# **COPING WITH HIV-SEROPOSITIVE STATUS:**

### A PSYCHONEUROIMMUNOLOGICAL

### **PERSPECTIVE**

### **NEIL M. ORR**

UNIVERSITY OF CAPE TOWN
1994

IN PARTIAL FULFILLMENT OF THE DEGREE M.A. (RESEARCH PSYCHOLOGY)

SUPERVISOR: DR DANIEL LE GRANGE

The University of Cape Town has been given the right to reproduce this thesis in whole or in part. Copyright is held by the author. The copyright of this thesis vests in the author. No quotation from it or information derived from it is to be published without full acknowledgement of the source. The thesis is to be used for private study or non-commercial research purposes only.

Published by the University of Cape Town (UCT) in terms of the non-exclusive license granted to UCT by the author.

### **ABSTRACT**

Twenty-seven HIV-infected homosexual men participating in an experimental drug trial were included in a psychoneuroimmunological investigation of the association between levels of short-term emotional distress, methods of coping, hopelessness, loneliness, joy, and CD8-cell counts, CD4-cell counts, and the cumulative rate of CD4-cell decline since infection. A survey of needs was included.

The sample (n = 27) was significantly more depressed (p < 0.01), angry (p < 0.001), confused (p < 0.05), and lonelier (p < 0.01) than normative groups. There were no significant differences in emotional distress between asymptomatic subjects (n = 16; CDC II and III) and symptomatic subjects (n = 11; CDC IVa, IVc1 and IVc2). Secondary analyses indicate that the asymptomatic subjects were significantly more distressed than comparable American asymptomatic HIV-infected men, while the symptomatic subjects were not more distressed than comparable American subjects. A survey of needs revealed that financial concerns related to HIV-infection were found to be a greater source of distress than symptom status.

The sample scored significantly lower than college student norms for coping by means of seeking social support for emotional reasons (p < 0.05), and significantly higher on all scales pertaining to avoidance coping (0.02 > p < 0.001), as well as acceptance coping (p < 0.001). No significant differences (p > 0.05) were found for all problem-focused coping and emotion-focusing coping scales, nor for coping by means of focusing upon and venting of emotions.

Five subjects who had been infected for less than two years were excluded from analyses regarding immune functioning. For the remaining 22 subjects, no significant associations between psychosocial factors and CD8-cell counts were found (p > 0.05), nor were there any significant associations between measures of short-term POMS scales of emotional distress and CD4-cell counts and rates of decline over time.

A regression model containing the coping scales of suppression of competing activities and mental disengagement predicted 33.3 percent of cross-sectional CD4-cell counts (F = 4.737, df = 2,19, p < 0.05). Both factors were negatively associated with CD4-cell counts.

A regression model containing the coping methods of focusing upon and venting of emotions and mental disengagement predicted 29 percent of CD4-rates of decline over time (F = 3.874, df = 2,19, p < 0.05). The venting of emotions scale was associated with slower rates of CD4-cell decline over time (r = -0.433, df = 21, p < 0.05), while mental disengagement coping was associated with faster rates of CD4-cell decline (r = +0.314, df = 21, p = 0.16).

A median-split of scores on the focusing upon and vonting of emotions coping scale and CD4-rates of decline reveals that high venting scores are found in 77 percent of subjects with slow rates of decline, while low scores are evident for 78 percent of those with fast rates of cumulative CD4-cell decline since infection.

It was concluded that these results are consistent with previous research concerning with the immunosuppressive effects of habitual repression of emotions and the long-term maladaptive effects of avoidance coping.

# **ACKNOWLEDGEMENTS**

This study would not have been possible without the trust and assistance of John Pegge, a man of rare courage and compassion, whom I consider an honour to know.

My deepest gratitude and respect is also due to the staff of ASET (Cape Town), and the men who were willing to participate in this study. I hope this study will be of benefit to these courageous people.

I am also indebted to the invaluable assistance and advice regarding the immunological aspects of this study imparted by Dr Pat Bouic.

I also want to acknowledge the patience and interest shown by the supervisor of this project, Dr Daniel le Grange, who was willing to undertake the supervision of this project despite the logistical difficulties involved. Thank you.

The financial assistance of the Centre for Science Development (HSRC, South Africa) towards this research is hereby acknowledged. Opinions expressed and conclusions arrived at, are those of the author and are not necessarily to be attributed to the Centre for Science Developent.

Finally, I want to acknowledge the patience, support, and mental contributions made by David Patient, whose commitment to survival has served as an inspiration and focal point for the understanding of the material data gathered during this study. I hope that studies of this nature will eventually lead to an understanding of the mechanisms of survival, and an awareness than HIV does not have to mean that death is inevitable.

# CONTENTS

ABSTRACT		PAGE 1
ACK	(NOWLEDGEMENTS	3
COI	NTENTS	4
CHA	APTER 1: INTRODUCTION & OUTLINE OF THESIS	13
CHA	APTER 2: CLINICAL AND IMMUNOLOGICAL ASPECTS OF HIV AND OTHER VIRAL INFECTIONS	20
2.1	INTRODUCTION	20
2.2	THE CLINICAL NATURE OF HIV INFECTION	21
2.3	NEUROPSYCHIATRIC CONSEQUENCES OF HIV INFECTION	23
2.4	IMMUNOLOGICAL ASPECTS OF HIV INFECTION	24
2.5	MEASUREMENT OF CELL-MEDIATED IMMUNITY	29
2.6	POSSIBLE CAUSAL CO-FACTORS IN AIDS	30
2.7	HERPES VIRUSES	31
2.8	CONCLUSIONS	32

CHA	PTER 3: PSYCHONEUROIMMUNOLOGY AND HIV INFECTION: A REVIEW	33
3.1	INTRODUCTION	34
3.2	GENERAL OVERVIEW	34
3.2.1	LIFE EVENTS AND IMMUNE RESPONSE	34
3.2.2	INDIVIDUAL COPING	36
3.2.3	SOCIAL SUPPORT	39
3.2.4	PSYCHOSOCIAL INTERVENTIONS	40
3.3	PSYCHOSOCIAL FACTORS AND VIRAL INFECTIONS	42
3.3.1	CROSS-SECTIONAL STUDIES	42
3.3.2	PROSPECTIVE STUDIES	43
3.4	PSYCHOSOCIAL FACTORS AND HIV INFECTION	45
3.4.1	SOURCES OF DISTRESS IN HIV INFECTION	45
3.4.2	COPING WITH HIV INFECTION	46
3.5	CONCLUSIONS	51
CHAI	PTER 4: RESEARCH DESIGN	54
CIIA	TER 4. RESEARCH SEGISTI	<b>0</b> 4
4.1	INTRODUCTION	54
4.2	RATIONALE AND OBJECTIVES OF THE STUDY	55
4.3	RESEARCH DESIGN: ISSUES OF TEMPORALITY	56
4.4	SAMPLING PROCEDURES AND CONSIDERATIONS	57
4.5	ESTABLISHING CAUSAL DIRECTION	62
4.6	VALIDITY AND RELIABILITY OF INSTRUMENTS	67
4.6.1	COPING METHODS	68
4.6.2	REPRESSOR-SENSITIZER INTERPERSONAL STYLES	75
4.6.3	LONELINESS	77
4.6.4	SINGLE ITEMS	80
	(a) HOPELESSNESS	81
	(b) JOY	81
4.6.5	PROFILE OF RECENT MOOD STATES	82

4.7	DEMOGRAPHIC DESCRIPTION OF RESPONDENTS	84
4.8	HIV-RELATED QUESTIONS	84
4.8.1	PERIOD OF INFECTION	85
4.8.2	SYMPTOM STATUS	<b>8</b> 7
4.8.3	IMMUNE MEASURES: CD4 AND CD8-CELL COUNTS	<sup>-</sup> 89
4.9	SURVEY OF NEEDS	91
4.10	SAMPLE SIZE & STATISTICAL POWER	93
4. 10. 1	COPING ORIENTATIONS TO PROBLEMS EXPERIENCED (COPE)	93
4.10.2	PROFILE OF MOODS STATES (POMS)	96
4.10.3	CONCLUSIONS REGARDING SAMPLE SIZE	103
4.11	CONCLUSIONS	104
CHAI	PTER 5: STATISTICAL ANALYSIS AND RESULTS	105
5.1	INTRODUCTION	105
5.2	HYPOTHESES & RESEARCH OBJECTIVES	106
5.3	DESCRIPTION OF THE SAMPLE	108
5.3.1	DEMOGRAPHIC DESCRIPTION OF THE SAMPLE	109
5.3.2	CLINICAL DESCRIPTION OF THE SAMPLE	110
5.3.3	IMMUNOLOGICAL PROFILE OF THE SAMPLE	111
5.3.4	ANTIRETROVIRAL & EXPERIMENTAL DRUG EFFECTS	114
5.3.5	TIME SINCE INFECTION	118
5.3.6	RATE OF CD4-CELL DECLINE OVER TIME	121
5.3.7	PSYCHOSOCIAL COMPARISONS WITH NORMS	125
5.4	STABILITY OF COPING MEASURES	131
5.5	SHORT-TERM EMOTIONAL CHANGES	133
5.6	ASSOCIATIONS WITH IMMUNE MEASURES	135
5.6.1	CD8-CELL COUNTS: LINEAR ASSOCIATIONS	136
5.6.2	CD4-CELL COUNTS & RATE OF CD4-CELL DECLINE:	
	LINEAR ASSOCIATIONS	138
5.6.3	FACTORIAL ANALYSIS OF CD4-CELL COUNTS	141
5.6.4	FACTORIAL ANALYSIS OF CD4-RATE OF DECLINE	144
5.7	SURVEY OF NEEDS	152

CHA	PTER 6: DISCUSSION OF RESULTS	155
	INTRODUCTION	155
6.1	INTRODUCTION	155
6.2	METHODOLOGICAL CONSIDERATIONS	156
6.2.1	REPRESENTATIVENESS OF THE SAMPLE	156
6.2.2	VALIDITY OF IMMUNE MEASURES	160
	(a) ADJUSTMENT FOR MEDICAL DRUG USAGE	160
	(b) TIME BETWEEN IMMUNE AND PSYCHOSOCIAL	
	MEASUREMENT	161
	(c) MORTALITY	163
6.2.3	RELIABILITY OF SELF-REPORTED DATA	164
	(a) RELIABILITY OF REPORTED PERIOD OF INFECTION	164
	(b) RELIABILITY OF REPORTED SYMPTOM-STATUS	164
6.2.4	STABILITY OF COPING CONSTRUCTS	165 ~
6.2.5	GENERAL METHODOLOGICAL CONCLUSIONS	169
6.3	PSYCHOSOCIAL DESCRIPTIVE ANALYSES	170
6.3.1	SHORT-TERM MOOD STATES	170
6.3.2	LONELINESS	171
6.3.3	COPING METHODS	172
6.3.4	OVERVIEW OF THE SAMPLE'S PSYCHOSOCIAL PROFILE	173
6.4	ASSOCIATIONS WITH IMMUNE MEASURES	175
6.4.1	ASSOCIATIONS WITH CD8-CELL COUNTS	175
6.4.2	CD4-CELL COUNTS & RATES OF DECLINE: ASSOCIATIONS	
	WITH CLINICAL AND DEMOGRAPHIC VARIABLES	176
6.4.3	CD4-CELL COUNTS AND RATES OF DECLINE: ASSOCIATIONS	
	WITH PSYCHOSOCIAL FACTORS	179
	(a) ASSOCIATIONS WITH MOOD STATES	179
	(b) LONELINESS, JOY, AND HOPELESSNESS	180
	(c) PROBLEM-FOCUSED COPING	181
	(d) EMOTION-FOCUSED COPING	183
	(e) EMOTIONAL REPRESSION AND AVOIDANCE COPING	184
6.4.4	GENERAL CONCLUSIONS REGARDING CAUSALITY	188
6.4.5	SUGGESTIONS FOR FUTURE RESEARCH	190

REFERENCE	S	` 192
APPENDICE	SS .	
APPENDIX 1:	RESEARCH INSTRUMENT	212
APPENDIX 2:	NORMATIVE DATA PERTAINING TO:	
	(1) COPING OREINTATIONS TO PROBLEMS	
	EXPERIENCED SCALE (CARVER ET AL., 1989)	
	(2) REVISED UCLA LONELINESS SCALE	
	(RUSSELL <i>ET AL.</i> , 1980)	
	(3) HOPELESSNESS AND JOY	
	(LEVENSTEIN ET AL., 1993)	221
APPENDIX 3:	NORMATIVE DATA PERTAINING TO MCNAIR,	
	LORR & DROPPLEMAN'S (1971)	
	PROFILE OF MOOD STATES	240
APPENDIX 4:	CENTERS FOR DISEASE CONTROL (1987 REVISED)	
	HIV INFECTION SYMPTOM CLASSIFICATION	247
APPENDIX 5:	RAW DATA	253

TABLES		
TABLE 1:	EFFECT SIZES FOR THE COPE: COLLEGE STUDENTS VERSUS SERONEGATIVE HOMOSEXUAL MALES	94
TABLE 2:	EFFECT SIZES FOR THE COPE: SERONEGATIVE VERSUS ASYMPTOMATIC HIV-SEROPOSITIVE MALES	95
TABLE 3:	POMS: COLLEGE MALES VERSUS HOMOSEXUAL MALES	97
TABLE 4:	POMS: EFFECT SIZES FOR SERONEGATIVE VERSUS SEROPOSITIVE (ASYMPTOMATIC) HOMOSEXUAL MALES	99
TABLE 5:	POMS: EFFECT SIZES FOR SERONEGATIVE VERSUS SYMPTOMATIC HOMOSEXUAL MALES	100
TABLE 6:	POMS: EFFECT SIZES FOR ASYMPTOMATIC VERSUS SYMPTOMATIC (ARC) HOMOSEXUAL MALES	102
TABLE 7:	DEMOGRAPHIC DESCRIPTIVE DATA (N = 2/)	109
TABLE 8:	CLINICAL PROFILE OF THE SAMPLE	110
TABLE 9:	CD4-CELL, CD8-CELL & MEAN LENGTH OF INFECTION	113
TABLE 10:	ADJUSTED VS UNADJUSTED CD4-CELL COUNTS FOR THREE GROUPS: 0-200, 201-475, AND 475+ CD4 CELLS/MICROLITRE	117
TABLE 11:	CD4-RATES OF DECLINE FOR SUBJECTS INFECTED LONGER THAN 18 MONTHS (N = $22$ )	123
TABLE 12:	PSYCHOSOCIAL MEASUREMENTS: COMPARISON OF SAMPLE (N = 27) WITH NORMS (PROFILE OF MOOD STATES)	125
TABLE 13:	PSYCHOSOCIAL MEASUREMENTS: COMPARISON OF SAMPLE (N = 27) WITH NORMS (UCLA LONELINESS SURVEY)	126
TABLE 14:	PSYCHOSOCIAL MEASUREMENTS: COMPARISON OF SAMPLE (N = 27) WITH NORMS (COPING ORIENTATIONS TO PROBLEMS EXPERIENCED)	127
TABLE 15:	POMS: COMPARISON OF SYMPTOMATIC SUBJECTS (N = 16) WITH ASYMPTOMATIC NORMS (N = 62)	129
TABLE 16:	POMS: COMPARISON OF SYMPTOMATIC SUBJECTS (N = 11) WITH SYMPTOMATIC NORMS (N = 13)	130
TABLE 17:	CORRELATIONS BETWEEN CLINICAL, DEMOGRAPHIC, COPING, & ATTITUDINAL MEASURES (N = 27)	132

TABLE 18:	CORRELATIONS BETWEEN CLINICAL, DEMOGRAPHIC, & MOOD STATES (N = 27)	133		
TABLE 19:	LEVELS OF PROFILE OF MOOD STATES FOR SYMPTOM-CATEGORIES: 1-WAY ANOVAS	134		
TABLE 20:	LINEAR ASSOCIATIONS BETWEEN CD8-CELL COUNTS, DEMOGRAPHIC, AND PSYCHOSOCIAL FACTORS	137		
TABLE 21:	LINEAR ASSOCIATIONS BETWEEN ADJUSTED CD4-CELL COUNTS, DEMOGRAPHIC, AND PSYCHOSOCIAL FACTORS	139		
TABLE 22:	LINEAR ASSOCIATIONS BETWEEN CD4-RATE OF DECLINE, DEMOGRAPHIC, AND PSYCHOSOCIAL FACTORS	140		
TABLE 23:	CORRELATION MATRIX OF ASSOCIATES OF CD4-CELL COUNTS (N = 22)	141		
TABLE 24:	MULTIFACTORIAL MODEL FOR PREDICTING CD4-CELL COUNTS: THREE-PREDICTOR MODEL	142		
TABLE 25:	MULTIFACTORIAL MODEL FOR PREDICTING CD4-CELL COUNTS: TWO-PREDICTOR MODEL	143		
TABLE 26:	CORRELATION MATRIX OF ASSOCIATES OF CD4-RATE OF DECLINE (N = $22$ )	145		
TABLE 27:	MULTIFACTORIAL MODEL FOR PREDICTING CD4-RATES OF DECLINE (TWO PREDICTORS)	148		
TABLE 28:	PSYCHOSOCIAL ASSOCIATES WITH PREDICTORS OF CD4-CELL COUNT AND CD4-RATE (N = 22)	149		
TABLES IN	TABLES IN APPENDICES			
TABLE 2-A:	COPE SUBSCALES: ITEMS LISTED BY <i>A PRIORI</i> SCALE ASSIGNMENT: WITH LOADINGS ON THE FACTOR TO WHICH EACH ITEM PERTAINS	222		
TABLE 2-B:	CORRELATIONS AMONG DISPOSITIONAL COPE SCALE, COMPUTED AS UNWEIGHTED SUMS OF THE ITEMS COMPOSING EACH SCALE (N = 978)	225		
TABLE 2-C:	CONVERGENT AND DISCRIMINANT VALIDITY OF THE COPE	226		
TABLE 2-D:	INTERNAL CONSISTENCY, TEST-RETEST RELIABILITY, AND NORMS OF COPE	228		
TABLE 2-E:	ORIGINAL INSTRUCTIONS AND RATING SCALES FOR COPE	229		

