0259 | -0.1915 |
| rol-emot | 0.6173 | -0.3861 | 0.1811 | -0.0885 | 0.1661 | -0.4079 |
| ment hlth | 0.4674 | -0.3364 | 0.2116 | -0.1086 | 0.0628 | -0.2461 |
| | 0.5071 | -0.3579 | 0.2008 | -0.0956 | -0.0628 | -0.3602 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by protein (energy%), fat (energy%), carbohydrate (energy%), alcohol (energy%), polyunsaturated fatty acids (energy%) and monounsaturated fatty acids (energy%) respectively.

## Correlation coefficient of different variables in patients with type 1 diabetes* (continued)

| Variable | SAFA(En%) | P/S | M/S | En (kJ) | Prot(g) | Prot/bwt |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| diab.yr | | | | | | |
| ins/bwt | | | | | | |
| BMI | | | | | | |
| B F% | | | | | | |
| sBP | | | | | | |
| dBP | | | | | | |
| HbA1c% | | | | | | |
| C (mmol/l) | | | | | | |
| HDL C (mmol/l) | | | | | | |
| LDL C (mmol/l) | | | | | | |
| trig (mmol/l) | | | | | | |
| cre(umol/l) | | | | | | |
| alb (g/l) | | | | | | |
| fibr (g/l) | | | | | | |
| log alb/cre1 | | | | | | |
| log alb/cre2 | | | | | | |
| Prot (En%) | | | | | | |
| Fat (En %) | | | | | | |
| CHO (En %) | | | | | | |
| Alc (En %) | | | | | | |
| PUFA(En%) | | | | | | |
| MUFA(En%) | | | | | | |
| SAFA(En%) | | | | | | |
| P/S | -0.4544 | | | | | |
| M/S | -0.2471 | 0.7716 | | | | |
| En (kJ) | 0.1898 | 0.5057 | 0.5739 | | | |
| Prot (g) | -0.1654 | 0.6898 | 0.6399 | 0.8246 | | |
| Prot/bwt | -0.3240 | 0.6765 | 0.6927 | 0.5944 | 0.8309 | |
| Fat(g) | 0.4427 | 0.4569 | 0.5787 | 0.8975 | 0.6932 | 0.4662 |
| CHO (g) | -0.0760 | 0.4181 | 0.4222 | 0.8105 | 0.6199 | 0.4839 |
| Alc (g) | 0.1293 | -0.3009 | -0.2382 | 0.0293 | -0.0743 | -0.1684 |
| Fibre(g) | 0.1555 | -0.0144 | -0.0983 | 0.3955 | 0.4045 | 0.2392 |
| Sugar (g) | -0.0275 | 0.0833 | -0.0371 | 0.4041 | 0.2045 | 0.1889 |
| Starch (g) | -0.0718 | 0.4705 | 0.5281 | 0.7986 | 0.6612 | 0.4821 |
| chol (mg) | 0.1840 | 0.3043 | 0.2857 | 0.5998 | 0.7483 | 0.5593 |
| Na (mg) | 0.2209 | 0.3708 | 0.4286 | 0.7781 | 0.5602 | 0.4941 |
| satisfaction | -0.2578 | 0.2697 | 0.1225 | -0.1098 | 0.1679 | 0.1794 |
| impact | -0.1057 | -0.0463 | 0.0784 | -0.0278 | 0.1151 | 0.0423 |
| soc wor | -0.2974 | 0.1816 | 0.2558 | 0.2751 | 0.4779 | 0.3126 |
| diab wor | -0.2709 | 0.1867 | 0.3006 | 0.0346 | 0.2594 | 0.2661 |
| total | -0.3133 | 0.2065 | 0.2602 | 0.0631 | 0.3431 | 0.2749 |
| phys func | -0.0116 | 0.2178 | 0.1161 | 0.1028 | 0.1905 | 0.0201 |
| rol-phys | -0.1341 | 0.0001 | 0.0935 | -0.1690 | 0.0519 | -0.0214 |
| body pain | -0.0763 | -0.0294 | 0.0513 | 0.0026 | 0.1895 | 0.0933 |
| gen hlth | -0.1054 | 0.1012 | 0.1032 | -0.0419 | 0.0713 | -0.0734 |
| vitality | -0.1759 | 0.0433 | -0.0698 | 0.1506 | 0.3563 | 0.1406 |
| soc funct | -0.5879 | 0.3326 | 0.1252 | 0.1076 | 0.4389 | 0.3109 |
| rol-emot | -0.5479 | 0.2087 | 0.2690 | 0.0518 | 0.3615 | 0.3604 |
| ment hlth | -0.3863 | 0.0228 | -0.0843 | -0.1678 | 0.1462 | 0.0201 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by saturated fatty acids (energy%), polyunsaturated to saturated fatty acids ratio, monounsaturated fatty acids to saturated fatty acids ratio, energy (kJ), protein (g) and protein (g)/body weight (kg) respectively.