TABLE 2-F:	MODIFIED INSTRUCTIONS AND RATING SCALES FOR COPE	230
TABLE 2-G:	TEST SEQUENCE OF COPE ITEMS	231
TABLE 2-H:	SCORING PROCEDURE FOR COPE SCALES	233
TABLE 2-I:	THE SURVEY FORM OF THE REVISED UCLA LONELINESS SCALE	234
TABLE 2-J:	MODIFIED INSTRUCTIONS AND RATING SCALES FOR UCLA LONELINESS SCALE (SURVEY FORM)	235
TABLE 2-K:	UCLA LONELINESS SURVEY FORM NORMATIVE MEANS AND STANDARD DEVIATIONS FOR DIFFERENT AGE GROUPS	236
TABLE 2-L:	SINGLE ITEMS FROM LEVENSTEIN <i>ET AL'S</i> (1993) PERCEIVED STRESS QUESTIONNAIRE	237
TABLE 2-M:	FACTOR LOADING AND RELATIONS OR 25Q ITEMS TO OTHER CONSTRUCTS	238
TABLE 2-N:	MODIFIED PSQ SINGLE ITEMS	239
TABLE 3-A:	PROFILE OF MOOD STATES: INTER-SCALE CORRELATIONS	241
TABLE 3-B:	PROFILE OF MOOD STATES: INTERNAL CONSISTENCY (K-R 20) OF SCALES	242
TABLE 3-C:	PROFILE OF MOOD STATES: TEST-RETEST RELIABILITY ( $r_{tt}$ )	243
TABLE 3-D:	PROFILE OF MOOD STATES: ASSOCIATIONS WITH SOCIAL DESIRABILITY	244
TABLE 3-E:	PROFILE OF MOOD STATES: SCORING PROCEDURE	245
TABLE 3-F:	PROFILE OF MOOD STATES: COLLEGE MALE NORMS (N=340)	246
TABLE 5-A:	DEMOGRAPHIC DESCRIPTION OF SAMPLE	254
TABLE 5-B:	SYMPTOMATOLOGY & (UNADJUSTED) IMMUNE MEASURES	255
TABLE 5-C:	REPORTED TIME OF INFECTION	256
TABLE 5-D:	PERIODS OF MEDICALLY-PRESCRIBED ANTIRETROVIRALS & EXPERIMENTAL DRUGS	258
TABLE 5-E:	ADJUSTMENTS TO CD4-CELL COUNTS FOR ANTIRETROVIRAL & EXPERIMENTAL DRUG USAGE	259
TABLE 5-F:	CALCULATING THE RATE OF CD4-CELL DECLINE FROM INFECTION UNTIL IMMUNE MEASUREMENT	260

TABLE 5-G:	PROFILE OF MOOD STATES (POMS) SCORES (N = 27)	261
TABLE 5-H:	COPING ORIENTATIONS TO PROBLEMS EXPERIENCED (COPE): PROBLEM-FOCUSED SCALES (N = $27$ )	262
TABLE 5-I:	COPING ORIENTATIONS TO PROBLEMS EXPERIENCED (COPE): OTHER SCALES (N = $27$ )	263
TABLE 5-J:	UCLA LONELINESS SCALE, HOPELESSNESS, & JOY (N = 27)	264
	, , , , , , , , , , , , , , , , , , ,	
FIGURES		
FIGURE 1a:	CD4 COUNTS IN HIV INFECTION: STATISTICAL MEANS: INFECTION TILL AIDS	25
FIGURE 1b:	CUMUL TTL CD4-CELL LOSS/TIME: CUMULATIVE FROM THE TIME OF INFECTION	25
FIGURE 2:	LEVELS OF CD8-CELLS FROM INFECTION UNTIL DIAGNOSIS OF AIDS	26
FIGURE 3:	SAMPLE CD4-CELL COUNT X TIME INFECTED N = 27 (CD4-UNADJUSTED)	112
FIGURE 4:	SAMPLE CD8-CELL COUNTS X TIME INFECTED N = 26	112
FIGURE 5:	ADJUSTED VS UNADJUSTED CD4-CELL COUNTS PER SUBJECT	116
FIGURE 6:	RANGE OF REPORTED PERIOD OF TIME INFECTED IN MONTHS	118
FIGURE 7:	CERTAINTY OF REPORTED INFECTION DATES  1 = VERY UNCERTAIN 7 = VERY CERTAIN	120
FIGURE 8:	CD4-RATE OF DECLINE (N = 22)	123
FIGURE 9:	CD4-CELL RATE X VENTING OF EMOTIONS	147

# CHAPTER 1

# INTRODUCTION & OUTLINE OF THESIS

AIDS may truly be considered the most serious epidemic of its kind in modern times, and behavioural methods may be the only method for controlling this infectious disease (Kelly and Murphy, 1992), largely due to the predominant mode of transmission, namely intimate sexual contact. The absence of enduring biomedical controls and treatments, which is regarded as being in it's infancy (King, 1993), has added a sense of urgency to efforts towards the prevention of the spread of HIV through education of those at risk, as well as efforts to manage the distressing consequences experienced by those who have already been infected.

The first official report of AIDS was recorded in June 1981 (Boo, 1991), which occurred approximately within a year of the discovery of the first retrovirus (of which HIV is an example) by Robert Gallo. In the early 1980's, prior to the identification of the Human Immunodeficiency Virus (HIV) by Robert Gallo and Luc Montagnier in 1984, AIDS was found almost exclusively among homosexual men of the San Francisco and other metropoles of the USA and Europe, and was initially termed Gay Related Immunodeficiency Disease (GRID). Due to the initial epidemiological occurrence of the disease, the disease was largely dismissed as a 'gay disease'. This erroneous belief has been exposed as the myth it is, largely due to the virus's rapid spread to all groups, with evident indifference to lifestyle, gender, socioeconomic status, or sexual preference (Jennison, 1993; McKenzie, 1991, p.1; Shilts, 1987).

Despite the biological definition of the disease, and the profound psychological and social consequences for those infected with the virus, the response to the epidemic has been characterised by political cynicism, social stigmatisation and fear, as well as the determination of others to counter such intransigence, as evidenced by the history of the epidemic in the USA during the early and mid-1980's (e.g., Shilts, 1987).

In 1993, AIDS became the top killer of men between the ages of 25 to 44 years in the USA, and the fourth largest killer of women in the same age category ("Aids top killer," 1993). In South Africa, it appears that the epidemic is in it's early stages, but that it is now entering a stage of rapid acceleration (Steinberg, 1992).

Recent epidemiological studies indicate an overall HIV incidence of 3% (approx. 1.2 million) in the general South African population, with higher levels reported for specific groups, such as patients reporting at STD (sexually-transmitted disease) clinics, women utilising antenatal clinics, and tuberculosis patients (Spencer, 1993). According to Wilson (1993), HIV in Africa is spreading at one hundred times the rate found in Europe, largely due to specific sexual patterns and the medical neglect of sexually transmitted diseases. In Zimbabwe, for example, up to 20 percent of mothers reporting to antenatal clinics, and 33 percent of those with STD's, are infected with HIV (Wilson, 1993).

Medical control of HIV infection with antiretroviral agents such as AZT (azidothymidine), dall (dideoxyinosine), or ddC (dideoxycytidine), has proved to be limited, with the virus developing resistance to certain drugs, and with limited efficacy in those who are already immune-compromised. Recent results from the Concorde trial have further indicated the limited benefits of AZT (National Institute of Allergy and Infectious Diseases, 1993; Medical Research Council, UK, 1993), as well as convergent therapies of AZT, dall and Nevirapine or Pyridinone, ("Report slams", 1994). Furthermore, the high costs of such drugs places such treatment beyond the economic means of most people in Africa. It is therefore not surprising that AIDS has become a metaphor for fear and death in these last two decades of the twentieth century, in much the same way that tuberculosis and cancer instilled fear in previous decades (Sontag, 1991).

Although the initial reaction to AIDS was slow, rapid developments in organised programmes for dealing with the epidemic have occurred in the last few years. These HIV-related psychological interventions and programmes appear to focus predominantly upon caring for people with AIDS (e.g., Sims & Moss, 1991; Huber & Schneider, 1992), modification of behaviour for populations at risk (e.g. Aspinwall, Kemeny, Taylor, Schneider, & Dudley, 1991; Stall, Coates & Hoff, 1988; Davidson et al., 1992; McKirnan & Peterson, 1989; Levy, 1988), limiting and understanding the distress of HIV antibody testing (e.g. Antoni, August et al., 1990; Ironson et al., 1990; Perry, Fishman, Jacobsberg, Young & Frances, 1991), adaptation to positive test results (e.g. LaPerriere et al., 1990; Antoni et al., 1991) and behavioural programmes aimed at maintaining health in HIV-positive persons (e.g. Coates, McKusick, Kuno, & Stites, 1989; Elliot, Goldberg & Coodley, 1992; Rigsby, Dishman, Jackson, MacLean & Raven, 1992a, 1992b).

The latter aspect, namely the maintenance of health in those infected, has been receiving increasing attention in the last few years, due to the realization that there is a small percentage of HIV-infected individuals who have remained well despite prolonged periods of infection, while others have deteriorated rapidly and died. The individual diversity of the progression of HIV-infection has resulted in a re-examination of the factors associated with HIV-illness progression, as well as recent shifts in treatments away from anti-retroviral medication towards immune-based approaches, with intense research currently being conducted in the areas of interleukin-12, cytokine therapy, T-cell transfusions, and other treatments directed at immuno-enhancement (Cohan, 1994).

In South Africa, as elsewhere, urbanised HIV-infected individuals have access to programmes and support groups with the express purpose of sharing and learning methods of remaining well, such as Body Positive. However, such programmes are mainly utilised by homosexual males (at least in South Africa), because of the history of the spread of the epidemic. Access to such programmes is further restricted by fears of disclosing seropositive status to others, regardless of the strict confidentiality and anonymity of such programmes.

The homosexual male population has historically been exposed to the social stigma and personal consequences associated with HIV-infection for a much longer period of time than the heterosexual population, both in South Africa and in the USA and Europe, and it is therefore not surprising that the genesis of many programmes concerning coping with infection is to be found in organisations servicing homosexuals.

lt is interestina to reflect upon the parallel development Psychoneuroimmunology (hereafter PNI), the specialised field which seeks to explicate issues related to associations between psychological, social and immunological factors. Although the notion that emotional states, personality variables and social or environmental stressors may affect physical health is by no means new, it was not until the late 197ú's that the body of evidence supporting such a notion began to become formally organised, and culminated in the seminal work *Psychoneuroimmunology* (1981) edited by Robert Ader.

Thus, from a PNI perspective, in order to construct a programme for maintaining health in HIV-infected people, it is essential to first identify those psychosocial factors which are most significantly associated with the immunological aspects of HIV illness progression. Although these psychosocial factors may not necessarily be causally involved in HIV-illness progression, there is a growing body of evidence suggesting that certain psycho-immunological associations may indeed be causal, while others may be more appropriately explained in terms of factors concerning illness progression itself, such as increased distress and neuropsychiatric complications associated with the development of symptoms (Ballieux, 1992; Camara & Danao, 1989; Hassan & Douglas, 1990; Solomon, Kemeny & Temoshok, 1991; Temoshok, 1988).

The purpose of the present study is to provide an initial basis for subsequent investigations concerning associations between various psychosocial factors, particularly coping methods, and the immunological progression of HIV-infection. Due to restrictions pertaining specifically to the lengthy average period of infection, a retrospective methodology is employed.

A secondary adjunct to this PNI investigation is a brief survey of needs of HIV-infected individuals concerning needs for assistance and receptivity to programmes related to coping with HIV-infection, within the context of the specific service organisation to which the person is affiliated, namely ASET (Aids Support and Education Trust, Cape Town).

From a virological perspective, the HIV appears to be a highly-specific virus, and thus the literature reviewed is largely restricted to those aspects of the immune system which feature prominently in HIV-infection, namely cell-mediated immunity (CMI), and CD4 cells in particular. Future prospective studies would of necessity need to further investigate and confirm any emergent associations. The psychosocial factors which are focused upon are selected from a review of previous studies investigating links between various psychosocial factors and immune functioning. If such associations are indeed present, and subsequent investigations demonstrate that some of these psychosocial factors are causally involved in HIV illness progression, there are several important implications, such as the possible benefits of focused intervention programmes for maintaining health in HIV-infected people.

It is, however, important to point out that these same psychological constructs may be viewed from a second (equally important) perspective, namely their relevance for the emotional and cognitive well-being of those who are HIV-infected. It is clearly necessary to establish patterns of characteristic emotional states at different stages of infection, as these have direct relevance for therapeutic interventions and the provision of services for those people living with HIV. The fear of impending symptoms may, for example, be more acute in those who are asymptomatic, while coming to terms with the possibility of dying may be more relevant for those who have already developed symptoms.

Related to these differences in emotional distress are possible differences in needs regarding assistance, as well as feelings towards organised programmes for either emotional or problem-directed coping. This aspect is of particular relevance to agencies who provide services for HIV-infected people.

Although it has been stated that the present study is primarily motivated to investigate the so-called stress-illness hypothesis, this all-encompassing label is perhaps a misnomer, as there is currently little agreement regarding the conceptual definition or measurement of stress (Marmot & Madge, 1987; Leventhal & Tomarken, 1987), and the term is more accurately descriptive of a broad collection of specific psychosocial factors with potentially detrimental biophysical consequences. Even the latter description is perhaps too specific, as stress does not necessarily result in negative consequences. Therefore, the concept of stress is avoided unless consulted sources refer specifically to this concept, usually in the context of life events. Instead, specific psychosocial factors which are amenable to clearer conceptual definition and standardised measurement, such as coping strategies, depression, anxiety and loneliness, are focused upon.

This thesis consists of three parts, commencing with part 1 (chapters 2 and 3), which consists of a review of immunological and PNI literature. In order to delimit the extent of the PNI review in chapter 3, chapter 2 focuses solely upon describing the most important clinical and immunological aspects of HIV infection and general viral infection immunology. Chapter 3 reviews PNI studies investigating psychosocial associations within the immunological structural and functional parameters established in chapter 2, commencing from a general overview of PNI associations, which is then focused more narrowly upon viral infections such as herpes infection, and finally, HIV-infection. Given the aims of the study, a fairly broad overview of the relevant psychoneuroimmunological literature was conducted, and this is used as the theoretical basis of the study.

An important component of this review concerns factors such as coping styles, social support, interpersonal relationships, loneliness, and depression. The reviewed literature was obtained from various sources, including MEDLINE, ERIC, Psychological Abstracts, BORIS, chapter reference lists contained in the seminal work, *Psychoneuroimmunology* (2nd Edition, 1991, edited by Ader, Felten & Cohen), and references contained in various articles concerning HIV-infection and psychosocial variables.

Part 2 (chapters 4 and 5) concerns the research methodology employed (chapter 4), while chapter 5 describes the results obtained in the study. Part 3 (chapter 6) is a discussion of the findings, as well as the implications of the obtained results for psychosocial intervention programmes and strategies.

The field of psychoneuroimmunology (PNI) is a rapidly-growing field, and only a limited selection of studies are discussed in detail. Area review references are included in the different sections for a more comprehensive understanding. For extensive overviews of the entire PNI area, the reader is referred to reviews by Udelman and Udelman (1983), Jemmott (1985), Baker (1987), Borysenko (1987), Geiser (1989), Stern (1988), Kiecolt-Glaser and Glaser (1989), Korneva (1989), Ratliff-Crain, Temoshok, Kiecolt-Glaser & Tamarkin (1989), and Vollhardt (1991).

# **CHAPTER 2**

# CLINICAL AND IMMUNOLOGICAL ASPECTS OF HIV AND OTHER VIRAL INFECTIONS

### 2.1 INTRODUCTION

Before we turn our attention to reviewing the psychosocial factors associated with HIV infection, it is necessary to first identify and delimit the review to those immune components which are principally involved in HIV infection, as each disease has distinct immunological characteristics and parameters.

In HIV infection, the primary target of the virus is the helper T-cell, hereafter referred to as CD4-cells. CD4-cells operate largely as the identifiers of foreign organisms (antigens) after initial exposure, particularly viruses, and they activate other immune components to counter these antigens in subsequent exposures. The destruction of CD4-cells by HIV effectively destroys the 'memory' of the immune system, and deactivates it's ability to identify and mobilise against antigens. The principal immunological characteristic of HIV infection is thus the decline in the number of CD4-cells over time (Sheppard, Lang, Ascher, Vittinghoff & Winkelstein, 1993). The development of AIDS - the emergence of life-threatening opportunistic infections or lymphomas - is usually the result of CD4-cell counts which approach levels of 200 CD4-cells per microlitre of plasma or less, which effectively reflects an immune system which, although potentially capable of counteracting various infections or lymphomas, does not become activated due to the virtual absence of the CD4-cells (Kirkwood & Lewis, 1989).

### 2.2 THE CLINICAL NATURE OF HIV INFECTION

HIV infection has two distinct biological consequences, namely impaired immune function (particularly cell-mediated immunity) and neurologic deficits (Baum & Nesselhof, 1988; McCutchan, 1990). HIV is also the cause of other related conditions, such as intestinal conditions (Aggleton, Homans, Mojsa, Watson & Watney, 1989). In conjunction with the biological consequences, HIV infection has profound personal implications and consequences for emotional and cognitive adaptation, and is a source of great distress in many respects (Batchelor, 1988).

Although it is widely believed that AIDS inevitably leads to death, this view does not reflect the true nature of the disease, which is far inore complex, and the individual course of infection is diverse. There are those who have remained asymptomatic for more than a decade ("Long-term survivors", 1993; Haney, 1994), some have recovered from serious bouts of symptoms and have regained health, while others have rapidly deteriorated and died after initial infection.

Various reasons for this diversity have been postulated, including the effects of positive attitudes (e.g., Nielsen, 1993), individual differences in levels of cytotoxic CD8 lymphocytes (Haney, 1994), and complex interactions between host responses and the viral pathogen (e.g., Sheppard *et al.* 1993).

According to Glasner and Kaslow (1990), the progression of HIV infection is difficult to predict, as the period between infection and development of AIDS symptomatology varies between 1 to 10 years, with an average of 7 to 8 years latency. Survival after the diagnosis of AIDS likewise varies widely. Prior to the use of AZT (azidothymidine), the median survival rate after AIDS diagnosis was about 1 year, with a small percentage (less than 15 %) surviving for longer than 4 years.

However, the value of AZT in prolonging survival has been subsequently questioned, particularly in the light of the results of the Concorde Trials (Medical Research Council, UK., 1993), which suggests that AZT does not appear to make much difference in CD4-cell counts, regardless of when treatment is commenced.

Nevertheless, the increased number of prophylactic treatments and the development of more effective drugs for countering opportunistic infections, will undoubtably serve to extend the mean survival rate of symptomatic people.

Various sources (e.g., Catalan, 1988; Baum & Nesselhof, 1988; Atkinson *et al.*, 1983), state that HIV infection can be categorised into three broad categories, depending upon the nature and presence of various symptoms. The first category concerns initial acute infection and the subsequent asymptomatic stage of infection, while the second category concerns persistent generalized lymphadenopathy (PGL). PGL may occur soon after infection and persist for many years without any other AIDS-related symptoms, and many of the studies examined consider PGL as part of the asymptomatic period.

The third category is AIDS, which is subdivided into five subcategories. The first of these categories concerns constitutional disease (e.g., chronic diarrhoea, involuntary weight loss, persistent fever), which, together with PGL and other non-life-threatening infections, was previously classified as AIDS-related Complex (ARC). It should be noted that the term 'ARC' is now less frequently used than in previous years, and is now referred to as AIDS category IVa, i.e., non-life-threatening constitutional illness (excluding PGL). The second subgroup concerns neurological disease (e.g., dementia or myelopathy), while the remaining three subgroups concern specific secondary infections, secondary cancers, and concurrent HIV-unrelated conditions, respectively.

# 2.3 NEUROPSYCHIATRIC CONSEQUENCES OF HIV INFECTION

In assessing the emotional and cognitive effects of HIV infection, it is difficult to separate the direct effects of HIV upon the brain and CNS from the psychological effects of the distress caused by the severity of symptoms, fear of possible death, social stigmatization, and many other sources of anxiety.

For this reason, studies concerning organic neuropsychiatric conditions (CDC Group IV-B) have been excluded from further review, and functional cognitive conditions are investigated instead.

The distinction between organic and functional conditions does not, however, imply an absence of interaction between the direct effects of HIV and the indirect emotional response to the disease.

The reader is referred to studies by Atkinson *et al.* (1988), Catalan (1988), Ostrow (1988), Rundell, Paolucci, Beatty, and Boswell (1988), and Tross & Hirsch, (1988), for a more comprehensive treatment of the neurological impairments accompanying HIV infection.

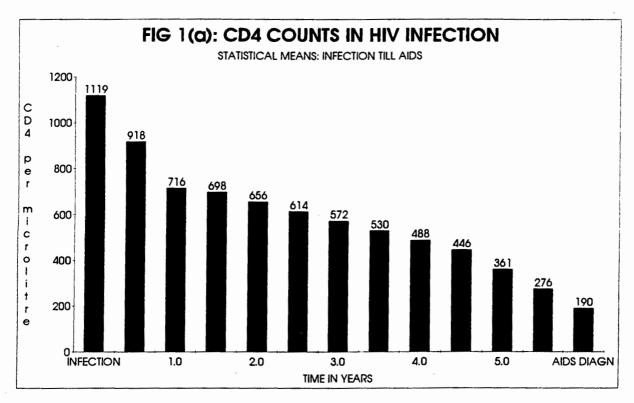
#### 2.4 IMMUNOLOGICAL ASPECTS OF HIV INFECTION

The immune components outlined in this section are themselves part of a much larger biological system involved in counteracting viruses and other antigens, such as the endocrine system, which includes the neuroendocrine system and its diverse hormonal components (Baum, Davidson, Singer & Street, 1987; Daruna & Morgan, 1990; Phillips, 1991). However, the cells and systems discussed appear to be the central areas of focus in HIV infection. More detailed overviews of the immune system can be obtained in Laudenslager and Reite (1984), Tecoma and Huey (1985), Borysenko (1987), and Ader, Felten and Cohen (1990).

It should be stated that the HIV does not 'destroy the immune system', as is so often stated. Instead, the HIV is highly selective (Batchelor, 1988). The prime target of HIV is the CD4 T-cell, and for this reason most studies investigating the role of psychosocial variables in HIV infection tend to focus upon this single aspect of immune structure, as decreases in CD4 helper T-cells and increases in CD8 suppressor T-cells appear to correlate with the course of HIV infection.

There is a general pattern of progression of the infection from seroconversion to AIDS diagnosis, which is closely correlated with the number of CD4 helper T-cells per microlitre (refer Figure 1). This progression of CD4 decline can be described as follows:

Most patients have less than 15-20% (200/mmn) of the normal number of (CD4) cells at the time AIDS is diagnosed. In addition, the remaining CD4 cells are functionally impaired. ... (In the progression of the disease from initial infection), three stages can be demonstrated (Lang et al., 1989). At the time of seroconversion, a rapid decline by about one third (1100 to 700) occurs within 12 to 18 months. ... (During the asymptomatic period which follows), a slower rate of decline of about 80 CD4 cells per year ensues. ... In the 2-year period before AIDS develops, a second accelerated period of CD4 destruction reduces levels from 400-500 to less than 200 at the time of AIDS diagnosis. (McCutchan, 1990, pp.8-9).



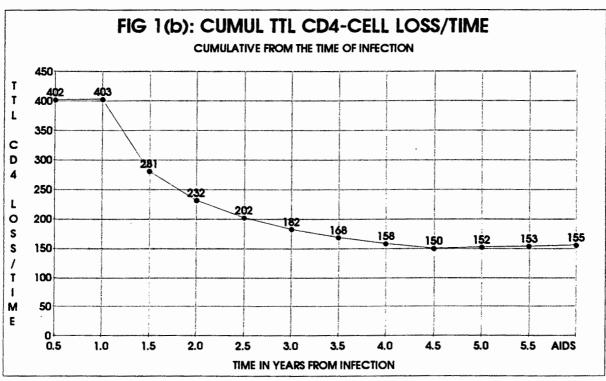


FIGURE 1
NORMATIVE MEAN CD4-CELL COUNTS & RATES OF DECLINE
FROM INITIAL HIV-INFECTION UNTIL DIAGNOSIS OF AIDS

Adapted from: "Patterns of T lymphocyte changes with Human Immunodeficiency Virus infection: From seroconversion to the development of AIDS", by Lang et al., 1989, Journal of Acquired Immune Deficiency Syndromes, 2, p.63-69. Copyright 1989 by Journal of Acquired Immunodeficiency Syndromes, 2.

Unlike the decrease in CD4-cells after infection, levels of CD8-cells rise after initial infection, and remain higher than normal until just prior to the development of AIDS (refer figure 2), at which stage CD8-cell levels plummet down to levels comparable to CD4-cell counts.

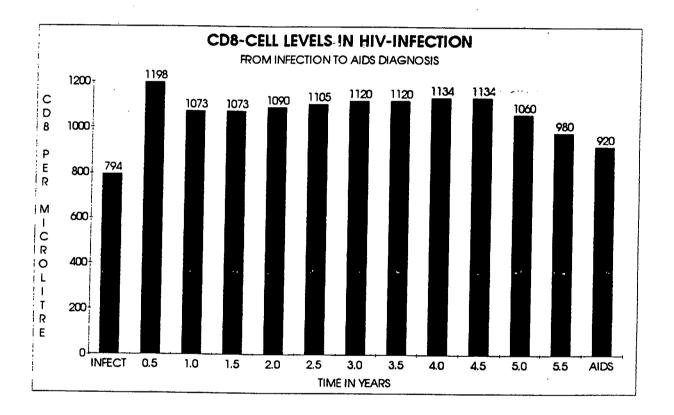


FIGURE 2
LEVELS OF CD8-CELLS FROM INFECTION UNTIL DIAGNOSIS OF AIDS

Adapted from: "Patterns of T lymphocyte changes with Human Immunodeficiency Virus infection: From seroconversion to the development of AIDS", by Lang et al., 1989, Journal of Acquired Immune Deficiency Syndromes, 2, p.63-69. Copyright 1989 by Journal of Acquired Immunodeficiency Syndromes, 2.

The immune system is a highly complex interdependent system, and CD4 and CD8-cells form only a small part of the many components of this system, albeit a crucial component (Weiner, 1991). For example, the 'front-line defence' in terms of countering infections, namely humoral immunity, is partially regulated by T-cells, by means of specific types of proteins (e.g., interleukins, gamma-globulins, and interferon) which serve as the immunological means of communication ("IL-12", 1993).

Therefore, in reviewing prior PNI research, it is necessary to include studies pertaining to other components of the immune system which affect the ability to counter viral infections, and which affect the functioning of CD4-cells. In particular, some CD8-cells act to suppress the immunological activity initiated by CD4-cells, while Natural Killer cells (large granular lymphocytes) operate relatively independently to destroy virally-infected cells. It should be noted that there are two types of CD8-cells, namely suppressor CD8-cells and cytotoxic CD8-cells. CD4 and CD8-cells form part of cell-mediated immunity, i.e., Tlymphocytes which destroy antigens within body tissue or cells (particularly in viral infections), as opposed to humoral immunity, i.e., B-lymphocytes and cells. which destroys plasma antigens through the production immunoglobulins and antibodies, particularly in bacterial infections.

Where the humoral system (B-celis) counteracts infections through antibody and immunoglobulin secretions, the cell-mediated system (T-cells) operates primarily on the basis of direct cell-antigen interaction. Natural Killer (NK) cells are nonspecific agents acting against virally-infected cells, and impairment of NK cells thus leads to an increased incidence of recurrent viral infections (Irwin, Daniels & Weiner, 1987).

In HIV infection, a period of viral latency is followed by active viral proliferation and activity, which may signal the development of symptomatology and increased destruction of CD4-cells.

Herpes infection follows a similar pattern, and is often used as a model for HIV infection. In Herpesvirus infection, for example, the virus is controlled and contained within host cells after an active infection. If this control is maintained, levels of circulating antibodies to the virus are low, but when the

virus is reactivated, i.e., when the immune system fails to repress the virus, antibodies are generated and the levels of these antibodies are thus high when measured in titration processes. When the cell-mediated immune response regains control of the virus, antibody levels drop again. In this sense, higher antibody titers are associated with less competent cell-mediated immune functioning (Glaser, Kiecolt-Glaser, Speicher, & Holliday, 1985; Kiecolt-Glaser et al., 1986; Kiecolt-Glaser & Glaser, 1991; Cohen, Tyrrell, & Smith, 1993).

The humoral and cell-mediated aspects of the greater immune system are interactive. For example, CD4 and CD8-cells may stimulate or diminish antibody production, while humoral factors, especially interferons, macrophages, monocytes, and immunoglobulins, are also important non-specific agents against viruses, particularly during initial exposure (Kirkwood & Lewis, 1989).

A recent theory postulated by Clerici and Shearer ("IL-12", 1993; Clerici & Shearer, 1993) proposes that, after initial exposure to HIV, the body counters the virus primarily by means of cell-mediated immunity (CMI), referred to as TH1 (Thelper cell pattern 1). At some point during the asymptomatic period of infection, the body switches from a CMI-based to an antigen-based response The latter is less effective in controlling the virus, and may precipitate a more rapid decline in CD4-cell counts and the development of AIDS symptomatology, referred to as TH2 (T-helper cell pattern 2). These two patterns are mutually suppressive. It appears that a crucial factor in this switch is the protein Interleukin-12 (IL-12), which is sometimes referred to as Natural Killer-cell Stimulatory Factor (NKSF), and which is not to be confused with another important factor, namely Interleukin-2. It is therefore apparent that, whatever factors are associated with the switch from optimal cell-mediated immunological control of the HIV to the less-than-optimal antigen-based pattern, the same factor is linked to Natural Killer-cell activity levels. For a more detailed description of these immunological aspects, the reader is referred to reviews by Polk et al. (1987), Solomon, Temoshok, O'Leary, and Zich (1987), Hall (1988), Aggleton et al. (1989), Antoni, Schneiderman et al. (1990), Glasner and Kaslow (1990), and McCutchan (1990).

### 2.5 MEASUREMENT OF CELL-MEDIATED IMMUNITY

Two basic methods are employed in investigating cell-mediated immune responses: The first concerns structural cell counts (e.g. CD4, CD8, and Total T-cell lymphocyte counts) or cell ratios (e.g., CD4:CD8 cell count ratios), where lower ratios imply immunosuppression, as lower ratios are the product of higher levels of suppressor CD8 cells accompanying comparatively lower levels of the activating CD4-cells.

The second measure concerns functional reactions of cells, such as mitogenically stimulated lymphocyte proliferation rates (MSLP) and Natural Killer-cell activity (NKA), alternatively called NK-cell cytotoxicity (NKCC) levels. Functional reactivity levels refer to the ability of cells to respond, as opposed to the sheer number of such cells (O'Leary, 1990; Kirkwood & Lewis, 1989).

The value of examining both structural and functional measurements becomes apparent when it is considered that cell-counts reflect only the current structural status of the cell-mediated immune system, while the functional response to MSLP reflects the extent to which these immune markers actively respond to foreign substances. For example, it is possible that, although cells such as NK-cells may remain intact during an infection, they may fail to actively attack and destroy viral antigens. In HIV infection, this would result in greater destruction of CD4-cells, due to the inability of NK-cells to reduce the number of viruses which destroy CD4-cells.

In order to facilitate uniformity and ease of reference, the following abbreviations will be utilised throughout this review:

TLC..... Total Lymphocyte count

CD4.....Helper T-cell lymphocytes

CD8.....Suppressor T-cell lymphocytes

NK...... Natural Killer-cells

NKA/NKCC..... Natural Killer-cell activity/cytotoxicity

MSLP..... Mitogenically-stimulated lymphocyte proliferation

### 2.6 POSSIBLE CAUSAL CO-FACTORS IN AIDS

A controversial alternative view of the aetiology of AIDS is that HIV may possibly be an opportunistic infection which only occurs in those who are already immunocompromised by an unidentified agent, possibly some other virus or bacterium, or even lifestyle factors such as drug abuse. With respect to possible biological cofactors, Esterling et al. (1992) cite several studies indicating a possible link between Epstein Barr Virus-transformed B-cells and the development of AIDS. Dr Peter Duesberg is a major opponent of the theory that HIV causes AIDS, and instead proposes that the HIV is a relatively harmless virus, and that lifestyle factors, particularly the frequent use of particular recreational drugs, is the cause of AIDS (Guccione, 1994).

According to Root-Bernstein (1992), several reasons are forwarded for postulating the existence of causal co-factors in AIDS. These include clinical observations that, although people with AIDS may have 90% of CD4 cells destroyed in the course of the illness, no more than 0.2% of these destroyed cells are infected with HIV. Furthermore, there is an increasing number of AIDS cases in which the HIV can not be detected, despite sophisticated detection techniques. These observations have been noted by eminent AIDS researchers, including Luc Montagnier, the co-discoverer of the HIV. Root-Bernstein proposes similar alternative causes or co-factors for AIDS, such as immunosuppressive contact with semen, recreational drugs, antibiotics, antidepressants, anaesthetics, vitamin-related malnutrition, blood therapy, multiple infections, and mycoplasma bacteria.

Although there are indications that a growing number of scientists are considering these possibilities more seriously (Root-Bernstein, 1992), in this paper we will assume the majority position, namely that AIDS is caused by HIV. Although the alternative views of Duesberg and Root-Bernstein can not be dismissed out of hand, the weight of evidence tends to suggest that HIV is indeed intimately associated with the development of AIDS, at least for the overwhelming majority of HIV-infected individuals.

#### 2.7 HERPES VIRUSES

As stated in the introduction, herpesvirus infection is often used as a model for understanding the general immunological nature of HIV infection. Therefore, a brief immunological description for herpesvirus infection is presented in order to provide an immunological basis for PNI studies reviewed in chapter 3 pertaining to herpesvirus infection.

There are several varieties of Herpesvirus, including Epstein-Barr Virus (hereafter referred to as EBV), which is the causal agent in Infectious Mononucleosis (IM), Herpes Simplex (HSV-1), the common cold sore, Herpes Zoster (shingles), and Cytomegalovirus (CMV), a less common Herpesvirus which rarely results in symptoms except in HIV infection. Human Herpesvirus Type-6 (HHV-6) is a less well known Herpesvirus variety, and both HIV and HHV-6 target CD4-cells and share the receptor sites on CD4 lymphocyte surfaces. Co-infection by these two viruses may aggravate AIDS, and these two viruses may enhance the destructive capacities of each other. There are also links between EBV and HIV, with evidence that EBV infection precedes certain lymphomas in AIDS patients, and that chemical factors related to EBV activation lead to stimulation of other factors which promote HIV (Esterling et al., 1992).

All these Herpes varieties are infectious, and are believed to remain in the body for life. They are latent most of the time, usually residing in the nerves near the sites of infection, until activated (Kiester, 1989; Gilder & Hodgkin, 1987). The reactivation of latent viruses such as Herpesvirus thus have direct relevance to issues concerning HIV-1 infection, and may serve as a model of how psychosocial factors may precipitate activity of latent viruses.

### 2.8 CONCLUSIONS

Several central immunological components pertinent to HIV infection have been identified, including CD4 and CD8-cell counts, CD4:CD8 ratios, antibody levels, Natural Killer cell counts and activity, as well as functional immune measures such as lymphocytic response to stimulation. It is also apparent that HIV infection contains a period of viral latency followed by viral activity and the development of symptoms. Furthermore, the individual diversity in this asymptomatic period of viral latency is a major feature of HIV infection, and investigations into the reasons for this diversity is of paramount importance for the maintenance of health in HIV-infected individuals.

# **CHAPTER 3**

# PSYCHONEUROIMMUNOLOGY AND HIV INFECTION: A REVIEW

One of the more commonly held assumptions about psychological influences on physiological functioning is that stress and the immune function are linked. Research supports this hypothesis. ... The study of immunity and stress in the HIV- positive, ARC, or AIDS patient can provide important information about the nature of the stress-immunity relation as well as about the disease itself. The HIV appears to remain in a latent stage after it enters the body and may not become 'active' or induce ARC or AIDS for some time. The length of time between infection and onset of one of these two syndromes is variable and could be a function of several factors. One factor may be stress: Research has suggested that stress plays a role in the activation of latent herpes viruses, presumably by weakening immunoregulation of the virus ... (and the) same could be true of the HIV (the virus may not be 'held in check' by an immune system weakened by stress). ... (Therefore) the role of stress in the immunological consequences of HIV infection is an important topic for study. Normal variations in stress, compounded by events and upsets associated with the HIV (e.g., learning that one is HIV-positive, seeing someone else become ill and die) may interact with the physical course of HIV and in that way affect the progression of the disease. (Baum & Nesselhof, 1988, p.903).

### 3.1 INTRODUCTION

Chapter 2 has delimited the immunological parameters pertinent to HIV infection, and this chapter reviews existing empirical PNI research related to these central immunological parameters. There are relatively few PNI studies utilising HIV-infected individuals, largely due to the recency of the discovery of the virus and disease, which is further restricted by limitations concerning the often lengthy period of infection.

Therefore, in order to adequately explore potential psychosocial factors associated with the immunological parameters established in chapter 2, this chapter does not restrict itself to studies utilising only HIV-infected individuals. Instead, a general review of PNI studies investigating the pertinent immune measures is conducted, which is then followed by a more restricted review of viral infections, particularly herpesvirus. Finally, studies focusing specifically upon HIV-infection are reviewed.

### 3.2 GENERAL OVERVIEW

The paper 'Emotions, immunity, and disease' by Solomon and Moos (1964), is often regarded as the first study directly linking immune measures and emotional distress. A wide variety of factors producing emotional distress have been subsequently found to produce immunoattenuating effects, such as lower CD4 cell counts, lower NKA, and lower MSLP rates.

#### 3.2.1 LIFE EVENTS AND IMMUNE RESPONSE

Distressing life events frequently associated with immunoattenuation include examination anxiety in college students (e.g., Glaser, Kiecolt-Glaser, Stout, et al., 1985; Glaser, Rice, Speicher, Stout & Kiecolt-Glaser, 1986; Kiecolt-Glaser et al., 1986), unemployment (e.g., Arnetz et al., 1987), bereavement (e.g., Bartrop, Luckhurst, Lazarus, & Kiloh, 1977; Calabrese, Kling & Gold, 1987; Naor, Assael, Pecht, Trainin, & Samuel, 1983; Irwin, Daniels, Risch, Bloom, & Weiner, 1988; Irwin, Daniels, & Weiner, 1987), interpersonal conflict, divorce, affiliative loss and separation (Schmale, 1958; House, Landis & Umberson, 1988; Irwin, Daniels &

Weiner, 1987; Kiecolt-Glaser et al., 1987; Kiecolt-Glaser et al., 1988), Ioneliness (Kiecolt-Glaser, Garner et al., 1984; Kiecolt-Glaser, Ricker et al., 1984), and depression which appears to be associated with decreased NKA and TLC, but associations with CD4/CD8 counts and MSLP rates are less evident (Denney, Stephenson, Penick, & Weller, 1988; Evans et al., 1992; Irwin, Daniels, Bloom, Smith & Weiner, 1987; Irwin, Smith, & Gillin, 1992; Kamen-Siegel, Rodin, Seligman, & Dwyer, 1991; Kronfol & House, 1984; Levy, Herberman, Lippman, & d'Angelo, 1987; Marshall, 1993; Schleifer, Keller, Bond, Cohen, & Stein, 1989; Schleifer et al., 1984; Stein, Keller & Schleifer, 1985; Stein, Miller & Trestman, 1991a, 1991b; Weisse, 1992).