## Correlation coefficient of different variables in patients with type 1 diabetes* (continued)

| Variable | Fat(g) | CHO (g) | Alc(g) | Fibre(g) | Sugar(g) | Starch(g) |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| diab.yr | | | | | | |
| ins/bwt | | | | | | |
| BMI | | | | | | |
| B F% | | | | | | |
| sBP | | | | | | |
| dBP | | | | | | |
| HbA1c% | | | | | | |
| C (mmol/l) | | | | | | |
| HDL C (mmol/l | | | | | | |
| LDL C (mmol/l | | | | | | |
| trig (mmol/l) | | | | | | |
| cre(umol/l) | | | | | | |
| alb (g/l) | | | | | | |
| fibr (g/l) | | | | | | |
| log alb/cre1 | | | | | | |
| log alb/cre2 | | | | | | |
| Prot (En%) | | | | | | |
| Fat (En %) | | | | | | |
| CHO (En %) | | | | | | |
| Alc (En %) | | | | | | |
| PUFA(En%) | | | | | | |
| MUFA(En%) | | | | | | |
| SAFA(En%) | | | | | | |
| P/S | | | | | | |
| M/S | | | | | | |
| En (kJ) | | | | | | |
| Prot (g) | | | | | | |
| Prot/bwt | | | | | | |
| Fat(g) | | | | | | |
| CHO (g) | 0.5505 | | | | | |
| Alc (g) | -0.0552 | -0.2433 | | | | |
| Fibre(g) | 0.2561 | 0.5069 | -0.2449 | | | |
| Sugar (g) | 0.2311 | 0.6283 | -0.1773 | 0.4437 | | |
| Starch (g) | 0.5680 | 0.9143 | -0.2080 | 0.3974 | 0.2625 | |
| chol (mg) | 0.5986 | 0.2949 | 0.0382 | 0.4228 | 0.2263 | 0.2561 |
| Na (mg) | 0.7055 | 0.6412 | 0.0545 | 0.0655 | 0.2293 | 0.6697 |
| satisfaction | -0.0847 | -0.0492 | -0.4423 | 0.1748 | 0.0118 | -0.0617 |
| impact | -0.0705 | -0.0592 | 0.0532 | 0.3268 | -0.0961 | -0.0271 |
| soc wor | 0.1335 | 0.2517 | 0.0007 | 0.4177 | 0.2491 | 0.1826 |
| diab wor | -0.0335 | -0.1013 | 0.2502 | 0.1767 | -0.2556 | 0.0016 |
| total | -0.0135 | 0.0146 | -0.0323 | 0.3488 | -0.0351 | 0.0350 |
| phys func | 0.0630 | 0.0810 | -0.0104 | 0.4239 | -0.0606 | 0.1271 |
| rol-phys | -0.1663 | -0.1424 | -0.1610 | 0.2501 | -0.0273 | -0.1629 |
| body pain | -0.0029 | -0.0332 | -0.1129 | 0.5173 | -0.0150 | -0.0280 |
| gen hlth | -0.0450 | -0.0431 | -0.0818 | 0.3328 | -0.0569 | -0.0075 |
| vitality | 0.0509 | 0.0611 | 0.1182 | 0.4113 | 0.0327 | 0.0851 |
| soc funct | -0.0743 | 0.1731 | -0.1034 | 0.2728 | 0.1130 | 0.1521 |
| rol-emot | -0.1017 | 0.1302 | -0.1204 | 0.1770 | 0.2188 | 0.0348 |
| ment hlth | -0.2195 | -0.1480 | -0.1057 | 0.2947 | -0.0238 | -0.1510 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by fat (g), carbohydrate (g), alcohol (g), fibre (g), sugar (g) and starch (g) respectively.

**Correlation coefficient of different variables in patients with type 1 diabetes* (continued)**

| Variable | chol(mg) | Na (mg) | satisfaction | impact | soc wor | diab wor |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| diab.yr | | | | | | |
| ins/bwt | | | | | | |
| BMI | | | | | | |
| B F% | | | | | | |
| sBP | | | | | | |
| dBP | | | | | | |
| HbA1c% | | | | | | |
| C (mmol/l) | | | | | | |
| HDL C (mmol/l) | | | | | | |
| LDL C (mmol/l) | | | | | | |
| trig (mmol/l) | | | | | | |
| cre(umol/l) | | | | | | |
| alb (g/l) | | | | | | |
| fibr (g/l) | | | | | | |
| log alb/cre1 | | | | | | |
| log alb/cre2 | | | | | | |
| Prot (En%) | | | | | | |
| Fat (En %) | | | | | | |
| CHO (En %) | | | | | | |
| Alc (En %) | | | | | | |
| PUFA(En%) | | | | | | |
| MUFA(En%) | | | | | | |
| SAFA(En%) | | | | | | |
| P/S | | | | | | |
| M/S | | | | | | |
| En (kJ) | | | | | | |
| Prot (g) | | | | | | |
| Prot/bwt | | | | | | |
| Fat(g) | | | | | | |
| CHO (g) | | | | | | |
| Alc (g) | | | | | | |
| Fibre(g) | | | | | | |
| Sugar (g) | | | | | | |
| Starch (g) | | | | | | |
| chol (mg) | | | | | | |
| Na (mg) | 0.2855 | | | | | |
| satisfaction | 0.1694 | -0.2588 | | | | |
| impact | 0.2003 | -0.2004 | 0.5775 | | | |
| soc wor | 0.5298 | -0.1151 | 0.2839 | 0.6084 | | |
| diab wor | 0.3030 | -0.2834 | 0.2753 | 0.6218 | 0.4538 | |
| total | 0.3999 | -0.2819 | 0.6715 | 0.8872 | 0.7583 | 0.7839 |
| phys func | 0.1041 | -0.1409 | 0.3308 | 0.5841 | 0.2213 | 0.4829 |
| rol-phys | 0.1215 | -0.4362 | 0.7021 | 0.7433 | 0.4442 | 0.4820 |
| body pain | 0.2930 | -0.4970 | 0.4936 | 0.5631 | 0.4376 | 0.6750 |
| gen hlth | 0.1581 | -0.4118 | 0.5875 | 0.7510 | 0.4823 | 0.6772 |
| vitality | 0.3769 | -0.1241 | 0.4625 | 0.6061 | 0.5064 | 0.4135 |
| soc funct | 0.1333 | -0.0766 | 0.4080 | 0.2746 | 0.4410 | 0.1735 |
| rol-emot | 0.2242 | -0.2003 | 0.4028 | 0.3606 | 0.6203 | 0.3347 |
| ment hlth | 0.2093 | -0.4599 | 0.5811 | 0.6485 | 0.5796 | 0.4631 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by cholesterol (mg), sodium (mg), satisfaction, impact, social worry and diabetes worry respectively.