The emotional contexts of losses and bereavement are diverse, and are influenced by factors such as the acceptance of the loss, prior anticipation, and the closeness of the relationship. Depression may result from loss, and this may in itself complicate the interpretation of immunological changes.

Despite the emotional complexity of individual losses, it appears that such experiences are clearly linked to depressed immunological activity levels and, to a lesser degree, structural immunological deficits.

According to Kennedy, Kiecolt-Glaser, and Glaser (1988), the cumulative evidence regarding interpersonal relationships and immunity suggests that immunosuppression (as opposed to direct health changes) may be a possible pathway through which interpersonal conflict may contribute towards illness. The same could probably be said about loneliness, suggesting that methods or programs for dealing with both these factors may be useful in ameliorating their negative immunological consequences.

It also appears that the effects of distressing events of very short duration on certain aspects of the immune system occur rapidly, usually within 15 minutes, but that immune responses rapidly return to baseline levels (e.g., Kiecolt-Glaser, Cacioppo, Malarkey & Glaser, 1992; Moss, Moss, & Peterson, 1989; Knapp *et al.*, 1992; Zakowski, McAllister, Deal & Baum, 1992). Acute distress does not therefore appear to be a significant factor in understanding more pervasive immunological changes, and our attention thus shifts towards more long-term factors.

### 3.2.2 INDIVIDUAL COPING

A casual review of the research literature on coping strategies suggests that strategies involving avoidant tactics are effective in reducing pain, stress, and anxiety in some cases, whereas nonavoidant strategies (called here attention), appear to be more effective in others. ... (The question arises regarding) the role of different kinds of attentional sets and also the role of time - whether some kinds of strategies work best in the early phases of the stress experience, and others are more efficacious in the later phases of the stress experience. Results of an overall ... (meta-analysis of 43) studies providing tests of attention versus avoidance indicated little evidence for one's strategy's superiority. However, supplementary analyses ... suggest (that) there are boundary conditions that define the relative efficacy of a specific strategy ... (Suls & Fletcher, 1985, p.249; Emphases in original).

Methods of coping may produce different behavioural, emotional and physiological responses, which need to be examined in the context of the specific distressing event. For example, emotion-focused coping (as opposed to denial or problem-focused coping) was associated with lower levels of distress in persistently distressing situations such as residing near the Three Mile Island nuclear plant (Baum, Fleming, & Singer, 1983). In contrast, denial and problem-solving coping were associated with shorter hospitalization duration and less use of analgesics (Wilson, 1981). It thus appears that coping styles which are adaptive in one context, may be maladaptive in another.

There are a diversity of possible mediating factors in psycho-immunological interactions. There appear to be those who are habitually high and low reactors to events, irrespective of the amount of objectively measured life change. High reactors and those who report comparatively more symptoms in the face of high levels of life change ('poor copers'), also have significantly lower MSLP and NKA rates (Locke *et al.*, 1984; Zakowski *et al.*, 1992). Clearly, the amount of life change is not the critical criteria. Rather, it is the response to events that influences immune responses (Locke *et al.*, 1984).

The notion that individuals differ in how they respond to, and deal with, life changes and threats to their well-being is not new. For example, the putative role of attitudes and beliefs in illness and healing have been strongly entrenched in cultural ideology, specifically the role of religious faith (e.g. Dowling, 1984), spontaneous remissions (Kent, Coates, Pelletier & O'Regan, 1989), and the placebo effect (e.g. Beecher, 1955, 1961; Levine, Gordon, & Fields, 1978; Kirkpatrick, 1981). The question naturally arises as to whether stable attitudes and dispositions regarding responses to life threats have a differential effect upon immune function.

In a series of studies (Pennebaker & Beall, 1986; Pennebaker, Hughes & O'Heeron, 1987; Pennebaker & Susman, 1988; Pennebaker, Kiecolt-Glaser & Glaser, 1988), it was found that the inhibition of disclosing traumatic events is emotionally distressing, produces more frequent health-care utilisation, and that self-disclosure has long-term benefits by reducing distress and elevating MSLP rates.

Other studies have suggested that inhibited affect may result in predisposition to depression and chronic pain (Beutler, Engle, Oro'-Beutler, Daldrup, & Meredith, 1986), and may detrimentally affect perceived coping ability (Cioffi & Holloway, 1993), thus indirectly affecting immune function. In the same vein, confession of guilt is reported to relieve cognitive dissonance (Stice, 1992), and emotional or factual disclosure may differentially affect arousal (Mendolia & Kleck, 1993).

Regarding inhibited affect, Weinberger, Schwartz and Davidson (1979) found that it is important to distinguish truly low anxious people from repressors who report low levels of anxiety, particularly those low-anxious repressors who also have high measures of defensiveness, as indicated by measures of social desirability. In a comparative study, repressors revealed significantly higher levels of physiological distress than (non-repressor) low anxiety subjects, despite these repressors reporting equivalent levels of low anxiety.

The actual emotional response evoked by restimulated disturbing past events has also been associated with different immune responses (e.g. Knapp *et al.*, 1992; McClelland, Patel, Brown & Kelner, 1991). Knapp *et al.* (1992) suggest that certain "riddance" or purging emotions, such as sadness, shame and disgust, may well have a distinctly different effect to other negative emotions, such as anxiety. In this regard, it is interesting to note that Kemeny, Fahey *et al.* (1989) paradoxically found that bereaved HIV-positive men had higher (more active) responses to MSLP, while non-bereaved depressed HIV-positive men had lower MSLP rates. The nature of the relationship with the deceased, and whether the loss evokes sadness or anxiety, are presumably important determining factors. It thus appears that the mode of coping with significant losses has a significant impact upon immune responses. A pertinent issue in this regard would centre around the acquisition of good coping skills, the emotions associated with such acquisition, and the effects thereof upon immune responsivity.

Weidenfeld et al. (1990) conducted a study in which a group of snake phobics were taught skills designed to enhance their perceived sense of self-efficacy in dealing with their phobia. Overall immunoenhancing effects were found during the acquisition stage, and the total number of lymphocytes, CD4 and CD8-cells were uniformly higher after maximal efficacy was achieved. Initial perceived coping efficacy predicted both the direction and magnitude of immune responses to the anxieties experienced in the acquisition of coping skills skills. acquisition of not produced Rapid coping only immunoenhancement, but also resulted in greater retention of elevated immune levels after skill acquisition. Slow acquisition of coping skills, thus slower growth of perceived self-efficacy, resulted in immunoattenuation during acquisition, followed by a faster return to baseline immune levels after maximal skill levels were achieved. It thus appears that the act of learning coping skills may be experienced differently, depending upon the initial level of selfefficacy, and that this would need to be taken into account when considering the advocacy of such intervention programs.

Finally, stressed power motivation (SPMS) and unstressed affiliation motivation (UAS) syndromes have been often cited as differential factors in illness frequency and susceptibility, with stressed power motivation being associated with greater susceptibility in a variety of illnesses (Jemmott *et al.*, 1990). A meta-analysis of three studies conducted by Jemmott *et al.* (1990) revealed that SPMS was highly significantly related to lower levels of NKA, while UAS was found to be significantly related to higher levels of NKA.

### 3.2.3 SOCIAL SUPPORT

The importance of social support in the lives of those who are HIV-infected is multi-faceted (Namir, Alumbaugh, Fawzy & Wolcott, 1989). In the light of findings regarding loneliness and interpersonal conflict, it is not surprising that the role of social support in reduced psychological and physiologic morbidity, independent of other factors such as alcohol consumption, smoking, physical exercise, socioeconomic status, and utilization of health services, has been found in several studies (Thomas, Goodwin & Goodwin, 1985; Levy, Herberman, Whiteside et al., 1990; Levy, Herberman, Lee et al., 1990; Baron, Cutrona, Hicklin, Russell, & Lubaroff, 1990).

Similarly, McNaughton, Smith, Patterson, and Grant (1990), and Thomas *et al.* (1985), found that immunosuppression in elderly women appears to be associated with dissatisfaction with social supports and high levels of life change in the last year, whereas immunocompetence is associated with problem-focused coping and satisfaction with emotional support. CD4-cell counts were positively correlated with problem-solving coping, while CD8-cell counts were positively correlated with high life change and lower levels of satisfaction with emotional support.

### 3.2.4 PSYCHOSOCIAL INTERVENTIONS

A discussion of the effects of various psychosocial factors on immune measures would not be complete without mention of conditions under which immune function can be enhanced by psychosocial interventions. Intervention programmes such as relaxation or support groups have been found to buffer the effects of unemployment and examination anxiety (e.g., Arnetz et al., 1987; Kiecolt-Glaser et al., 1986), indicating that the links between emotional states and immune functioning are bidirectional. Perhaps the most disputed case concerns the reported case of a reversal of AIDS through healing imagery by William Calderon (Bolen, 1985; Braud, 1986). There are also numerous popular publications containing psychosocial programmes for various diseases, particularly cancer, and which emphasise the beneficial effects of relaxation and visualisation in reversing various physical diseases (eg., LeShan, 1989; Proto, 1990; Siegel, 1986; Simonton, Matthews-Creighton & Creighton, 1978).

Robert Ader and Nicholas Cohen's (1975) behaviourally conditioned immunosuppression in rats is often cited as an important precursor to later psychoneuroimmunological studies demonstrating that the immune system can be directly influenced by means of psychosocial intervention. According to Neveu, Crestani and Le Moal (1987), such behavioural conditioning of the immune system has resulted in the reconsideration of the immune system as a self-regulated system. A select sample of studies demonstrating that certain cognitive-behavioural strategies may enhance immune functioning, are thus briefly discussed.

In one such study, significant increases in NKA by means of relaxation and social contact in a geriatric sample was reported by Kiecolt-Glaser *et al.* (1985). Increases of NKA in humans via behavioural conditioning (Buske-Kirschbaum, Kirschbaum, Stierle, Lehnert & Hellhammer, 1992), and relaxation and guided imagery (Zachariae *et al.*, 1990) have also been demonstrated to be possible. Viral conditions, such as warts, have also been significantly reduced through hypnosis (e.g., Spanos, Williams, & Gwynn, 1990), and behavioural conditioning has been used to significantly reduce physiological responses to tuberculin (Smith & McDaniel, 1983).

Immunoglobulin A (IgA) is an important protective agent against various infections (Kirkwood & Lewis, 1989), and it is therefore interesting to note that levels of IgA have been significantly increased with imagery (Rider, Achterberg, et al., 1990) and imagery accompanied by music (Rider & Weldin, 1990).

Relaxation and biofeedback have likewise been used to increase IgA levels (Green, Green, & Santoro, 1988), as well as increases in MSLP rates (McGrady et al., 1992). Movies which aroused uninhibited affiliative motivation (as opposed to power motivation) also increased IgA levels (McClelland & Kirshnit, 1988), and positive emotional states and humour have also been found to positively affect IgA levels (Martin & Dobbin, 1988; Dillon, Minchoff & Baker, 1985).

These studies suggest that psychotherapy may have a beneficial effect upon immune measures and the reduction of emotional distress. A more extensive overview of psychosocial interventions can be obtained in reviews by Halley (1991), Hall and O'Grady (1991), and Kiecolt-Glaser and Glaser (1992).

### 3.3 PSYCHOSOCIAL FACTORS AND VIRAL INFECTIONS

Following from the evidence reviewed thusfar, the possibility that psychosocial factors can significantly affect viral infection progression arises.

It is clear that viral infection needs to be viewed as the consequence of two main factors, namely the presence of the viral agent itself, and the immune response to that agent at the time, which may be influenced by psychosocial factors. Therefore, in order to understand the possible effects of psychosocial variables upon the reactivation of latent viruses, various studies concerning mainly Herpes viral infection are examined.

### 3.3.1 CROSS-SECTIONAL STUDIES

Although evidence is presented concerning associations between various psychosocial factors and viral infection in the studies which follow, these studies are cross-sectional, and no direct causality can be implied from such associations. Nevertheless, the cumulative evidence is compelling.

Evidence exists for an immunoattenuating effect accompanying chronic distress, such as living close to the Three Mile Island (TMI) nuclear reactor. In one such study, McKinnon, Weisse, Reynolds, Bowles, and Baum (1989) found that TMI area residents had higher HSV-1 and CMV antibody levels than a comparable control group. TMI residents also had significantly fewer NK cells than controls.

Separation, divorce, and dissatisfaction with marital quality have also been associated with higher levels of EBV and HSV-1 antibodies in men and women (e.g., Kiecolt-Glaser *et al.*, 1987; 1988; Kennedy *et al.*, 1988). The poorer immune functioning indicated by elevated EBV antibody levels was accompanied by higher levels of depression and distress. It was concluded that women and men are more likely to have reactivations of latent EBV and HSV-1 when experiencing the loneliness, depression and distress of marriages of comparatively low quality, as well as separation or divorce.

Regarding social support, Glaser *et al.* (1992) found that medical students who seroconverted to hepatitis B inoculation and who had higher levels of social support, had stronger responses to the vaccine, as measured by both antibody titres and MSLP response.

Esterling, Antoni, Kumar and Schneiderman (1990) found that individual interpersonal styles accounted for a great deal of EBV antigen levels immediately after subjects had written a 30-minute essay relating a distressing life experience. Repressors had significantly higher EBV antibody levels compared to sensitizers. The degree of disclosure revealed in the essay further interacted with interpersonal style, with low disclosure levels associated with higher antibody levels. Studies such as this strongly suggest that individual styles or trait-like qualities may have significant consequences for immune functioning under certain circumstances.

### 3.3.2 PROSPECTIVE STUDIES

The major advantage of prospective studies over cross-sectional studies is that it often possible to explicate issues of causality, as it can usually be established which factor preceded the other, in terms of time. Furthermore, prospective studies enable the examination of effects which might only emerge over time, and which would otherwise not be apparent when using short-term cross-sectional measures.

For example, Luborksy, Mintz, Brightman, and Katcher (1976) found that daily mood states were totally unrelated to the short-term onset of HSV-1, but that a measurement of **general** unhappiness did predict HSV-1 frequency over the next 12 months. It thus appears that relatively stable emotional states, as opposed to short-term daily fluctuations of mood states, are important psychosocial factors to investigate in the long-term progression of viral infections, and that their effects may only be discernible over long periods of time. This confirms conclusions reached in section 3.2.1, in which events with acute distressing effects did not produce any significant long-term immunological changes.

Kasl, Evans & Niederman (1979), in a 4-year prospective study of 1327 military cadets, provided considerable evidence that psychosocial factors were implicated in the seroconversion to EBV, as well as subsequent development of clinical infectious mononucleosis (IM) in those who seroconverted. Composite scores reflecting the degree of over-achievement of the cadet's father were significantly higher in those cadets who developed clinical IM, as compared to those who developed subclinical or inapparent infection.

Furthermore, those cadets who developed IM were found to have significantly higher initial levels of motivation to their military career, compared to other seroconverters. When levels of motivation were related to ability to meet academic demands, as measured by academic achievement, this interaction was highly correlated with development of clinical IM. This effect was stronger in the last two years of the four-year course, presumably due to increased urgency to achieve in the final stages of the course. In years 3 and 4, cadets with a combination of high motivation and below-average ability to achieve represented 60% of those who developed clinical IM. Thus, demand-ability imbalances had significant predictive association with future illness formation, and this effect was enhanced by an increased pressure to perform.

In a later study, Glaser, Kiecolt-Glaser, Speicher, and Holliday (1985) found that medical students with high loneliness had significantly higher EBV antibody titers at all pre-exam, exam, and post-exam measurement times, while HSV-1 antibody levels decreased significantly from the exam to the post-exam points. This confirms previous findings regarding the immunological main effects of loneliness, independent of other factors.

Kemeny, Cohen, Zegans, and Conant (1989) investigated the relationship between HSV recurrence, negative mood states, and 'stressful' life events, measured monthly over six months. Overall stress scores and mood scales were found to be uncorrelated with HSV recurrence rates. However, subjects with high 6-month depression scores displayed twice as many episodes of HSV recurrences. The relationship between HSV recurrence and depression was independent of health activities such as alcohol consumption, exercise and sleep.

# 3.4 PSYCHOSOCIAL FACTORS AND HIV INFECTION

This section will largely (but not exclusively) be limited to research published from 1988 to the present, which coincides with the period following the publication of the Centre for Disease Control (CDC) case classification system in 1987 (Centers for Disease Control, 1987), which was the first widely accepted system for HIV symptom classification.

### 3.4.1 SOURCES OF DISTRESS IN HIV INFECTION

There are a diversity of sources of distress for those who are HIV-infected. Not only does a person who is HIV-seropositive have to face social hostility and stigmatisation, HIV-seropositive status has direct detrimental consequences for utilising health-care services if seropositive status becomes known, such as possible job discrimination, losing insurance coverage, and harassment (Herek & Glunt, 1988).

There are a multitude of sources of distresses for those who are living with HIV, starting with the anticipation of HIV-test results, discovery of positive seropositive status, coming to terms with an uncertain future, profound implications for relationships, including sex and intimacy, social isolation, depression, morbid preoccupation and fatalism, the possibility of future dependency on others, and many other related issues (McKusick, 1988; Kaisch & Anton-Culver, 1989).

Of these sources of distress, there are indications that distress associated with discovering HIV-seropositivity does not persist, but that the development of symptoms are a great cause for chronic distress (Kessler *et al.*, 1988). AIDS-related bereavement and coping with the notion of mortality has also been cited as a source of distress (Coxon, 1990). In this regard, Martin and Dean (1993) report that the intensity and duration of such losses seems to diminish over time.

Levels of distress are also not uniform across the different stages of HIV infection. Various studies indicate that people with ARC (non-life-threatening infections) have higher levels of distress and psychiatric morbidity than people with AIDS, as well as higher than those who are asymptomatic HIV-seropositive (e.g. Atkinson *et ai.*, 1988). Adjustment disorders are the most common conditions mentioned by these studies.

Current psychiatric morbidity is most frequently associated with a history of major affective or anxiety disorder. A further prominent factor associated with psychiatric morbidity is the number of physical symptoms present (Catalan, 1988).

Distress-related behaviour has also been linked to increased levels of high-risk sexual behaviour, by means of the utilization of sex as a coping behaviour (Folkman, Chesney, Pollack & Phillips, 1992).

The scope of this paper precludes a more detailed discussion of these issues, and the reader is referred to Dilley, Pies and Helquist (1989), McKusick (1988), and Kelly and St.Lawrence (1989), for a more detailed discussion.

### 3.4.2 COPING WITH HIV INFECTION

In the preceding sections, evidence has been presented for the immunosuppressive effects of various psychosocial factors, including interpersonal factors such as loneliness, social support, and affiliative loss. Chronic emotional states, particularly depression, as well as relatively stable individual qualities such as coping strategies and repressor and sensitizer interpersonal styles, have been demonstrated to have fairly robust functional immunological effects.