**Correlation coefficient of different variables in patients with type 1 diabetes* (continued)**

| Variable | total | phy fun | role-phy | body pain | gen hlth | vitality |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| diab.yr | | | | | | |
| ins/bwt | | | | | | |
| BMI | | | | | | |
| B F% | | | | | | |
| sBP | | | | | | |
| dBP | | | | | | |
| HbA1c% | | | | | | |
| C (mmol/l) | | | | | | |
| HDL C (mmol/l) | | | | | | |
| LDL C (mmol/l) | | | | | | |
| trig (mmol/l) | | | | | | |
| cre(umol/l) | | | | | | |
| alb (g/l) | | | | | | |
| fibr (g/l) | | | | | | |
| log alb/cre1 | | | | | | |
| log alb/cre2 | | | | | | |
| Prot (En%) | | | | | | |
| Fat (En %) | | | | | | |
| CHO (En %) | | | | | | |
| Alc (En %) | | | | | | |
| PUFA(En%) | | | | | | |
| MUFA(En%) | | | | | | |
| SAFA(En%) | | | | | | |
| P/S | | | | | | |
| M/S | | | | | | |
| En (kJ) | | | | | | |
| Prot (g) | | | | | | |
| Prot/bwt | | | | | | |
| Fat(g) | | | | | | |
| CHO (g) | | | | | | |
| Alc (g) | | | | | | |
| Fibre(g) | | | | | | |
| Sugar (g) | | | | | | |
| Starch (g) | | | | | | |
| chol (mg) | | | | | | |
| Na (mg) | | | | | | |
| satisfaction | | | | | | |
| impact | | | | | | |
| soc wor | | | | | | |
| diab wor | | | | | | |
| total | | | | | | |
| phys func | 0.5168 | | | | | |
| rol-phys | 0.7518 | 0.6268 | | | | |
| body pain | 0.7093 | 0.4685 | 0.6591 | | | |
| gen hlth | 0.8042 | 0.6754 | 0.7347 | 0.6857 | | |
| vitality | 0.6339 | 0.3056 | 0.4603 | 0.5807 | 0.5417 | |
| soc funct | 0.4166 | 0.1788 | 0.3912 | 0.3946 | 0.1147 | 0.5154 |
| rol-emot | 0.5590 | 0.0954 | 0.5276 | 0.4640 | 0.1992 | 0.4290 |
| ment hlth | 0.7265 | 0.3498 | 0.5867 | 0.6178 | 0.6697 | 0.8218 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by total subscale, physical functioning, role-physical, bodily pain, general health and vitality respectively.

**Correlation coefficient of different variables in patients with type 1 diabetes* (continued)**

| Variable | soc func | role-emo | ment hlth |
|---|---|---|---|
| Age | | | |
| diab.yr | | | |
| ins/bwt | | | |
| BMI | | | |
| B F% | | | |
| sBP | | | |
| dBP | | | |
| HbA1c% | | | |
| C (mmol/l) | | | |
| HDL C (mmol/l) | | | |
| LDL C (mmol/l) | | | |
| trig (mmol/l) | | | |
| cre(umol/l) | | | |
| alb (g/l) | | | |
| fibr (g/l) | | | |
| log alb/cre1 | | | |
| log alb/cre2 | | | |
| Prot (En%) | | | |
| Fat (En %) | | | |
| CHO (En %) | | | |
| Alc (En %) | | | |
| PUFA(En%) | | | |
| MUFA(En%) | | | |
| SAFA(En%) | | | |
| P/S | | | |
| M/S | | | |
| En (kJ) | | | |
| Prot (g) | | | |
| Prot/bwt | | | |
| Fat(g) | | | |
| CHO (g) | | | |
| Alc (g) | | | |
| Fibre(g) | | | |
| Sugar (g) | | | |
| Starch (g) | | | |
| chol (mg) | | | |
| Na (mg) | | | |
| satisfaction | | | |
| impact | | | |
| soc wor | | | |
| diab wor | | | |
| total | | | |
| phys func | | | |
| rol-phys | | | |
| body pain | | | |
| gen hlth | | | |
| vitality | | | |
| soc funct | | | |
| rol-emot | 0.6897 | | |
| ment hlth | 0.4415 | 0.5588 | |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by social functioning, role-emotional and mental health respectively.