There is however a specific psychosocial coping dimension relevant to HIV infection that has not been addressed in previous non-HIV studies, namely dealing with the possibility of dying, and the absence of a medical cure.

In this regard, the maintenance of hope and the prevention of a sense of hopelessness is a central therapeutic issue when working with clients with HIV-spectrum conditions (Rabkin, Williams, Neugebauer, Remien & Goetz, 1990). In a review regarding hope and health status, Cohen (1988) states that:

A feeling of hope associated with either an increased sense that one can cope (or be helped by others to cope) with the problems with which one is confronted ... may be accompanied by biological changes that enhance physical as well as mental health. (Cohen, 1988, p. 106).

Rabkin et al. (1990), in a cross-sectional study comparing HIV-positive (excluding those with diagnoses of AIDS or neutrologic disorders) and HIV-negative homosexual men, found significant positive correlations between hopelessness and social conflict, external locus of control, and depression. Insignificant correlations with the sum of negative events, HIV symptoms and CD4 cell counts were found. Negative correlations with hopelessness were found with perceived emotional and material support, affirmation, objective and subjective social integration, the sum of positive life events, internal locus of control, and commitment. Due to the cross-sectional nature of this study, the role of hopelessness in the progression of illness could not be determined. However, Rabkin et al. (1990) state that the absence of significant correlations between concurrent hope and physical health were not surprising, as other studies (e.g., Temoshok, 1988, unpublished data, cited in Rabkin et al., 1990) suggest that such psychological measures are more predictive of future health and immune measures than of concurrent health status.

The association between intrusive or avoidant thoughts is closely linked to the subjective impact of an event (Horowitz, Wilner, & Alvarez, 1979). It is therefore interesting to note that, in a study of HIV-infected individuals by Perry, Fishman, Jacobsberg and Frances (1992), the only psychosocial variables which proved significant predictors of CD4 counts 12 months later were intrusive thoughts items from the Impact of Events Scale (IES, Horowitz, Wilner, & Alvarez, 1979), and two individual items from the Brief Symptom Inventory (BSI, Derogatis & Melisaratos, 1983) and the Beck Depression Inventory (Beck, Ward, Mendelson, Mock & Erbaugh, 1961). The fact that both of these latter two items measure hopelessness may be significant.

Related to hope, the concept of self-efficacy, i.e., the sense that one can personally make necessary changes to improve health, is important for both the psychological and physical well-being of people with HIV-spectrum infection. Conversely, the emphasizing of the negative can have detrimental consequences for both physical and psychological health (Blaney, Millon, Morgan, Eisdorfer, and Szapocznik, 1990). Behavioral mechanisms, such as the attribution of responsibility for health behaviours as internal or external, and biological mechanisms, such as distress-hormone immunosuppression when self-efficacy is low, may partially explain the effect of negative focusing on health, which may be exacerbated in persons who already have compromised immune systems (Blaney et al., 1990).

Goodkin, Blaney et al. (1992) investigated the association between Natural Killer-cell cytotoxicity (NKCC) and three psychosocial factors, namely coping style, distressing life changes, and social support, in HIV-infected homosexual men. NKCC was examined as it is an important defense against viral infection, and decrements in NKCC are associated with HIV-1 infection, particularly in symptomatic stages.

Only one of the psychosocial variables was significant in predicting NKCC, namely the active coping composite score, while the coping strategy of focusing upon and venting of emotions (reminiscent of the sensitizer interpersonal style previously discussed) approached significance. Only the interaction between life changes and social support approached significance, indicating that social support has a beneficial effect upon NKCC for those with a lower number of life change counts. Goodkin, Blaney et al. (1992) utilised the Profile of Mood States (POMS; McNair, Lorr & Droppleman, 1971) composite distress scores, but unfortunately did not examine possible correlations of NKCC with the various POMS subscales, such as the Fatigue-Inertia subscale, which previous studies (e.g., Levy et al., 1987) have found to be significantly negatively correlated with NK activity in breast cancer patients. Other studies of HIV which have utilised the POMS have also found significant differences between various HIV-symptom groups with respect to other POMS subscales (e.g., Atkinson et al., 1988).

One such study (Ostrow, 1988) found that the POMS subscales for confusion, depression-dejection, fatigue-inertia, and tension-anxiety, were all significantly positively correlated with TLC (p < 0.04 for all subscales), while the vigour-activity subscale was negatively correlated with TLC (p < 0.10). The POMS anger-hostility subscale was not significantly correlated with TLC. However, subsequent longitudinal comparisons between ARC patients who were still alive or deceased, revealed that those who were later still alive had previously scored low on measurements of social desirability, and high on the POMS anger-hostility subscale. These results are consistent with outcome studies of cancer patients regarding the negative survival implications of the so-called Type-C coping style, and the positive benefits of having a "fighting spirit".

In a less comprehensive study of 11 asymptomatic HIV-infected homosexual men focusing upon the number of major life change events in the previous year and coping style, Goodkin, Fuchs, Feaster, Leeka and Rishel (1992) found that life-change event scores were negatively correlated with CD4 and overall TLC. Conversely, active coping style was significantly positively correlated with CD4 count, and correlations with CD4:CD8 ratios and TLC were likewise positive, but did not attain significance. The independence of the life change scores and coping constructs was also apparent in this sample. NKCC was not significantly correlated with any other immune measures, and exhibited correlations approaching zero with the psychosocial variables. This is not surprising, as previous studies have found that NKCC changes are more pronounced in symptomatic stages of HIV-1 infection (Goodkin, Blaney et al., 1992). This fact, in combination with the small sample size of only 11 subjects, may have obscured any significant NKCC changes. Interaction effects between high/low life-change events and active/passive coping revealed that the highest CD4 counts and TLC were found in low life-change/active copers, with high lifechange/passive copers having the lowest CD4 counts. The other combinations were intermediate between these two extremes in terms of CD4 counts and TLC. It thus appears that the individual coping style becomes more important in conditions of high life change or challenge.

Two prospective studies of HIV-infected subjects, namely Perry *et al.* (1992) and Rabkin *et al.* (1991), found little evidence that a wide variety of psychosocial factors, including trait anxiety, depression, and various psychiatric symptoms, were predictive of CD4 cell counts 6 and 12 months later. However, both studies failed to measure coping strategies or interaction effects between any of the psychosocial variables.

Both studies did however find cross-sectional support for previous studies (e.g., Ostrow et al., 1989), regarding the association between the severity of current HIV-related symptoms and diverse psychological variables, such as depression, trait anxiety, psychiatric symptoms, intrusive and avoidant thoughts, hopelessness, and demoralization. These correlations were also significant across measurement points, which suggests that the severity of initial physical symptoms predicts subsequent psychosocial distress levels.

In contrast to the latter two studies, Solano *et al.* (1993) utilised three stories which elicited the HIV subject's attitudes towards the illness, as well as a social support scale and the UCLA Loneliness scale (Russell, Peplau & Cutrona, 1980). Furthermore, they distinguished between low and high CD4 counts, and included a functional immune test relating to skin response to various antigens. The 100 asymptomatic subjects (21 stage II and 79 CDC stage III) were assessed at baseline, 6 months and 12 months later.

They found that fighting spirit and denial/ repression attitudes had significant impact on the course of HIV infection, at the 12 month follow-up. Social support and psychological distress emerged as significant factors only in subjects with immune systems which were already compromised. No significant effects were found for other independent psychosocial variables, including education, time since diagnosis, social support, loneliness, relationships with parents, and psychological distress. The results at the 6 month comparisons were similar but less significant than those at 12 months, indicating that the impact of those psychosocial variables which emerged as significant predictors of disease progression are more noticeable over longer periods of time.

### 3.5 CONCLUSIONS

In this chapter, a number of factors have been found to be significantly associated with immune functions central to HIV infection. It appears that events producing acute distress have little enduring immunological impact, but that long-term emotional distress and chronic distressing situations have a negative impact upon the containment of viral infections. Furthermore, the individual perception of, and reactions to, potentially distressing events seems to be more important than the event itself, in terms of immunological consequences in viral infections. Clearly, relatively stable coping methods and emotional states are more important factors in psycho-immune associations in viral infections, particularly over lengthy periods of time, such as HIV infection.

There are also suggestions that problem-focused methods of coping are more effective for dealing with short-term distressing events, while emotion-focused coping methods appear to be associated with beneficial immunological consequences in chronically distressing situations. In this regard, the repression of emotions appears to a consistent correlate with immunoattenuation in viral infections, and it seems that repressors tend to report false low levels of distress and anger, as well as higher levels in measurements reflecting social desirability.

The impact of depression and loneliness, independent of many other factors, have also been found to be associated with negative immunological conditions in viral infections, although these effects were less evident in studies of HIV-infected individuals.

Ability-demand dynamics, particularly the combination of a desire to achieve with an inability to attain desired goals, are also implicated in the activation of latent Herpesvirus.

It is interesting to note that interpersonal factors, such as social support, divorce and separation, and general loneliness, appear to be consistently associated with poorer immunological conditions, as indicated by higher viral antigens levels. The robustness of these effects is evidenced by the number of studies which have found such negative immunological associations.

The immunological effects of social support are more difficult to discern, and appear to be more evident in situations of high life change, or in those who already have compromised immune systems.

Although associations between immune functioning and certain psychosocial variables (e.g., active coping and repressor-type interpersonal styles) have been found in several of the reviewed studies, several methodological problems in such studies are evident. Most notably, the complexity of the immune system, and the length of time necessary for effects to emerge. Furthermore, the interactive nature of both psychosocial and immune variables seems to necessitate methods of analysis which examine the interaction between different psychosocial factors, such as high life change and social support, or demand-ability imbalances.

A notable methodological weakness in HIV studies examining associations between psychosocial factors and CD4-cell counts, is the application of simplistic analytic methods, such as the analysis of the main effects of psychosocial factors upon CD4-cell counts at different intervals of time. This approach fails to take into consideration the interactive nature of psychosocial factors, and fails to consider the lengthy period of time involved for such variables to have potentially discernible immunological effects. This approach also tends to overlook the diversity of individual HIV illness progression, which might be more meaningfully approached in terms of the individual rate of CD4cell decline, as opposed to the use of CD4-cell counts. Analyses utilising CD4cell counts would be more susceptible to shorter-term fluctuations (and thus obscure existing associations with psychosocial variables), while rates of decline since infection would be less likely to fluctuate due to the longer period of time over which it is calculated. By using the rate of CD4-cell decline, comparative investigations of diverse grouping of HIV-infected individuals would be enabled, such as those who rapidly deteriorate after infection, compared to those who have remained asymptomatic for longer than most. This would place the emphasis upon factors which are associated with such longer-term differences, with implications for intervention strategies.

When it is considered that the median time for development of symptoms is approximately 7 or 8 years from time of infection, even a prospective study extending for up to a year may fail to indicate the immunological effects of various psychosocial factors.

The use of the time of diagnosis is also questionable, as diagnosis may occur at any time before, during, or after the emergence of symptoms. The effects of apparently stable psychosocial variables on current and future immune functioning in HIV infection thus seem to require studies which examine infection over lengthy periods of time in order for any significant associations with immunological changes to emerge.

# **CHAPTER 4**

# RESEARCH DESIGN

### 4.1 INTRODUCTION

Chapter 3 presented evidence linking certain psychosocial factors (ē.g., coping strategies, loneliness, hopelessness, depression, repressor-sensitiser interpersonal styles, and social support) with immunological changes. This chapter focuses upon the research design and methodology used in the present study for investigating the associations between predominantly stable psychological constructs and the rate of progression of HIV infection, defined in terms of the rate of CD4-cell decline over time.

Due to the lengthy period of time involved in HIV infection, a retrospective methodology is employed, and the methodological restrictions and advantages surrounding the issue of the direction of causality are explored, centred upon the stability of the various psychological constructs over lengthy periods of time. The rationale for using exclusively homosexual HIV-seropositive males is also discussed within the South African context, as well as methodological restrictions pertaining to confidentiality and anonymity in HIV research. Thereafter, the measurement instruments selected, notably the Coping Orientations to Problems Experienced (COPE; Carver, Scheier & Weintraub, 1989), the Profile of Mood States (POMS; McNair *et al.*, 1971), the Revised UCLA Loneliness Scale (Russell *et al.*, 1980), and four single items, are described and evaluated in terms of internal and construct validity.

The clinical aspects of HIV infection, such as establishing the time of infection, symptom status, and measurement of CD4 and CD8-cell counts are discussed. Methods utilised to reduce error variance (e.g., correcting cell-counts for the known effects of medical drugs) within a context of an opportunistic study are evaluated, as all immunological measurements are derived from data gathered from a concurrent drug-trial. Finally, the required sample size necessary for detecting significant differences between groups are ascertained as being between 20 and 34 subjects, based upon analyses of effect sizes from prior research findings.

### 4.2 RATIONALE AND OBJECTIVES OF THE STUDY

Evidence for significar it associations between certain stable individual characteristic and immune functioning has been found in the reviewed literature. However, there appears to be an absence of information regarding the association between such factors and the progression of HIV infection over periods of time exceeding a year, although long term prospective studies in this area are currently being conducted (e.g., Solomon *et al.*, 1987).

Therefore, this present study seeks to examine whether certain stable psychosocial factors, specifically dispositional coping strategies, loneliness, repressor-sensitizer styles and hopelessness, are significantly associated with HIV-infection progression from the time of infection until the most recent measurements of CD4 and CD8-cell measurements, i.e., the entire length of the period of infection, regardless of symptom-status. The purpose of investigating these associations is to establish a basis for further (prospective) research into the possible causal role of such psychosocial variables in HIV illness progression.

Additional objectives of the study include a descriptive profile of coping methods and emotional states of HIV-infected people at different stages of infection, as well as a brief survey of the needs of people infected with HIV. The purpose of both the descriptive profile and survey of needs of people at different stages of HIV-infection is to identify areas where organised intervention would be most beneficial for those infected.

For reasons elaborated upon later in this chapter, this study will focus upon homosexual males, and there are consequently several references to normative data for homosexual males regarding the various research instruments in this chapter.

A basic objective of the present study is to focus upon psychosocial factors which are amenable to change by the individual, in order to establish a basis for future investigations of the effects of modifying such factors on illness progression. Therefore, the emphasis is upon those factors over which a person who is HIV-infected has some element of control. For example, the seeking of social support and dispositional loneliness are of interest to this study, as opposed to objective social network sizes or life change events.

As no study can measure all possible factors, only a few of the psychosocial factors which have emerged as potentially significant associates of illness progression in the literature are examined in this study. These are coping methods (including the seeking of social support), repressor-sensitizer interpersonal styles, loneliness, hopelessness, and measures of emotional distress, such as anxiety, depression, and anger-hostility.

### 4.3 RESEARCH DESIGN: ISSUES OF TEMPORALITY

A basic design requirement centres around temporality, as HIV-infection occurs along a continuum of time, progressing from stage to stage over a number of years. Furthermore, it has been indicated that the effects of psychosocial variables may likewise only become apparent over a lengthy period of time.

The most obvious choice of design would be a prospective study, which would follow a cohort of HIV-infected people from initial infection until symptom development. This would require a study conducted for at least 7 to 8 years, which is considered the median time from infection until development of symptoms. Such a study would probably have the ability to clearly explicate issues of causality if baseline measures of the psychosocial variables are conducted, and if it can be assumed that such psychosocial factors remain stable over time.

However, until such time as a medical cure is found, alternative avenues which can be explored in the short- to medium-term need to be found, thus indicating that a cross-sectional study would be of greater immediate practical benefit for those infected. For example, if significant associations between psychosocial factors and the rate of CD4-cell decline over time are found, intervention programmes which address these factors could be implemented in the short-term, and their effects prospectively monitored in order to determine causality.

In contrast, if the present study is conducted prospectively over the necessary period of time, the results of the study could only begin to be implemented after the completion of such a study, by which time many currently HIV-infected people may have already succumbed to the disease. Thus, practical considerations necessitate that the present study directs itself towards identifying those psychosocial factors which are **most likely** to be causally implicated in the speed of HIV illness progression, the latter being defined in terms of the rate of CD4-cell decline since infection.

As randomisation to HIV-illness categories is not a methodological possibility, a non-randomised quasi-experimental design is indicated. Furthermore, in order to incorporate the temporal aspect previously mentioned, the study would need to incorporate retrospective measurements of the period of infection and dates of subsequent symptom development.

### 4.4 SAMPLING PROCEDURES AND CONSIDERATIONS

In all of the studies reviewed regarding HIV-infection, homosexual males have been used as subjects. The main reason for the focus upon this population is historical, as the AIDS epidemic was originally documented largely in the homosexual men of New York, San Francisco, and other large cities in the USA. However, the disease is now found across the globe, and currently affects heterosexual males and females in large numbers, with the heterosexuals being the largest group of infected people in Africa.

The spread of the disease to the heterosexual population has had a profound effect upon the sociopolitical context of AIDS education and research, as agencies have had to educate the general population regarding the fallacy that AIDS is confined to homosexual men, and to enforce the notion that everyone who engages in sexual activity needs to consider their risk for infection.

However, despite this change in the epidemiological character of AIDS, most PNI studies continue to utilise homosexual males as subjects. A major factor for this continued focus is that homosexual males tend to be better informed and educated about sexual risk factors, and generally have had far greater exposure to the consequences of infection. Particularly in studies using self-reported HIV-related information, homosexual males are considered to be more likely to provide informed responses. Furthermore, organisations serving homosexuals were the first to mobilise to care for those infected, educate those at risk, and lobby governments and institutions for research and funding. It is therefore not surprising that homosexual males tend to be quite willing to volunteer for research in the area of AIDS.

There is, however, the possibility that the continued focus on this population may serve to counter efforts to change the perception of AIDS as a homosexual disease, although it is reasonable to assume that the readers of published research would be likely to be aware of the true epidemiological nature of the epidemic.

In the current sociocultural context of first world nations such as the USA, PNI research using heterosexuals as subjects may be considered a viable option. However, in third-world nations such as South Africa, there are many social, political and economic disparities which may confound interpretations of results from PNI studies. For example, widespread poverty amongst economically disadvantaged communities produces confounding factors such as malnutrition, which may be a major factor in determining immunological progression of HIV infection, as well as the presence of untreated concurrent diseases such as tuberculosis and hepatitis. Additional factors, such as access to adequate health services, also serve to reduce the validity of findings from PNI studies using heterosexual populations in Africa.

Therefore, the present study will include only homosexual urbanised males who utilise the services of the Cape Town branch of ASET (Aids Support and Education Trust), which is an organised educational and service agency affiliated to the homosexual counselling and education service, GASA 6010.

Although it has been previously stated that homosexual males tend to be willing volunteers for research concerning AIDS, there is also a sense of wariness (and weariness) regarding psychosocial research in this area, as there is the perception that such research often holds little direct benefit for those who participate. Furthermore, due to the social stigmatisation of those who are HIV infected, confidentiality and anonymity are considered to be a basic prerequisite for any such research.

Therefore, a standardised questionnaire which could be completed in the absence of the researcher, and research procedures which guarantee anonymity of the respondents, are necessary. In addition, a cover letter which emphasises the potential benefits for the respondents, and which explicitly describes steps taken to ensure anonymity of the respondents, were considered essential.

In order to comply with these requirements, a cover letter (refer Appendix 1, i) was constructed. The cover letter clearly states the purpose of the study, namely to ascertain whether coping methods are associated with HIV progression. The theoretical basis, namely 'stress' research, is mentioned, and the respondent is informed that it is as yet unclear which methods of coping are most beneficial to those infected by the HIV.

Respondents are further informed that the researcher intends to use the data obtained in the present study in order to design a programme for enhancing coping skills in those infected, as well as the intention to provide the information regarding specific needs of those infected to ASET.

A general description of the proposed sampling procedure is explicitly stated in the cover letter, and it clearly stated that at no time will the identity of the respondent be known to the researcher. As stated, the researcher would provide batches of the questionnaire to ASET, who would then distribute the questionnaire. All questionnaires are entirely anonymous, and respondents are provided with a stamped envelope which is addressed to the researcher. In this manner, respondents were ensured that their anonymity is guaranteed.

The actual sampling procedure followed the above general outline, in that the questionnaires were delivered to a counsellor of considerable experience in the field of AIDS at ASET, who then distributed them to clients at the clinic, which occurred once a week. The questionnaire was distributed only to those people utilising the clinic and who were participants in an ongoing drug-trial, so that immunological data for each person was available. This occurred over a period of three months, during which the majority of the durg-trial participants completed the questionnaire. The total number of participants was 31, as the majority of drug-trial participants utilised another clinic.

The nature of the study and the required sample was clarified prior to the commencement of the study, and problems regarding the validity of self-report data obtained from those with neuropsychiatric symptomology were discussed, and it was agreed that such people would not be included in the study. Therefore, only people who had no signs or symptoms of neuropsychiatric disorders related to HIV infection, as determined by medical examination, were included in the study.

As previously mentioned that, in order to obtain immunological measurements for the participants of this study, it was necessary to utilise only HIV-infected individuals who are participating in another study, a clinical drug-trial. As this drug-trial is ongoing at the time of writing, the identity of the drug can not be disclosed, except that the drug is not an antiretrovial agent, and has no known toxic properties or serious side-effects. The drug is an naturally occurring compound with immuno-enhancing properties.

Participants in the drug-trial all had immunological measurements done at the beginning of the trial, and then at 3 to 5-month intervals thereafter. As the participants in the drug-trial did not commence the drug-trial at the same time, the questionnaire distributed as part of this present study was completed by participants who had been participating in the drug-trial for periods ranging between five to zero months (i.e., had recently enrolled in the drug-trial).

The CD4 and CD8-cell counts and other medical information pertaining to symptom-status for validation of reported symptomology were obtained from medical records of each participant. Information from the medical files were accessed by ASET staff after completion of the questionnaire, and the required data was then anonymously inserted in each questionnaire.

This was made possible by virtue of the fact that only two or three persons completed the questionnaire each week, and their medical files were filed according to their dates of birth, which was also indicated on the questionnaire. Additional demographic and symptom-status information contained in the questionnaire was then utilised by the ASET counsellor to verify that the correct medical file was being accessed. This method proved to highly effective in obtaining the correct medical information without violating the confidentiality or anonymity of the respondent, as the researcher did not at any time have contact with such confidential files.

It should be noted that it is assumed that the sample will be comprised of HIV-1 infected individuals, as medical files reveal that all subjects participating in the drug-trial have tested positive for this specific strain of the HIV. As there is little reason to expect significant differences regarding immunological functioning from samples obtained in the United States of America (USA), normative immunologic data from USA studies (eg., Lang et al., 1989) will be used as control norms. No comparable existing data for South African homosexual males was located for control purposes, for both immunologic and the specific psychosocial measurements. Due to the methodological difficulties involved in accessing a comparable control group of homosexual males with medically verified HIV-seronegative status, data obtained in USA studies will be utilized as control data for all psychosocial measurements.

### 4.5 ESTABLISHING CAUSAL DIRECTION

The main disadvantage of a cross-sectional study concerns the loss of clarity regarding causal direction. For example, if those who become symptomatic sooner after infection than average are demonstrated to be low-active (passive) copers, it would be difficult to ascertain whether the passive coping style preceded symptom-development, or whether the development of symptoms led to a sense of despair, and thus a more passive coping style.

An empirical association between a psychosocial variable and health status may ... reflect some process other than one in which that variable is causally antecedent to health change. Often, there is good reason to suspect that disease, or processes related to disease, have exerted an influence on the 'risk factor'... (For example), the disease itself may produce behaviourally relevant neurological, endocrine, and metabolic changes. As a result, early stages of the disease may be associated with prodromal symptoms such as fatigue and depression. It must also be considered that a correlation between a suspected psychosocial risk factor and health status may be brought about by the operation of some third factor, with no direct causal effect in elther direction between behaviour and disease. For example, ... psychosocial variables ... (such as) socio-economic status, cultural background, and personality traits, ... (may be) associated with (other) risk factors ... which may not be of interest to the investigator, such as cigarette smoking... This type of confounding can produce data giving the false suggestion of an aetiologically significant relationship for the putative risk factor of interest. (Contrada & Krantz, 1987, p.59).

Despite this serious methodological disadvantage of cross-sectional studies, Cook and Campbell (1976) state that it is not impossible to make inferences regarding directions of causality in such a study, if certain methodological criteria are met:

Experiments are conducted to make decisions. ... (When all threats to the validity of the causal direction of a relationship cannot be excluded), ... then the investigator has to conclude that a demonstrated relationship between two variables may or may not be causal. Sometimes he (sic) will have to act as if the relationship were causal because practical decisions have to be made and it is possible that the relationship may be causal. At other times, the alternative interpretations may seem implausible enough to be ignored and the investigator will be inclined to dismiss them. He (sic) can dismiss them with a great deal of confidence when the alternative interpretation seems unlikely on the basis of findings from a research tradition with a large number of relevant and replicated findings. Often, however, it will be difficult to obtain high inter-judge agreement about the plausibility of a particular alternative explanation. (Cook & Campbell, 1976, p.230; pp.229-230; emphasis in original).

According to Cook and Campbell (1976), causal direction can only be established if (a) temporal antecedence can be established, (b) the two variables vary together (statistical conclusion validity), and (c) alternative explanations can be dismissed (threats to internal validity and construct validity). They further state that the locus of causality is strengthened by using tests of adequate statistical power (i.e., low probabilities for Type I and Type II errors), the utilisation of a homogenous sample which would reduce error variation due to subject characteristics, and the use of reliable measurement instruments, which further reduces error variance. Between-group designs would also be more likely to indicate directions of causality, compared to within-subject designs.

Ambiguity regarding causality can be substantially reduced if it can be established that the psychosocial coping constructs used in the study are trait-like and stable over considerable periods of time, and do not change despite immunological decline. In order to investigate this possibility, we shall focus upon symptom development in HIV infection, which represents a major source of distress which are quite likely to precipitate changes in habitual coping method and emotional states. Symptom development is closely associated with CD4-cell counts reaching low levels, and thus represents the manifestation of immune decline over time.

If it can be established that trait-like coping methods remain unchanged despite symptom development, temporal antecedence of the psychosocial factor can be inferred, and we can tentatively assume that, for example, a currently symptomatic person who is a passive coper, probably was a passive coper before the development of symptoms. It could then be reasonably deduced that the decline of CD4-cells over time does not cause significant changes in habitual coping methods. Evidence regarding the stability of such psychosocial constructs is therefore essential if the plausibility of the causal role of these psychosocial factors in HIV illness progression is to be reasonably entertained as one possible explanation of such associations.

It should be clearly stated that, in a study of this nature, causality can never be 'proved'. Therefore, the purpose of this section is to determine whether the hypothetical causal association between psychosocial constructs and illness progression can be *plausibly dismissed*. We thus turn our attention to evidence regarding these issues.

In a study of coping methods designed specifically to investigate the possibility that specific coping methods are situational-specific (as opposed to situation-independent), and related to the severity of the event, Billings and Moos (1981) found that there was no correlation between the severity of the situation and the coping methods used. The situations investigated included illness, death, economic problems, children, and various interpersonal and non-interpersonal situations. The coping strategies measured included active cognitive, active behavioural, avoidance, problem-focused, and emotion-focused coping strategies.

A previously discussed study of HIV-infected homosexual men (Goodkin, Fuchs et al., 1992) similarly found that life change scores (i.e., the frequency and severity of 'stressful' life events), was statistically independent of the measured coping methods. Both studies indicate that people do not change their characteristic coping methods to suit the severity or frequency of situational distress, including illness, for periods of up to a year.

There is also direct evidence that, for example, active coping has a positive linear relationship with Natural Killer Cell Cytotoxicity (e.g., Goodkin, Blaney *et al.*, 1992), thus indicating that coping methods and immune measures do indeed co-vary.

Although it might be assumed that attributional dispositions (e.g. hopelessness) and loneliness might be situationally influenced, particularly by symptom development in HIV infection, this does not appear to be the case. For example, Rabkin *et al.* (1991) found that there was no significant change in hopelessness measures in HIV-infected homosexual males over 6 months, despite the fact that there were significant increases within the sample regarding symptoms across measuring points.

Rabkin *et al.* (1990) also found that, contrary to expectations, there was no association between symptom scores and the level of hope, and no differences in levels of hope between those with and without HIV symptoms. It thus appears that hopelessness is relatively independent of HIV-related situational factors and the severity of the illness, indicating that it is, like coping methods, relatively stable over periods of time of up to a year.

Similarly, Ioneliness (measured by the Revised UCLA Loneliness Scale of Russell et al., 1980), had no significant main effect in comparisons between HIV-seropositive subjects whose symptom-status remained unchanged versus changed, with respect to clinical evolution of the illness when assessed at both 6 months and 12 months post-baseline.

The stability of measurements of loneliness is further evidenced in other studies (e.g., Longo, Clum & Yaeger, 1988), in which loneliness scores of herpes-infected waiting-list control subjects remained relatively unchanged from the pretreatment to the post-treatment, the 3-month, and the 6-month follow-up points. Interestingly, those subjects who received coping skill interventions had significant reductions in loneliness. The latter finding, along with other studies involving coping-skill interventions (e.g. Weidenfeld *et al.*, 1990) serves to inject a note of caution regarding the stability of coping methods, hopelessness, and loneliness, as they are clearly amenable to change through direct psychosocial intervention, although it seems that they are otherwise stable over time. It would thus be important to establish whether subjects have received any such intervention, as this may possibly confound associations between cross-sectional measuremer. So of coping and symptom-status.

Given the evidence supporting the stability of coping and other constructs mentioned, it is not surprising that prospective studies incorporating coping constructs (e.g. Solano et al., 1993) tend to measure coping methods only at baseline, and regard such measurements as applicable to follow-up periods of time. Therefore, a cross-sectional measurement of such constructs may be equally valid as a retrospective indication of such individual qualities, at least for similar periods of time. This does not, however, imply that it can be stated with any degree of certainty that such constructs are indeed stable over considerably longer periods of time. Instead, the evidence merely suggests that this possibility seems quite plausible over periods of at least 6 months to a year, thus implying temporal antecedence of dispositional coping methods in associations with immune or symptom changes for such periods of time.

Thusfar, the evidence seems to suggest that certain psychosocial factors, including dispositional coping methods, loneliness and hopelessness, appears to remain stable over periods of up to a year, despite intervening life events and symptom developments. However, there are several studies which have criticised the assumption of consistency in coping methods over time or even that coping method measurements reflect actual coping behaviour (Cohen, 1987). There is also evidence that people use different methods of coping at different stages of a distressing event (Cohen, 1987).

It thus seems clear that a dispositional measure of coping will not be able to characterize the array of coping strategies used in dealing with a complex stressful event. However, to the extent that dispositional coping measures tap general dimensions of personality, rather than tendencies to cope in a particular way, they may be meaningfully related to health-relevant outcomes and show good predictive validity (Cohen, 1987, p.287).

In summary, several of Cook and Campbell's (1976) criteria for establishing causal direction have been addressed, and it has been found that coping methods, loneliness, and the sense of hopelessness are fairly stable over periods of up to a year, and do not change significantly despite illness and other disruptive life events.

# 4.6 VALIDITY AND RELIABILITY OF INSTRUMENTS

Prior to a discussion regarding the construction of the research instrument and validity and reliability of the scales it contains, it should be mentioned that a pilot questionnaire, containing several additional scales to those discussed in this section, was distributed to a small sample of homosexual males (N = 35) of unknown HIV-status in order to ascertain the length of time taken to complete each scale, and willingness to complete such a questionnaire. Only five people (14 %) returned completed questionnaires, and the general feedback from most of those included in the pilot study indicated that the questionnaire was too long. Those who completed the questionnaire reported an average of more than 1 hour to complete the questionnaire. The general consensus was that the length of the pilot questionnaire reduced willingness to complete the questionnaire.

Therefore, several changes were made, including the use of the shorter survey version of the UCLA Loneliness Scale (Russell *et al.*,1980), and the elimination of a number of standardised scales. Substantial reductions in the number of items in the survey of needs was also made.

Subsequent pilot applications of the revised questionnaire indicates that it takes approximately 15 to 25 minutes to complete, and it is expected that this would increase the willingness to participate in the study.

The final (revised) research instrument (refer Appendix 1) consists of 126 items pertaining to standardised psychosocial measurements, 10 items concerning demographic details, 2 items related to current HIV-serological status, 2 items probing the date/period of infection and certainty regarding the reported time, 13 current and recent HIV-related items concerning symptom-status, 1 item regarding antiretroviral or similar medication, and 11 items (of which 9 are open-ended) concerning needs for services.

### 4.6.1 COPING METHODS

According to Carver *et al.* (1989), the coping construct comprises numerous conceptually distinct responses. The following description of coping by Billings and Moos (1980, pp.140-141) serves to indicate the complexity of the construct:

Individual's attempts to cope with life usually have been viewed as a complex set of processes directed toward moderating the impact of such events on their physical, social, and emotional functioning. Conceptualizations of the nature and role of coping responses and related measurement strategies have evolved rapidly in recent years (see Coelho et al., 1974).

One such approach has viewed coping as a cluster of primarily intrapsychic processes (such as denial) by which an individual's emotional functioning is protected from external and intrapsychic threat. This line of inquiry has included research on ego-defence mechanisms (Haan, 1977; Vaillant, 1977) and trait definitions (such as the sensitization-repression dimension) of coping ...

More recent approaches have broadened the conceptualization of coping to include cognitive and behavioral responses attempting to deal with the external stressor as well as behavioral responses that serve to avoid the problem (Lazarus, 1980; Moos, 1976, 1977)...

One formulation of coping... has divided active attempts to resolve the stressful event into cognitive (intrapsychic) and behavioral strategies, while separately clustering responses which attempt to avoid the problem or reduce the emotional tension associated with the stressor (Lazarus, 1966; Moos, 1977). Active cognitive coping includes attempts to manage one's appraisal of the stressfulness of the event, ... (while) Active-behavioral coping refers to overt behavioral attempts to deal directly with the problem and its effects... Avoidance coping refers to attempts to avoid actively confronting the problem... or to indirectly reduce emotional tension by such behavior as eating or smoking more.

Another formulation is composed of two categories concerned with the focus of coping: problem-focused and emotion-focused (cf. Antonovsky, 1979; Lazarus, 1980; Pearlin and Schooler, 1978). Problem-focused coping includes attempts to modify or eliminate the sources of stress through one's own behavior. Emotion-focused coping includes behavioral or cognitive processes whose primary function is to manage the emotional consequences of stressors and to help maintain one's emotional equilibrium.

Cohen (1987, pp.283-284) further defines coping as:

(Coping may be viewed as) efforts, both action-oriented and intrapsychic, to manage (that is, to master, tolerate, reduce, minimize) environmental and internal demands, and conflicts among them, which tax or exceed a person's resources' (Cohen and Lazarus, 1979, p.219). Coping can occur in anticipation of a stressful confrontation or in reaction to a present or past situation. ... Thus, 'coping' and 'coping processes' refer to any efforts to manage demands, including processes that other might label as defences.

In their meta-analysis of 43 studies concerning the comparative short-term and long-term efficacy of avoidant and attentional coping strategies, Suls and Fletcher (1985) define *avoidant strategies* as any form of denial, repression, suppression, and distraction from the distressing event, and state that the common factor to these coping strategies is that they all divert focused attention away from the somatic and/or psychological distress being experienced. In contrast, *attention strategies* consist of focusing attention upon the psychological and/or somatic distress being experienced. Attention strategies may further be categorised as either *emotion-focused or sensory-focused attention strategies*, which are defined in terms of processing information in terms of an emotional or (non-emotional) sensory cognitive set.

Carver et al.'s 53-item (1989) Coping Orientations to Problems Experienced (COPE) has been used in previous research concerning coping with HIV-infection (e.g., Goodkin, Blaney et al., 1992; Antoni, August et al., 1990; Blaney et al., 1990). In terms of face validity, the COPE appears to adequately measure all the major constructs described above. A closer examination of the various constructs measured by the COPE subscales and it's internal validity and test-retest reliability, as detailed in the following paragraphs, indicates that the COPE would be an appropriate instrument for measuring coping methods in the present study.

The COPE has a total of 53 items, with 13 subscales of 4 items each, and one additional single-item subscale (refer Appendix 2, Table 2-A). The first COPE subscale, active coping, concerns taking direct behavioural actions to deal with a distressing situation or problem, while the planning subscale concerns thinking about action strategies of how to deal with the problem. Suppression of competing activities involves the detachment from other projects, in order to focus upon dealing with the problem at hand. Restraint coping is both active and passive, in that it concerns waiting until the most opportune time to take action, and not acting prematurely. The fifth subscale, seeking support for instrumental reasons concerns the active seeking of support. It is apparent that these first five subscales may be subsumed under the general heading of problem-focused coping methods.

Emotion-focused coping methods subscales include seeking support for emotional reasons (e.g., eliciting sympathy and understanding from others), focusing on and venting of emotions, in which the person focuses upon the distressing feelings being experienced, and the venting of these feelings, acceptance (i.e., accepting the reality of the situation, indicating willingness to realistically appraise and deal with it), denial (i.e., a primary appraisal which denies the reality of the situation), and positive reinterpretation and growth, which involves focusing upon dealing with distressing feelings, as opposed to dealing with the situation itself. Turning to religion in times of distress is also considered an emotion-focused coping strategy. The COPE also includes subscales measuring behavioural disengagement and mental disengagement. The former concerns reducing efforts to deal with a problem, or even giving up the attempt, while the latter concerns distracting one's thinking away from the problem. There is also a single-item subscale concerning drug-alcohol disengagement.

The various subscales are, with few exceptions, not strongly inter-correlated (refer Appendix 2, Table 2-B). Therefore, the effects of different subscales may be examined separately, which would be advantageous in an investigation seeking to distinguish between the effects of different coping methods. Furthermore, the subscales do tend to correlate in theoretically meaningful ways, although such correlations are not very strong. These groupings include seemingly adaptive methods (i.e., the problem-focused subscales, as well as the seeking of emotional support, positive reinterpretation, and acceptance subscales), and maladaptive methods (i.e., denial, behavioural, mental and alcohol-drug disengagement, and focus on venting emotions). The apparently maladaptive methods are also negatively correlated with the adaptive methods.