## Appendix 32: Correlation coefficient of different variables in control subjects*

| Variable | Age | BMI | BF% | WHR | sBP | dBP |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| BMI | -0.0449 | | | | | |
| BF% | 0.1362 | 0.3593 | | | | |
| WHR | 0.0571 | 0.2601 | -0.3649 | | | |
| sBP | 0.0535 | 0.2610 | -0.0932 | 0.1220 | | |
| dBP | 0.1586 | 0.1490 | -0.1532 | 0.1520 | 0.4713 | |
| HbA1c% | -0.1765 | 0.1082 | 0.0826 | 0.2945 | -0.0884 | -0.1586 |
| C(mmol/ l) | 0.0908 | 0.1318 | -0.1536 | 0.3617 | 0.1712 | 0.0969 |
| HDL C(mmol/ l) | 0.3795 | -0.4260 | 0.2274 | -0.5390 | -0.1272 | 0.0710 |
| LDL C(mmol/ l) | 0.0424 | 0.2797 | -0.1387 | 0.4389 | 0.2317 | 0.0403 |
| trig(mmol/ l) | -0.1883 | 0.0693 | -0.2635 | 0.3789 | 0.0201 | 0.0896 |
| cre(umol/ l) | 0.0061 | 0.2812 | -0.4941 | 0.2188 | 0.5252 | 0.1653 |
| alb(g/ l) | -0.0908 | -0.0473 | -0.1470 | 0.0693 | 0.3475 | -0.0266 |
| fibr(g/ l) | -0.2486 | 0.1927 | 0.4554 | -0.4157 | 0.0613 | -0.3389 |
| log alb/ cre 1 | -0.1435 | -0.1118 | 0.2948 | -0.4918 | 0.0791 | -0.0204 |
| log alb/ cre 2 | -0.1268 | -0.2281 | -0.2634 | -0.0495 | -0.1231 | 0.0408 |
| Prot(En%) | -0.2017 | 0.0510 | 0.1433 | -0.0549 | 0.1105 | 0.1597 |
| Fat(En%) | 0.1144 | 0.1182 | -0.0345 | 0.5071 | -0.0400 | 0.0375 |
| CHO(En%) | -0.0306 | -0.2286 | -0.1737 | -0.4119 | 0.0453 | 0.1903 |
| Alc (energy%) | 0.0139 | 0.1252 | 0.2055 | -0.0746 | -0.0973 | -0.4560 |
| PUFA(En%) | 0.0990 | -0.2029 | 0.0579 | 0.1117 | -0.1528 | 0.0274 |
| MUFA(En%) | 0.1196 | 0.1206 | -0.1588 | 0.5124 | 0.0565 | 0.0929 |
| SAFA(En%) | 0.0625 | 0.2096 | 0.0286 | 0.4166 | -0.0389 | -0.0183 |
| P/ S | 0.0575 | -0.2993 | 0.0509 | -0.1991 | -0.0912 | 0.0319 |
| M/ S | 0.0228 | -0.0223 | -0.1399 | 0.0186 | 0.1404 | 0.2692 |
| En (kJ) | -0.1145 | 0.1498 | -0.6126 | 0.6375 | 0.2900 | 0.2953 |
| Prot(g) | -0.2349 | 0.1549 | -0.4339 | 0.5744 | 0.2961 | 0.3355 |
| Prot/ bwt | -0.2180 | -0.3312 | -0.4632 | 0.3090 | 0.1579 | 0.2583 |
| Fat(g) | -0.0118 | 0.1678 | -0.4579 | 0.7114 | 0.2168 | 0.2474 |
| CHO (g) | -0.1321 | 0.0472 | -0.6559 | 0.3769 | 0.2767 | 0.3364 |
| Alc(g) | 0.0325 | 0.1141 | 0.1257 | -0.0256 | -0.0715 | -0.4302 |
| Fibre(g) | -0.0393 | 0.0849 | -0.1614 | 0.0693 | 0.1000 | 0.1615 |
| Sugar(g) | -0.1507 | -0.0113 | -0.4929 | 0.1674 | 0.0350 | 0.0010 |
| Starch(g) | -0.0590 | 0.1013 | -0.5316 | 0.4335 | 0.4066 | 0.5298 |
| chol(mg) | 0.0222 | -0.0157 | -0.3107 | 0.5317 | 0.1019 | 0.1145 |
| Na (mg) | 0.1401 | 0.0697 | -0.3105 | 0.4161 | 0.0741 | 0.4197 |
| phy fun | 0.1681 | -0.6613 | -0.5319 | 0.0076 | 0.0627 | 0.0690 |
| role-phy | 0.2941 | -0.3156 | -0.3951 | 0.1865 | -0.0819 | 0.1643 |
| body pain | 0.0102 | -0.1846 | -0.3019 | 0.2456 | 0.2015 | 0.1893 |
| gen hlth | 0.1887 | -0.3641 | -0.3449 | -0.1784 | -0.1437 | -0.1295 |
| vitality | 0.0761 | -0.1707 | -0.1474 | 0.2799 | -0.0128 | 0.0497 |
| soc func | 0.2798 | -0.4135 | -0.3639 | 0.0047 | 0.2563 | 0.2924 |
| role-emo | 0.0636 | -0.1284 | -0.0885 | 0.2139 | 0.1671 | -0.0469 |
| ment hlth | 0.0834 | -0.1893 | -0.0918 | 0.0470 | 0.2747 | 0.3233 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by age, body mass index, body fat%, waist to hip ratio, systolic blood pressure and diastolic blood pressure respectively.