Convergent and discriminant validity for each subscale with a variety of constructs have also been established (Carver et al., 1989; refer Appendix 2, Table 2-C for an adapted table of results). These constructs include optimism, control, self-esteem, internal locus of control, personality hardiness, Type A personality, monitoring (seeking information) and blunting (distraction from distressing stimuli), social desirability, and trait-anxiety.

It is interesting to note that, with few exceptions, the adaptive subscales are positively correlated with optimism, while the maladaptive subscales are uniformly negatively correlated with optimism. Similar trends are evident for control, self-esteem, hardiness, Type A personality, and internality of locus of control, although the trend is not uniform. The reverse seems to be true for trait anxiety.

Monitoring and blunting do not discriminate between adaptive and maladaptive coping methods, with blunting unrelated to all subscales, and monitoring correlating positively with a few adaptive and maladaptive subscales. Of the adaptive subscales, only the positive reinterpretation and growth subscale is positively correlated with social desirability, indicating that these subscales are largely free of demand expectancies.

With the exception of denial and mental disengagement, the maladaptive subscales are negatively correlated with social desirability, which adds to the perception of these subscales as reflecting socially maladaptive constructs.

The absence of association with social desirability for the denial and mental disengagement subscales suggests that these coping strategies are not necessarily maladaptive in the same sense as the other maladaptive subscales, and that the former coping methods may only be viewed as maladaptive in specific circumstances, while the others appear to be more maladaptive in terms of social interactions.

The labelling of certain coping methods as socially 'maladaptive' needs to be treated with caution. Styles of coping considered to indicate 'good coping', i.e., positive attitudes, 'good adjustment' and co-operation, are positively correlated with negative health outcomes in seriously ill cancer patients, while coping styles considered indicators of maladjustment (e.g., negative affect, hostility) are negatively correlated with worst outcomes (Levy, 1988).

Numerous reports of those who have survived terminal illnesses (e.g., Simonton, Matthews-Simonton & Creighton, 1978; Siegel, 1986; Le Shan, 1989; Proto, 1990) indicate that those who forego certain social conventions, such as refusing to passively accept prescribed treatment regimes and insisting on being consulted in treatment decisions, are more likely to survive. These people are commonly viewed as "difficult" patients, indicating that expected social conventions are not being adhered to in such behaviour.

In this regard, it is interesting to note that, although the various COPE scales distinguish between avoidant and nonavoidant (attentional) strategies, they do not provide a clear separation between the emotional and nonemotional (sensory) cognitive processing attentional subsets. For example, the subscale of focusing on and venting of emotions contains items reflecting both types of attentional coping, such as simply being aware of feelings versus the expression of emotions. Similarly, the COPE subscale of positive reinterpretation and growth contains items reflecting both avoidant mental strategies, such as 'trying to see the positive aspect of every event', as well as more reality-oriented attentional strategies, such as recognising the reality of situations and learning from them. Thus, although these COPE subscales seem to provide a homogenous measure of specific constructs, this apparent homogeniety may conceal important differences in coping strategies.

Therefore, with respect to construct validity, the COPE appears to adequately measure the diverse constructs referred to by Billings and Moos (1980). However, certain other theoretical coping constructs are not directly reflected in the various subscales, and can only be ascertained and measured by examining specific items. Internal consistency amongst items per subscale and test-retest reliabilities over 8 weeks (refer Appendix 2, Table 2-D) also appear to be good, when it is considered that subscales have only 4 items each.

A comparison between the normative means and standard deviations obtained for a college student sample (Carver *et al.*, 1989; refer Appendix 2, Table 2-D) and samples of HIV-seronegative and seropositive homosexual males (Blaney *et al.*, 1990; refer Appendix 2, Table 2-D) reveals that the HIV sample means differ very little from Carver *et al.*'s large validation sample.

The standard deviations in these two samples indicate comparable variability as well, suggesting that homosexual males, whether HIV-seropositive or seronegative, do not differ from the norm in terms of general coping methods. Of particular interest is that HIV-seropositive homosexual males do not differ in coping methods from seronegative homosexual males (Blaney *et al.*, 1990).

The original set of instructions and rating scales have been slightly modified for the purposes of the present study, in order to improve clarity. A comparison between the original instructions (refer Appendix 2, Table 2-E) and the modified version (refer Appendix 2, Table 2-F) reveals that the meaning of both sets of instructions and response options are largely the same. Whereas the modified instructions are shorter and more succinct, the original response format is elaborated upon, with paraphrased options added to the original format. The purpose of the latter is to provide greater continuity with scales which follow, which have slightly different response formats.

As the article by Carver et al. (1989) which contains the 53 items (refer Appendix 2, Table 2-A) does not present the test-sequence of the items, the 53 items were numbered to form a sampling framework, and then assigned randomised positions. The sequence of items was then visually checked to eliminate proximity of items from the same subscale, to produce the final scale used in this study and the scoring procedures for each subscale (refer Appendix 2, Table 2-G; Appendix 1, ii-iii).

As several constructs previously associated with immune function appear to be measured directly or indirectly by the COPE, additional measurement of these variables would appear redundant. Specifically, the COPE has two subscales for social support, and sensitizer-repressor interpersonal styles can be inferred from the 'focus on and venting of emotions' subscale, as detailed in section 4.5.2.

However, the COPE does not measure loneliness, nor does it directly measure optimism-hopelessness, although most subscales correlate significantly with the latter. Furthermore, measurements of emotional states, such as depression, have also been indicated as being necessary. Therefore, separate measuring instruments for these factors are required.

#### 4.6.2 REPRESSOR-SENSITIZER INTERPERSONAL STYLES

A number of studies (e.g., Esterling et al., 1990; Solano et al., 1993) have indicated that repressor and sensitizer interpersonal styles have significant effects upon immune functioning in viral infections. According to Cohen (1987), the concept of the repression-sensitization dichotomy has emerged from literature of perceptual defence mechanisms, where it was found that some people defend against threatening stimuli, such as having comparatively slower reactions to emotional words than neutral words, while others appear to demonstrate the opposite, namely faster reactions to emotional words than neutral words (i.e., perceptual vigilance). Cohen (1987) further states that the repression-sensitization dichotomy can be viewed as a basic distinction between avoidant and approach/vigilant psychodynamic defence processes.

According to the Esterling *et al.* (1990), repression-sensitization can be defined as follows:

Subjects with elevations on Repressive interpersonal styles ostensibly have an inner need to deny negative feelings to themselves and others, tend to appear content in the face of problems, and may attempt to please others with self-sacrificing behaviours. Individuals with elevations in Sensitizer styles ostensibly come across to others as being overbearing, aggressive, rivalrous, and confident, tend to have a low level of frustration tolerance, and are quick to express their negative feelings. (Esteriing et al., 1990, p.400).

In their study, Esterling et al. (1990) found that the number of emotional words used in written essay describing a distressing past event was significantly different for three groups, namely repressor, sensitizer, and neither style. Sensitizers used comparatively high numbers of emotional words, while repressors used few such words, and the neither-style group was intermediate in emotional word usage.

However, Weinberger et al. (1979) point out that there are two possible reasons for reporting low levels of emotional distress, namely that some people are simply not experiencing high levels of distress (i.e., true low-anxiety subjects), while others are indeed experiencing high levels of distress but are repressing such anxious feelings (true repressors). They therefore state that measurements of repressor styles need to consider aspects such as social desirability and anxiety levels, with true repressors scoring high in measures of social desirability and low on anxiety, and with true low-anxiety subjects scoring low on both anxiety and social desirability.

In subsequent investigations conducted by Weinberger et al. (1979), this distinction between true repressors and low-anxious people proved to produce significant differences in performance tasks and physiological reactions to distressing stimuli, with true repressors' task performances being slower and less accurate, and such people also had higher physiological responses to emotionally evocative stimuli.

Given the previous definitions of the construct, repression-sensitization styles could be adequately measured with the COPE's *focus on and venting of emotions* subscale. This subscale is significantly positively correlated with anxiety and Type A personality, as well as significantly negatively correlated with control and social desirability (refer Appendix 2, Table 2-C). Thus, low scorers in a median-split or upper-lower quartile comparison on this subscale would be low in anxiety, high in control, and high in social desirability, indicating repressor style. High scorers would conversely be low in control, high in Type A personality traits, and high in anxiety, indicating a sensitizer style in dealing with distressing events.

Furthermore, as this subscale measures the extent to which a person expresses emotion when distressed, it appears to have adequate construct validity in terms of measuring a central aspect of the repressor-sensitizer construct, namely repression or expression of emotions.

#### 4.6.3 LONELINESS

All reviewed studies which measured loneliness utilised the Revised UCLA Loneliness Scale (Russell *et al.*, 1980), either in it's 20-item long form or it's 4-item survey form. As the latter contains an optimal subset of items predicting self-reported loneliness, this short form will be utilised in the current study (refer Appendix 2, Table 2-I), and this would simultaneously reduce the item burden on respondents.

#### Loneliness has been defined as:

The unpleasant experience that occurs when a person's network of social relations is deficient in some important way, either quantitatively or qualitatively (Perlman & Peplau, 1981, p.31, in Peplau & Perlman, 1982, p.4).

An affective state in which the individual is aware of the feeling of being apart from others, along with the experience of a vague need for other individuals (Leiderman, 1980, p.387, in Peplau & Perlman, 1982, p.4).

Loneliness can be distinguished from being alone in that the latter refers only to the quantitative or objective absence of others, while loneliness refers to subjective perceptions and feelings of relational deficit related to desired social contacts, regardless of the objective presence or absence of people.

In terms of coping with life changes, chronic loneliness has several implications. According to Peplau and Perlman (1982), lonely people tend to be more self-focused, may ask fewer questions of others, and may be more depressed if the cause of loneliness is attributed to stable personal characteristics. Lonely people also have different self-disclosure patterns from other people, such as either sporadic excessive self-disclosure or very little self-disclosure, and they tend to be less assertive in social encounters. Loneliness is correlated with anxiety and depression, and has been found to be significantly correlated with hopelessness and being self-enclosed.

Regarding the 4-item survey form of the scale, Russell (1982) reports an internal consistency coefficient alpha of 0.75. Normative means and standard deviations (refer Appendix 2, Table 2-J) were obtained in a telephone survey of adults, and show that loneliness is negatively correlated with age.

There is little published information regarding convergent or discriminant validity and test-retest reliability analyses of the shortened survey form. information regarding these aspects can be indirectly obtained from studies which examined these aspects with the longer 20-item version of the scale, which contains all 4 of the survey items. Pertaining to construct validity, a previously mentioned study by Longo et al. (1988), which utilised the 20-item form of the scale in an investigation of people with herpes virus, found that subjects who received social support or psychosocial intervention, were significantly less lonely than waiting list controls. Measurements of loneliness at four different times over a period of 6 months in the waiting-list control group remained quite constant, indicating that the 20-item form has adequate testretest reliability over this period of time, and that the construct being measured concerns loneliness experienced over a relatively long period of time. suggests that the 20-item UCLA Loneliness Scale appears to be measuring a stable (chronic) form of loneliness. As it has been previously indicated, the 4item survey scale is an optimal subset of items from the 20-item version, and it can be reasonably assumed that the survey scale likewise measures chronic loneliness.

According to Russell (1982), the 20-item scale also has a high internal consistency coefficient alpha (0.94), a high correlation (0.79) with self-reported loneliness (r = 0.71), and a high correlations with other loneliness measures.

Convergence validity with depression (r = 0.62), dissatisfaction, shyness, unhappiness and anxiety has been demonstrated, as well as discriminant validity with unrelated constructs, such as being hard-working or having wide interests. Test-retest reliabilities of r = 0.73 over 2 months, and r = 0.62 over 7 months have also been found (Russell, 1982).

Subsequent discriminant analyses demonstrated that the 20-item loneliness scale was significantly correlated with greater amounts of time spent alone, lower numbers of friends, and low affiliative and low social risk behaviour, even after statistically controlling for the effects of mood and personality variables (Russell, 1982). This finding, in conjunction with the previously mentioned validity studies, indicates that the loneliness construct measured by the UCLA Loneliness scale is conceptually distinct from other mood and personality constructs, and is related to affiliative motivation and social risk behaviour. In summary, the construct measured by the UCLA Loneliness Scale appears to be measuring chronic loneliness, which is distinct from other constructs.

The instructions used in the present study incorporated a modified version of the original instructions (refer Appendix 2, Table 2-I, Table 2-K). The original instructions are largely retained, despite alterations to the sequence of the wording, which was necessary in order to incorporate a clause referring to the experience of such feelings over the previous year. The latter was included in order to emphasise the dispositional aspect of the measurements, to produce the final version of the scale (refer Appendix 1, iii, items 55, 57, 58 & 60).

The original response format has also been slightly modified in order to provide format discontinuity with the main instrument, the COPE. Although both instruments use response formats ranging from "1" to "4", the corresponding meanings of each number are slightly different. In particular, the number "3" corresponds to "Often" in the COPE, while the UCLA Loneliness Scale response number "4" also corresponds to "Often". Therefore, it was considered necessary to modify the original UCLA Loneliness format in order to bring this difference to the respondent's attention, and thus reduce response-set problems. The alterations are not expected to meaningfully alter response choices, as the original range of options is unlikely to be understood differently from an "Almost Never - Often" continuum.

#### 4.6.4 SINGLE ITEMS

For reasons previously elaborated upon concerning the length of the questionnaire, several scales were excised from the pilot questionnaire, including Levenstein *et al.*'s (1993) Perceived Stress Questionnaire (PSQ) and Scheier and Carver's (1985) Life Orientation Test, the latter being a measure of dispositional optimism. However, it was considered necessary to retain some measure of future-related hopelessness, as this factor has been previously indicated as being a potentially significant factor in associations with immune functioning (e.g., Perry *et al.*, 1992).

Furthermore, some indication of present-time dysphoria would be useful in forming descriptive profiles of people at different stages of HIV infection. Therefore, four of the items from the PSQ (items 7, 9, 21, and 22) have been retained (refer Appendix 2, Table 2-L). Factor analyses (Levenstein et al., 1993; refer Appendix 2, Table 2-M) indicate that two of these items (items 7 and 21) load on the factor "lack of joy", and the other two items (items 9 and 22) load on the factor "worries". The former two items were selected from other similar items on the basis of their face validity concerning present-time pleasure ("joy") in living, while the latter two items were chosen due to their face validity concerning future-related "worries", as opposed to general "worries".

As PSQ items have been standardised by using a 4-point response format which is similar to that of the UCLA Loneliness Scale, only minimal adjustments would be necessary in order to combine these items with the UCLA Loneliness items into one scale, thus reducing the number of sets of response instructions. Furthermore, as the two items concerning "lack of joy" are positively worded and the items concerning future-related "worries" are negatively phrased, these four items retain the balance between positively and negatively worded items in the UCLA Loneliness Survey. The only modifications to the original PSQ items concerned the changing of the personal pronoun of "You" to "I", in order to conform with the format of both the UCLA Loneliness Scale and the COPE (refer Appendix 2, Table 2-N). It is unlikely that such a change would impact upon the response to the items.

#### (a) HOPELESSNESS

Hopelessness and pessimism are considered to be conceptually similar, as both concern a person's negative expectancies concerning her/himself and his/her future (Beck, Weissman, Lester & Trexler, 1974), while optimism refers to positive expectations of future outcomes (Scheier & Carver, 1985). Therefore, the pessimism-optimism continuum construct will be used to explicate issues of hopelessness in the following discussion.

Pessimism has been identified as a core factor in depression (Beck *et al.*, 1974), it is therefore of particular relevance to the present investigation, as both hopelessness and depression have been previously found to be associated with negative immunological changes. For example, it has been previously mentioned that, in the prospective study by Perry *et al.* (1992) of HIV-infected people, only two individual items (Beck Depression Inventory item 2, Beck *et al.*, 1961; Brief Symptom Inventory item 35, Derogatis & Melisaratos. 1983) were significant predictors of CD4 cell counts 12 months later. Both these items measured pessimism-optimism specifically regarding the future.

#### (b) JOY

In terms of face validity, the two positively worded items appear to measure pleasure ('joy') in general activities. An examination of these two items (refer Appendix 2, Table 2-M) reveals that both items are negatively correlated with trait anxiety, although only one of these correlations attains significance, and both items are significantly correlated with two other measures of perceived stress (Levenstein *et al.*, 1993).

The mentioned associations seem to indicate that the construct being measured concerns low levels of perceived stress and anxiety, which Levenstein *et al.*(1993) have decided to term 'joy'. In terms of the present study, these items would thus indicate general levels of joy in the previous year.

#### 4.6.5 PROFILE OF RECENT MOOD STATES

In order to obtain a descriptive profile of levels of various current mood states, the "one week" version of the Profile of Mood States (POMS; McNair et al., 1971) is used, as this instrument has been used for this purpose in a number of the reviewed HIV-related studies (e.g., Antoni, August et al., 1990; Antoni et al., 1991; Atkinson et al., 1988; Blaney et al., 1990; Esterling et al., 1992; Goodkin, Blaney et al., 1992; LaPerriere et al., 1990; Solomon et al., 1987), as well as other PNI studies reviewed (e.g., Glaser et al., 1992; Kemeny, Cohen, et al., 1989; Moss et al., 1989).

The POMS consists of 65 adjectives describing feelings experienced in the previous week, each of which is rated on a 5-point scale (refer Appendix 1, iv). There are seven POMS scales, although only the first six are usually utilised. The first six scales are *tension-anxiety* (abbr. 'TA'), defined in terms of somatic tension, *depression-dejection* (abbr. 'DD') indicating depression and feelings of personal inadequacy, *anger-hostility* (abbr. 'AH'), i.e., antipathy to others and anger, *vigour-activity* (abbr. 'VA'), i.e., high energy and feeling vigorous, *fatigue-inertia* (abbr. 'FI'), indicating low energy levels and weariness, and *confusion-bewilderment* (abbr. 'CB'), which indicates cognitive inefficiency and bewilderment. A total mood disturbance score is obtained by totalling the scores of these six scales, with vigour-activity scored negatively.

Although inter-scale correlations between several scales are rather high (refer Appendix 3, Table 3-A), principal components analyses show that the POMS contains six distinct factors with latent roots greater than 1.00 (McNair *et al.*, 1971). Therefore, a reduction in the number of factors would be inadvisable.

Internal consistency per scale is high (refer Appendix 3, Table 3-B), although test-retest reliability predictably decreases over time (refer Appendix 3, Table 3-C), which indicates that the POMS does not appear to measure stable trait-like qualities, but rather that it measures short-term mood states which change over time. Therefore, the POMS would be a useful indicator of mood profiles for groups at different stages of infection, such as pre- and post-symptom development.

Construct, predictive, and concurrent validity studies (McNair *et al.*, 1971, pp. 10-15) have been conducted, which support the face validity of the individual items and scales. Of interest regarding social desirability, the angerhostility scale was found to be significantly associated with social desirability (r = -0.52), while the other scales appear to be comparatively independent of this measure (refer Appendix 3, Table 3-D). Scoring procedures have been standardised (refer Appendix 3, Table 3-E), and norms for various groups, including college students, have also been established (refer Appendix 3, Table 3-F). No alterations to test instructions have been made for the present study.

#### 4.7 DEMOGRAPHIC DESCRIPTION OF RESPONDENTS

In order to obtain a general profile of the study respondents (refer Appendix 1, v), they are asked to indicate their present age, gender, sexual preference, home language, income per month, and highest educational level attained. These details would be necessary for descriptive and comparative norm purposes. Respondents are also asked to indicate residential proximity to agencies providing services to those infected with HIV, as well as whether they have their own means of transport.