## Correlation coefficient of different variables in control subjects (continued)*

| Variable | HbA1c% | C(mmol/l) | HDL C(mmol/l) | LDL C(mmol/l) | trig(mmol/l) | cre(umol/l) | alb(g/l) |
|---|---|---|---|---|---|---|---|
| Age | | | | | | | |
| BMI | | | | | | | |
| B F% | | | | | | | |
| WHR | | | | | | | |
| sBP | | | | | | | |
| dBP | | | | | | | |
| HbA1c% | | | | | | | |
| C (mmol/l) | 0.0923 | | | | | | |
| HDL C (mmol/l) | -0.0173 | -0.0427 | | | | | |
| LDL C (mmol/l) | 0.0793 | 0.8938 | -0.2951 | | | | |
| trig (mmol/l) | 0.0671 | 0.5319 | -0.2987 | 0.2524 | | | |
| cre(umol/l) | -0.1537 | -0.0375 | -0.3884 | 0.1189 | -0.0254 | | |
| alb (g/l) | -0.2205 | -0.0562 | -0.1338 | 0.1239 | -0.2953 | 0.3572 | |
| fibr (g/l) | -0.1390 | -0.0424 | -0.0132 | 0.0236 | -0.1441 | -0.1630 | 0.1523 |
| log alb/cre1 | -0.2064 | -0.1062 | 0.3777 | -0.0829 | -0.3952 | -0.2350 | 0.1840 |
| log alb/cre2 | -0.4905 | -0.0672 | -0.1021 | -0.0090 | -0.0590 | -0.0601 | 0.1795 |
| Prot (En%) | 0.1196 | 0.2826 | -0.0363 | 0.1517 | 0.3905 | -0.1791 | 0.0689 |
| Fat (En %) | 0.0493 | -0.1090 | -0.2701 | -0.0345 | 0.0349 | -0.0372 | 0.3270 |
| CHO (En %) | -0.1615 | -0.0242 | 0.4423 | -0.1008 | -0.2099 | -0.0115 | -0.3610 |
| Alc (En %) | 0.0813 | -0.0271 | -0.2444 | 0.0696 | -0.0132 | 0.1756 | 0.0013 |
| PUFA(En%) | 0.4626 | 0.1073 | 0.2848 | -0.1765 | 0.4083 | -0.3957 | -0.1695 |
| MUFA(En%) | -0.0521 | -0.1742 | -0.2970 | -0.0421 | -0.0854 | 0.1330 | 0.2841 |
| SAFA(En%) | -0.1162 | -0.1090 | -0.3754 | 0.0624 | -0.0880 | 0.0346 | 0.4283 |
| P/S | 0.4075 | 0.1996 | 0.5067 | -0.1403 | 0.3692 | -0.3662 | -0.3860 |
| M/S | 0.1024 | -0.0389 | 0.1476 | -0.1029 | 0.0056 | 0.0903 | -0.2651 |
| En (kJ) | -0.0769 | 0.0914 | -0.5536 | 0.1444 | 0.3725 | 0.4572 | 0.1348 |
| Prot (g) | 0.0402 | 0.2775 | -0.5227 | 0.2438 | 0.5847 | 0.2759 | 0.2039 |
| Prot/bwt | -0.0053 | 0.1759 | -0.2052 | 0.0363 | 0.5227 | 0.0494 | 0.1746 |
| Fat(g) | 0.0065 | 0.0193 | -0.5424 | 0.1071 | 0.2680 | 0.3068 | 0.2401 |
| CHO (g) | -0.2001 | 0.0322 | -0.3128 | 0.0508 | 0.2298 | 0.4513 | -0.0342 |
| Alc (g) | 0.0574 | -0.0134 | -0.2787 | 0.0807 | 0.0248 | 0.2276 | 0.0027 |
| Fibre(g) | -0.2497 | 0.1091 | -0.2176 | 0.1209 | 0.1853 | 0.0345 | -0.0475 |
| Sugar (g) | -0.2151 | -0.0991 | -0.3752 | 0.0126 | 0.0445 | 0.3331 | -0.0002 |
| Starch (g) | -0.0971 | 0.1430 | -0.1244 | 0.0696 | 0.3019 | 0.3892 | -0.0410 |
| chol (mg) | -0.0053 | 0.3390 | -0.3772 | 0.4213 | 0.2264 | 0.0696 | 0.3090 |
| Na (mg) | -0.2070 | 0.0771 | -0.1990 | 0.0219 | 0.3074 | 0.0979 | 0.0427 |
| phys func | -0.0305 | -0.0488 | 0.3878 | -0.1618 | -0.0915 | -0.0114 | 0.0359 |
| rol-phys | 0.2115 | -0.0209 | 0.0743 | -0.1006 | 0.1057 | 0.0492 | -0.1970 |
| body pain | -0.0443 | 0.0966 | -0.3094 | 0.3045 | -0.1684 | 0.1927 | 0.1230 |
| gen hlth | -0.1230 | -0.1002 | 0.2615 | -0.1899 | -0.0501 | 0.1640 | -0.1804 |
| vitality | 0.0860 | 0.0222 | -0.0564 | 0.0683 | -0.0472 | 0.1486 | 0.2455 |
| soc funct | -0.1648 | 0.2143 | 0.2127 | 0.1516 | 0.0153 | 0.1509 | 0.0886 |
| rol-emot | -0.3236 | 0.2312 | -0.1049 | 0.2160 | 0.1821 | 0.0576 | 0.3033 |
| ment hlth | -0.1278 | 0.2671 | 0.0476 | 0.2229 | 0.1274 | -0.0020 | 0.2574 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by HbA1c%, total cholesterol, HDL cholesterol, LDL cholesterol, triglyceride, serum creatinine and serum albumin respectively.

## Correlation coefficient of different variables in control subjects (continued)*

| Variable | fibr(g/l) | log alb/cre 1 | log alb/cre 2 | Prot (En%) | Fat (En%) | CHO (En%) |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| BMI | | | | | | |
| B F% | | | | | | |
| WHR | | | | | | |
| sBP | | | | | | |
| dBP | | | | | | |
| HbA1c% | | | | | | |
| C (mmol/l) | | | | | | |
| HDL C (mmol/l) | | | | | | |
| LDL C (mmol/l) | | | | | | |
| trig (mmol/l) | | | | | | |
| cre(umol/l) | | | | | | |
| alb (g/l) | | | | | | |
| fibr (g/l) | | | | | | |
| log alb/cre1 | 0.4494 | | | | | |
| log alb/cre2 | -0.0482 | 0.3678 | | | | |
| Prot (En%) | 0.1530 | -0.1020 | -0.2705 | | | |
| Fat (En %) | -0.1917 | -0.0714 | 0.1448 | -0.1841 | | |
| CHO (En %) | 0.0270 | 0.2399 | 0.0876 | -0.2564 | -0.6668 | |
| Alc (En %) | 0.1167 | -0.1543 | -0.1069 | -0.1443 | -0.2723 | -0.3403 |
| PUFA(En%) | -0.0158 | -0.0387 | -0.3168 | 0.1984 | 0.2813 | -0.2590 |
| MUFA(En%) | -0.3749 | -0.0863 | 0.2948 | -0.3066 | 0.8714 | -0.5526 |
| SAFA(En%) | -0.0380 | -0.0387 | 0.1893 | -0.1898 | 0.9010 | -0.5813 |
| P/S | 0.0023 | 0.0076 | -0.3918 | 0.3148 | -0.3589 | 0.1922 |
| M/S | -0.4075 | -0.0057 | 0.1103 | -0.0985 | -0.1950 | 0.1309 |
| En (kJ) | -0.3165 | -0.3321 | 0.3170 | -0.2062 | 0.3865 | -0.1896 |
| Prot (g) | -0.1667 | -0.3539 | 0.0990 | 0.4618 | 0.2665 | -0.4016 |
| Prot/bwt | -0.2048 | -0.2437 | 0.1151 | 0.5069 | 0.1138 | -0.2507 |
| Fat(g) | -0.3605 | -0.3232 | 0.2549 | -0.2033 | 0.7292 | -0.4474 |
| CHO (g) | -0.2456 | -0.1398 | 0.4009 | -0.4022 | 0.0632 | 0.3149 |
| Alc (g) | 0.0675 | -0.2212 | -0.1037 | -0.1694 | -0.2210 | -0.3665 |
| Fibre(g) | 0.1445 | -0.3317 | -0.1821 | -0.0054 | -0.1106 | 0.0408 |
| Sugar (g) | -0.0885 | -0.0719 | 0.4898 | -0.5180 | 0.0761 | 0.2548 |
| Starch (g) | -0.2941 | -0.1439 | 0.1371 | -0.1133 | 0.0340 | 0.2310 |
| chol (mg) | -0.1606 | -0.3052 | 0.2289 | 0.0892 | 0.4330 | -0.3741 |
| Na (mg) | -0.3719 | -0.2463 | 0.1478 | 0.1161 | 0.3594 | -0.2437 |
| phys func | -0.3450 | 0.0704 | 0.3835 | -0.2531 | 0.0301 | 0.2790 |
| rol-phys | -0.3988 | -0.2392 | 0.0830 | -0.2083 | -0.1471 | 0.1439 |
| body pain | -0.1707 | 0.0975 | 0.1634 | -0.2429 | -0.0160 | 0.0571 |
| gen hlth | -0.1839 | -0.2030 | -0.0078 | -0.2300 | -0.2589 | 0.2913 |
| vitality | -0.2313 | 0.0778 | 0.2241 | -0.2953 | 0.2364 | -0.2876 |
| soc funct | -0.4390 | 0.1613 | 0.3753 | -0.0742 | -0.0918 | 0.1242 |
| rol-emot | -0.0808 | 0.2126 | 0.4524 | -0.0208 | 0.3285 | -0.3886 |
| ment hlth | -0.1849 | 0.2100 | 0.1642 | 0.1008 | -0.0397 | -0.0510 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by fibrinogen, log of albumin: creatinine ratio in spot urine sample, log of albumin: creatinine ratio in early morning urine sample, protein (energy%), fat (energy%) and carbohydrate (energy%) resspectively.

## Correlation coefficient of different variables in control subjects (continued)*

| Variable | Alc (energy%) | PUFA(En%) | MUFA(En%) | SAFA(En%) | P/S | M/S |
|---|---|---|---|---|---|---|
| Age | | | | | | |
| BMI | | | | | | |
| B F% | | | | | | |
| WHR | | | | | | |
| sBP | | | | | | |
| dBP | | | | | | |
| HbA1c% | | | | | | |
| C (mmol/l) | | | | | | |
| HDL C (mmol/l) | | | | | | |
| LDL C (mmol/l) | | | | | | |
| trig (mmol/l) | | | | | | |
| cre(umol/l) | | | | | | |
| alb (g/l) | | | | | | |
| fibr (g/l) | | | | | | |
| log alb/cre1 | | | | | | |
| log alb/cre2 | | | | | | |
| Prot (En%) | | | | | | |
| Fat (En %) | | | | | | |
| CHO (En %) | | | | | | |
| Alc (En %) | | | | | | |
| PUFA(En%) | -0.1361 | | | | | |
| MUFA(En%) | -0.1705 | -0.0007 | | | | |
| SAFA(En%) | -0.2672 | -0.0299 | 0.7285 | | | |
| P/S | 0.0073 | 0.7639 | -0.4968 | -0.6255 | | |
| M/S | 0.1712 | -0.0061 | 0.2055 | -0.4869 | 0.2300 | |
| En (kJ) | -0.0907 | -0.0583 | 0.4603 | 0.3357 | -0.2828 | 0.0782 |
| Prot (g) | -0.1341 | 0.0899 | 0.2436 | 0.2164 | -0.0672 | -0.0095 |
| Prot/bwt | -0.1718 | 0.2079 | 0.0571 | 0.0388 | 0.1557 | -0.0305 |
| Fat(g) | -0.1961 | 0.0742 | 0.7247 | 0.6535 | -0.3654 | -0.0407 |
| CHO (g) | -0.2284 | -0.1929 | 0.1903 | 0.0599 | -0.2124 | 0.1347 |
| Alc (g) | 0.9875 | -0.1183 | -0.1085 | -0.2362 | -0.0001 | 0.1910 |
| Fibre(g) | 0.0952 | -0.0166 | -0.1972 | -0.0229 | -0.0363 | -0.2497 |
| Sugar (g) | -0.0704 | -0.3463 | 0.1841 | 0.1670 | -0.4020 | -0.0092 |
| Starch (g) | -0.2931 | 0.0329 | 0.1257 | -0.0574 | 0.0481 | 0.2110 |
| chol (mg) | -0.1365 | -0.0765 | 0.3928 | 0.4855 | -0.3363 | -0.1709 |
| Na (mg) | -0.2342 | 0.1908 | 0.3174 | 0.2779 | -0.0351 | -0.0717 |
| phys func | -0.2603 | 0.0666 | 0.2147 | -0.1371 | 0.1001 | 0.3535 |
| rol-phys | 0.1532 | 0.2781 | -0.0569 | -0.3397 | 0.3328 | 0.3336 |
| body pain | 0.1240 | -0.2102 | 0.1236 | -0.0144 | -0.1708 | 0.1984 |
| gen hlth | 0.0899 | -0.0177 | -0.1089 | -0.3405 | 0.1023 | 0.1923 |
| vitality | 0.3245 | 0.1930 | 0.3254 | 0.0631 | 0.0326 | 0.3103 |
| soc funct | 0.0015 | -0.0235 | 0.1131 | -0.2237 | 0.0633 | 0.3947 |
| rol-emot | 0.1301 | -0.0016 | 0.3795 | 0.2693 | -0.2407 | 0.0098 |
| ment hlth | 0.0589 | 0.1396 | -0.1039 | -0.0594 | 0.1164 | -0.0293 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by alcohol (energy%), polyunsaturated fatty acids (energy%), monounsaturated fatty acids, saturated fatty acids (energy%), polyunsaturated fatty acids to saturated fatty acids ratio and monunsaturated fatty acids to saturated fatty acids ratio respectively.

## Correlation coefficient of different variables in control subjects (continued)*

| Variable | En (kJ) | Prot(g) | Prot/bwt | Fat(g) | CHO (g) | Alc(g) | Fibre(g) |
|---|---|---|---|---|---|---|---|
| Age | | | | | | | |
| BMI | | | | | | | |
| B F% | | | | | | | |
| WHR | | | | | | | |
| sBP | | | | | | | |
| dBP | | | | | | | |
| HbA1c% | | | | | | | |
| C (mmol/l) | | | | | | | |
| HDL C (mmol/l) | | | | | | | |
| LDL C (mmol/l) | | | | | | | |
| trig (mmol/l) | | | | | | | |
| cre(umol/l) | | | | | | | |
| alb (g/l) | | | | | | | |
| fibr (g/l) | | | | | | | |
| log alb/cre1 | | | | | | | |
| log alb/cre2 | | | | | | | |
| Prot (En%) | | | | | | | |
| Fat (En %) | | | | | | | |
| CHO (En %) | | | | | | | |
| Alc (En %) | | | | | | | |
| PUFA(En%) | | | | | | | |
| MUFA(En%) | | | | | | | |
| SAFA(En%) | | | | | | | |
| P/S | | | | | | | |
| M/S | | | | | | | |
| En (kJ) | | | | | | | |
| Prot (g) | 0.7581 | | | | | | |
| Prot/bwt | 0.5514 | 0.8487 | | | | | |
| Fat(g) | 0.8989 | 0.6817 | 0.4612 | | | | |
| CHO (g) | 0.8587 | 0.4723 | 0.3389 | 0.6276 | | | |
| Alc (g) | 0.0034 | -0.0752 | -0.1250 | -0.1001 | -0.1565 | | |
| Fibre(g) | 0.2115 | 0.1824 | 0.1673 | 0.1213 | 0.1954 | 0.1305 | |
| Sugar (g) | 0.6234 | 0.2075 | 0.1119 | 0.4415 | 0.7909 | -0.0459 | 0.0997 |
| Starch (g) | 0.7230 | 0.5329 | 0.4105 | 0.5491 | 0.7721 | -0.2041 | 0.2036 |
| chol (mg) | 0.5016 | 0.5518 | 0.5116 | 0.5791 | 0.2568 | -0.0987 | 0.1432 |
| Na (mg) | 0.5559 | 0.5497 | 0.4652 | 0.6084 | 0.3546 | -0.1452 | 0.3640 |
| phys func | 0.1939 | 0.0107 | 0.2823 | 0.1389 | 0.3189 | -0.2227 | -0.1543 |
| rol-phys | 0.2971 | 0.1437 | 0.2493 | 0.1656 | 0.3260 | 0.1793 | 0.1617 |
| body pain | 0.2585 | 0.0958 | 0.1159 | 0.2037 | 0.2595 | 0.1254 | 0.1356 |
| gen hlth | -0.0154 | -0.1586 | 0.0434 | -0.1691 | 0.1574 | 0.1053 | 0.3184 |
| vitality | 0.1960 | 0.0647 | 0.0958 | 0.2235 | 0.0636 | 0.3218 | -0.0504 |
| soc funct | 0.1477 | 0.0861 | 0.2776 | 0.0485 | 0.2069 | 0.0101 | -0.0638 |
| rol-emot | 0.2373 | 0.2296 | 0.2665 | 0.2689 | 0.0892 | 0.1416 | 0.0247 |
| ment hlth | 0.0105 | 0.0782 | 0.2011 | -0.0276 | 0.0110 | 0.0201 | 0.1800 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by energy (kJ), protein (g), protein (g)/ body weight (kg), fat (g), carbohydrate (g), alcohol (g), fibre (g) respectively.

## Correlation coefficient of different variables in control subjects (continued)*

| Variable | Sugar(g) | Starch(g) | chol(mg) | Na (mg) | phy fun | role-phy | body pain |
|---|---|---|---|---|---|---|---|
| Age | | | | | | | |
| BMI | | | | | | | |
| B F% | | | | | | | |
| WHR | | | | | | | |
| sBP | | | | | | | |
| dBP | | | | | | | |
| HbA1c% | | | | | | | |
| C (mmol/ l) | | | | | | | |
| HDL C (mmol/ l) | | | | | | | |
| LDL C (mmol/ l) | | | | | | | |
| trig (mmol/ l) | | | | | | | |
| cre(umol/ l) | | | | | | | |
| alb (g/ l) | | | | | | | |
| fibr (g/ l) | | | | | | | |
| log alb/ cre1 | | | | | | | |
| log alb/ cre2 | | | | | | | |
| Prot (En%) | | | | | | | |
| Fat (En %) | | | | | | | |
| CHO (En %) | | | | | | | |
| Alc (En %) | | | | | | | |
| PUFA(En%) | | | | | | | |
| MUFA(En%) | | | | | | | |
| SAFA(En%) | | | | | | | |
| P/ S | | | | | | | |
| M/ S | | | | | | | |
| En (kJ) | | | | | | | |
| Prot (g) | | | | | | | |
| Prot/ bwt | | | | | | | |
| Fat(g) | | | | | | | |
| CHO (g) | | | | | | | |
| Alc (g) | | | | | | | |
| Fibre(g) | | | | | | | |
| Sugar (g) | | | | | | | |
| Starch (g) | 0.2226 | | | | | | |
| chol (mg) | 0.2771 | 0.1275 | | | | | |
| Na (mg) | -0.0563 | 0.6276 | 0.2835 | | | | |
| phys func | 0.2342 | 0.2603 | 0.2370 | 0.0107 | | | |
| rol-phys | 0.2185 | 0.2837 | 0.1432 | 0.3180 | 0.4299 | | |
| body pain | 0.3411 | 0.0576 | 0.2773 | 0.0504 | 0.0559 | 0.3705 | |
| gen hlth | 0.2558 | -0.0183 | -0.0324 | -0.0105 | 0.4805 | 0.4368 | 0.0308 |
| vitality | 0.0171 | 0.0870 | 0.2047 | 0.1627 | 0.1755 | 0.3891 | 0.4845 |
| soc funct | 0.1719 | 0.1454 | 0.2113 | 0.1691 | 0.6118 | 0.4932 | 0.3872 |
| rol-emot | 0.0928 | 0.0508 | 0.2676 | 0.2746 | 0.2215 | -0.0110 | 0.1350 |
| ment hlth | 0.0493 | -0.0442 | 0.1042 | 0.1393 | 0.0343 | 0.2417 | 0.4188 |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by sugar (g), starch (g), cholesterol (mg), sodium (mg), physical functioning, role-physical, bodily pain respectively.

## Correlation coefficient of different variables in control subjects (continued)*

| Variable | gen hlth | vitality | soc func | role-emo | ment hlth |
|---|---|---|---|---|---|
| Age | | | | | |
| BMI | | | | | |
| B F% | | | | | |
| WHR | | | | | |
| sBP | | | | | |
| dBP | | | | | |
| HbA1c% | | | | | |
| C (mmol/l) | | | | | |
| HDL C (mmol/l) | | | | | |
| LDL C (mmol/l) | | | | | |
| trig (mmol/l) | | | | | |
| cre(umol/l) | | | | | |
| alb (g/l) | | | | | |
| fibr (g/l) | | | | | |
| log alb/cre1 | | | | | |
| log alb/cre2 | | | | | |
| Prot (En%) | | | | | |
| Fat (En %) | | | | | |
| CHO (En %) | | | | | |
| Alc (En %) | | | | | |
| PUFA(En%) | | | | | |
| MUFA(En%) | | | | | |
| SAFA(En%) | | | | | |
| P/S | | | | | |
| M/S | | | | | |
| En (kJ) | | | | | |
| Prot (g) | | | | | |
| Prot/bwt | | | | | |
| Fat(g) | | | | | |
| CHO (g) | | | | | |
| Alc (g) | | | | | |
| Fibre(g) | | | | | |
| Sugar (g) | | | | | |
| Starch (g) | | | | | |
| chol (mg) | | | | | |
| Na (mg) | | | | | |
| phys func | | | | | |
| rol-phys | | | | | |
| body pain | | | | | |
| gen hlth | | | | | |
| vitality | 0.1928 | | | | |
| soc funct | 0.5194 | 0.4603 | | | |
| rol-emot | 0.2272 | 0.4237 | 0.5927 | | |
| ment hlth | 0.1112 | 0.3276 | 0.6174 | 0.5582 | |

* Values which are underlined are statistically significant. A negative sign indicates an inverse relation. The coefficients are shown for each of variables by general health vitality, social functioning, role-emotional and mental health respectively.