Finally, current HIV-status is requested, as well as the date of the first HIV test. The latter HIV-related items were included in order to allow for possible application of the instrument to HIV-negative respondents who would serve as a comparative norm group, as well as to prevent respondents being identified as being HIV-positive by virtue of receiving the questionnaire or having it in their possession. Thus, casual observation of the questionnaire reveals that all people who have been tested for HIV, whether the result was negative or positive, are being approached to participate.

#### 4.8 HIV-RELATED QUESTIONS

There are two temporal aspects of HIV infection investigated in the present study. The first concerns the time of initial infection, and the second relates to the commencement of AIDS-related symptoms, thus indicating the end of the asymptomatic period of infection.

It is the former aspect, namely determining the date or period of infection, which requires careful methodological consideration, as there are few existing standard procedures for retrospectively ascertaining this information apart from consulting medical records if seroconversion illness was medically recorded. However, due to the often mild symptoms associated with seroconversion illness, not many of those infected seek medical treatment at the time, thus limiting the usefulness of such methods for determining the period of infection.

#### 4.8.1 PERIOD OF INFECTION

In this part of the questionnaire (refer Appendix 1, v), respondents are asked to reflect upon the most likely date or period of time in which they became infected with HIV. In order to ensure that respondents are aware of the typical modes of infection, these are briefly described. Furthermore, in order to maximise accuracy (and thus reduce error variance), respondents are requested to indicate either the date or period of time in which they think (or know) that they were infected. Thus, they can indicate a specific date or a period of months - even years - in which infection was most likely to have occurred, without any undue obligation of specificity which might either produce methodologically-induced non-response or random guessing artefacts.

The reported period of infection can then be corroborated by means of medical records if possible, as well as information obtained during post-test counselling. It is considered likely that many HIV-infected people who were notified of seropositive status at a recognised AIDS-related service agency would have a fairly accurate idea of the period of infection, due to standard partner notification and tracing procedures applied by such agencies.

In order to reflect the perceived certainty of the indicated date or period of time, respondents are asked to indicate how certain they feel about the date or period that they provide, on a 7-point Likert-type scale ranging from "very certain" to "very uncertain". It is considered reasonable to assume that those who have determined the path of infection would reflect a high degree of certainty regarding the date they provide, while those who have little idea of the path of infection would indicate very little certainty regarding the date provided. It is of course possible that high uncertainty could be the product of lack of knowledge concerning the nature of risks for infection.

In order to reduce error due to memory-loss and uncertainty due to absence of knowing the date of infection, all responses regarding the time of infection would need to be scrutinised and verified. Particularly those reported periods of infection which indicate very low certainty, a likely period of infection spanning a considerable period of time, or which indicate not knowing when infection occurred, would be excluded from analyses concerning the duration of infection, unless additional confirming data (eg., medical records and personal history files compiled for the purposes of partner-notification) are obtained which increases the level of certainty or more accurately pinpoints the time of infection. If a substantial proportion of respondents indicate that they are fairly certain about the time of infection, and subsequent investigation supports the accuracy of such self-reports, this would add a valuable methodological tool for investigations of long-term HIV infection.

There is, of course, the possibility that these estimates are biased or inaccurate for various reasons, including memory deficits and the passage of time. However, a sample which includes a wide range of such estimated periods of infection would serve to reduce the effects of individual inaccuracies. It would also be important to consider neurological deficits found in some people with AIDS, as described in the following section, and to exclude such possible confounding factors. Finally, the obtained variation and median length of the asymptomatic period could be statistically compared to the general norms already established (e.g., Lang *et al.*, 1989; McCutchan, 1990). In this way, the overall accuracy of reported dates could be fairly accurately assessed.

#### 4.8.2 SYMPTOM STATUS

The present study will utilise the 1987 (Revised) Centers for Disease Control (CDC) classification system (in Kirkwood & Lewis, 1989, refer Appendix 4), as this would provide methodological continuity with previous PNI research in this area (e.g., Polk *et al.*, 1987; Atkinson *et al.*, 1988; Ostrow *et al.*, 1989; Rabkin *et al.*, 1991; Perry *et al.*, 1992; Goodkin, Blaney *et al.*, 1992; Solano *et al.*, 1993).

From Appendix 4 (page i), it is apparent that previous classification systems distinguished between the seroconversion illness, the asymptomatic stage ('healthy carriers'), AIDS-related Complex (ARC), and AIDS. The revised system (refer Appendix 4, i) elaborates upon this classification symptom, and describes a series of stages of illness, starting from (seroconversion) acute infection (Group I), asymptomatic infection (Group II), persistent generalised lymphadenopathy (PGL, Group III), and finally, five sub-categories of AIDS (Group IV, subgroups A to E).

Group IV Subgroup A concerns constitutional disease, such as persistent fever, rapid weight loss, chronic diarrhoea, while Group IV Subgroup C concerns secondary infections, such as serious protozoal infections (e.g., pneumocystis carinii pneumonia, toxoplasmosis, cryptococcosis, and isosporiasis), serious viral infections (e.g., cytomegalovirus) and serious fungal infections (e.g., mycobacterium).

Group IV Subgroup D concerns secondary cancers such as Kaposi's sarcoma or other lymphomas. Subgroup B refers specifically to neurological disease, while Subgroup E refers to other (atypical) symptomology related to HIV infection. Appendix 4 contains a more explicit and detailed exposition of the previous classification system.

Although a comprehensive checklist of symptoms would not be feasible, the most more frequently encountered symptoms at the various post-asymptomatic stages of infection are included in the questionnaire (refer Appendix 1, vi). Subjects are asked to indicate whether they have experienced each symptom, and in order to distinguish some of these symptoms from seroconversion symptoms, they are requested to indicate only those symptoms which have persisted for at least two weeks. They are asked to indicate when each symptom first manifested, and whether they are currently using (or have used in the past) any antiretroviral medication (e.g. AZT). They are asked to indicate the name of the medication, as well as the period of usage.

Study participants will be symptomatically grouped according to the guidelines contained in Appendix 4. In this regard, it has been previously noted that some people overcome symptoms and become asymptomatic again. However, this is not the norm, and the use of the previous-described procedure would provide a fairly accurate estimate of general disease progression. It should also be noted that the described self-report checklist and classification system has been used successfully by other researchers (e.g., Perry et al., 1992), who found that by weighting symptoms according to severity, this measurement is significantly correlated (p < 0.05) with actual CD4 cell counts in a series of measurements over a year. However, as the present study is not primarily concerned with the severity of symptoms, but rather with their presence or absence, such weighting would be methodologically unnecessary.

After receipt of the completed questionnaire, those sections pertaining to demographic details and self-reported symptomology will be returned to ASET, who will then verify the reported information by comparing it with the medical history of each respondent, and the disease classification of each person will thus be ascertained.

In addition, the reported period of infection will be compared with prior information known regarding each respondent, in order to determine whether low levels of certainty regarding the time of infection are warranted, and whether more accurate estimates of the time of infection can be reasonably determined in those cases which report a wide range of the possible period of infection.

This verification procedure is made possible due to the fact that the questionnaires will be individually distributed by ASET to small groups per week, and it is thus possible for them to identify each subject by virtue of their age and other information reported in the questionnaire. Naturally, at no time is the identity of the subject revealed to the researcher during this procedure. Any additional information pertaining to symptom status, immune measurements, and time of infection will be written on the questionnaire, as well as the known medical classification, and then returned to the researcher.

#### 4.8.3 IMMUNE MEASURES: CD4 AND CD8-CELL COUNTS

Each person's most recent CD4 and CD8-cell counts are obtainable from the medical records pertaining to their participation in the drug trial. The following procedure is followed regarding obtaining immunophenotyping (Dr Pat Bouic, personal communication, 15 April 1994):

#### Reagents:

Monoclonal antibodies used in this study were purchased from Becton Dickinson (Bactlab Systems) and consisted of dual labelled monoclonals for the different subsets of T-cells as well as B- and NK-cells. The antibodies used were the following:

CD3-FITC + CD4-RPE

CD3-FTC + CD8-RPE

CD3-FITC + CD19-RPE

CD3-FITC + CD16+56-RPE

Isotypic Controls (IgG1-FITC + IgG2-RPE)

Leucogate (CD45-FITC + CD14-RPE)

All monoclonal antibodies were used at optimal dilutions as indicated by the suppliers.

#### Methods:

Venous peripheral blood was collected into tubes containing anticoagulants (EDTA) and the blood was processed within 4 hours of
collection. Briefly, 100 microlitre blood was incubated with an aliquot
of monoclonal antibody in the dark. After 20 minutes, 1 ml of lysing
buffer (Becton Dickinson) was added and the tubes were vortexed
and incubated a further 10 minutes in the dark. The tubes were then
centrifuged at 1800 rpm for 5 minutes and the supernatants were
decanted. One ml of PBS (phosphate buffered saline) was added to
each tube and the centrifugation was repeated. After the final wash;
500 microlitre fixative (PBS containing 1% Formaldehyde) was added
to the cells. Analysis of the cells was conducted on a flow cytometer
(FACScan, Becton Dickinson) using the software Simulset. The flow
cytometer was calibrated using Calibrite beads and the automatic
compensation software Autocomp.

The Simulset software sets up a lymphocyte gate using the first tube containing the Leucogate reagents (back-gating of granulocytes and monocytes). The software programme then analyzes only those cells falling in the so-called lymphocyte gate. The results were expressed as percentage of lymphocytes positive for the marker in question and the absolute number of cells expressing CD3 CD4 or CD3 CD8 were calculated from the routine haematology full blood count and differential counts conducted on an aliquot of blood in parallel.

#### 4.9 SURVEY OF NEEDS

In the introduction to this section of the questionnaire, respondents are informed that the purpose of this section concerns ascertaining the emotional and practical needs of those infected with HIV (refer Appendix 1, vii). They are also told that it is often difficult to ascertain these needs, due largely to the secrecy regarding HIV-seropositive status.

The first question (refer Appendix 1, vii, question a-i and a-ii) concerns the use of services (e.g., counselling or coping skills programs) in the past, and whether these services suited the respondents needs. In order to determine whether such services were obtained from recognised agencies such as Aids Support and Education Trust (ASET) or Aids Training and Information Centre (ATIC), or therapists in private practice, respondents are requested to indicate the source of the received counselling or program. They are further asked to state their reasons for utilising these services.

The next three items (refer Appendix 1, vii, questions b, c, and d) concern services needed. The first concerns emotional needs and counselling for anxieties experienced, while the second and third items concern practical (problem-oriented) needs, such as needs for specific information (question c), and advice regarding medical, legal or financial assistance (question d).

Question (e) and (f) relate directly to coping skill programs. The first question (refer Appendix 1, viii, question e-i, e-ii, and e-iii) concerning the respondents awareness of different methods of coping with HIV infection, such as relaxation techniques, visualization, and healthy diets, and whether the respondent also would like additional assistance and information regarding such methods. While the previous two items are fixed-response, the third is open ended, and seeks to elicit the respondents interest in obtaining more information about specific coping skills.

In the following two open-ended items (refer Appendix 1, viii, question f-i and f-ii), the respondent's reservations regarding organised coping programs are probed, as well as perceived needs in terms of the type of program that the respondent feels will be of benefit. With respect to the latter aspect, the respondent is prompted with two examples, namely emotion-focused and problem-focused programs. This item also seeks to ascertain additional issues, such as reasons for the reported reluctance of those who develop symptoms to continue programs designed to maintain health in HIV-seropositive people, such as Body Positive.

In order to provide group services to HIV-spectrum people, it would thus be of particular importance to establish the nature of reservations and objections regarding such programs, as well as to determine possible differences in needs for such programs at different stages of the illness. The final item (refer Appendix 1, viii, question g) is an open-ended question in which the respondent is given the opportunity to state any other need or issue not previously covered.

At the end of this section, the respondent is informed that many of the mentioned services are indeed available, and that they could obtain more information regarding the agency providing such services from Life Line, which has a resource listing for such purposes.

Finally, the respondent is thanked for the information provided, and instructed to return the completed questionnaire in the addressed and stamped envelope provided. The respondent is also informed of the researcher's name and affiliation, which is stated in the introductory cover letter.

#### 4.10 SAMPLE SIZE & STATISTICAL POWER

In order to ascertain the necessary sample sizes for obtaining statistically meaningful results in between-groups comparisons with the two main instruments utilised, namely the Profile of Mood States (McNair et al., 1971) and the Coping Orientations to Problems Experienced (Carver et al., 1989), an analysis of effect sizes and power was conducted by examining previous research which used these instruments in HIV-related studies.

The purpose of this exercise is to comply with another of Cook and Campbell's (1976) criteria for strengthening assumptions of causality, namely tests of adequate statistical power. Thus, Type I and Type II errors would need to be kept within acceptable limits, which requires ascertaining adequate sample sizes for testing the expected effects of the various psychosocial factors.

## 4.10.1 COPING ORIENTATIONS TO PROBLEMS EXPERIENCED (COPE)

Two studies are examined: Goodkin, Blaney et al. (1992), and Blaney et al. (1990). Neither study included (CDC Group III and IV) ARC subjects, but focused upon HIV-seropositive asymptomatic subjects (CDC Group II). Blaney et al. (1990) included a control group of 13 HIV-seronegative subjects. All the subjects in both studies were homosexual males. As the study by Goodkin, Blaney et al. (1992) combined related subscales, the analysis of effect sizes likewise groups the fourteen subscales together into four main factors, namely Active Coping ('Active'), Denial and Disengagement ('Deny'), Focus on and Venting of Emotions ('Vent'), and Turning to Religion ('Relig').

The college students norms (N = 1030) for these four factors (Carver *et al.*, 1989), as well as the statistics for HIV-seronegative homosexual males (N = 13) obtained by Blaney *et al.* (1990), are presented in Table 1.

TABLE 1
EFFECT SIZES FOR THE COPE:

## COLLEGE STUDENTS VERSUS SERONEGATIVE HOMOSEXUAL MALES

	College Students	HIV-seroneg Homosexual Males	Effect Size	
	N = 1030	N = 13		
	Mean StDev	Mean StDev	(d) %Non- Overlap	
Active	91.4 7.6	94.2 6.6	0.4 27.4 %	
Deny	21.9 4.1	18.2 3.3	0.9 51.6 %	
Vent	10.1 3.1	10.3 2.8	0.1 7.7 %	
Relig	8.8 4.1	7.9 4.7	0.2 14.7 %	

The Denial factor appears to have a large effect size, indicating that homosexual males have a lower mean level of denial. However, the small sample size of the homosexual males suggests caution regarding interpreting this large effect. None of the other factors attain moderate effect sizes.

However, a cursory overview of the standard deviations indicates that the two groups do not have significantly different variances, suggesting that they were drawn from the same population. It is therefore possible that being a homosexual male may have a distinct effect upon Denial scores, but the value of basing further comparisons on such values is uncertain due to the very small sample of homosexual males. Therefore, effect sizes for being HIV-seropositive is hereafter calculated utilising only the college male norms.

In order to determine the size of COPE subscale effects between seronegative and seropositive males, the college male norms are compared with the weighted means and pooled standard deviations of the four factors in a group (N = 106) of HIV-seropositive asymptomatic subjects (refer Table 2). The latter was obtained by combining data from Goodkin, Blaney *et al.* (1992), with N = 62, and Blaney *et al.*(1990), with N = 44. It is notable that both studies included CDC Group II and Group III (lymphadenopathy) in their groups of asymptomatic subjects.

TABLE 2

#### **EFFECT SIZES FOR THE COPE:**

## SERONEGATIVE VERSUS ASYMPTOMATIC HIV-SEROPOSITIVE MALES

	College Students N = 1030	HIV-pos. Homosexuai Males N = 106	Effect Size	
	Mean StDev	Wght. Pool. Mean StDev	(d) %Non-Overlap	
Active Deny Vent Relig	91.4 7.6 21.9 4.1 10.1 3.1 8.8 4.1	98.2 10.8 22.3 4.8 10.2 2.5 9.1 3.9	0.9 51.6 % 0.1 7.7 % 0.0 0.0 % 0.1 7.7 %	

Only the active composite coping factor produced an effect size exceeding moderate size. This effect size of 0.9 is thus used to find the required sample sizes for yielding significant differences. From the power tables in Cohen (1969, p.28-29) for 1-tailed tests, a sample size of approximately 17 subjects would be required to provide a statistically significant difference for such an effect at the probability of Type I error (alpha) set at 5% and probability for Type II error (beta) at 20%, i.e., for power set at 0.80. For alpha still at 5%, but for beta reduced to 5% (i.e., power set at 0.95), a sample size of approximately 28 subjects would be required.

#### 4.10.2 PROFILE OF MOOD STATES (POMS)

Two studies are examined: Blaney et al. (1990), and Atkinson et al. (1988). Blaney et al. (1990) included homosexual males who were either HIV-seronegative or HIV-seropositive (asymptomatic), while Atkinson et al. (1988) additionally included those who had AIDS-Related Complex (ARC), and subjects with AIDS. Atkinson et al. (1988) does not indicate whether those with AIDS were screened for neuropsychiatric complications associated with HIV infection, and this data is thus not examined.

The six subscales of the POMS are analysed individually, and the following abbreviations are hereafter utilised to indicate each subscale: TA (Tension-Anxiety), DD (Depression-Dejection), AH (Anger-Hostility), VA (Vigour-Activity), FI (Fatigue-Inertia), and CB (Confusion-Bewilderment). The mean and standard deviation norms per subscale for male college students (N = 340) provided by McNair *et al.* (1971; refer Appendix 3, Table 3-F) and the weighted mean and pooled standard deviation for HIV-seronegative homosexual males in Blaney *et al.* (N = 13) and Atkinson *et al.* (N = 11) are tabulated below. Based upon the assumption of homogeneity of variance for HIV-seronegative homosexual males, pooled standard deviations for unequal samples sizes were calculated by means of the procedure outlined by Howell (1989, p.192).

As the proposed sample is exclusively homosexual males, it was considered prudent to ascertain whether the male college student norms provided by McNair et al. (1971) are appropriate as a normative basis for further statistical analyses. The effect size index (d), and percentage of non-overlap of the two samples, as defined by Cohen (1969, p.18-20) was calculated (refer Table 3), using the college student standard deviation as the basis of comparison, assuming that homosexual males and male college students are drawn from the same population with equal variance. A cursory comparison of the two groups' standard deviations tends to support this latter assumption.

TABLE 3

POMS: COLLEGE MALES

VERSUS HOMOSEXUAL MALES

	Male College Students N = 340	HIV-seronegative Homosexual Males N = 24	Effect Size
	Mean StDev	Mean StDev(p)	(d) %Non-overlap
TA DD AH VA FI CB	12.9 6.8 13.1 10.5 10.1 7.8 15.6 6.0 10.4 6.2 10.2 5.2	9.8 6.2 10.5 10.7 9.1 7.9 19.0 6.2 6.8 5.6 6.2 4.8	0.5 33.0 % 0.3 21.3 % 0.1 7.7 % 0.6 38.2 % 0.6 38.2 % 0.8 47.4 %

According to the outlines for interpreting effect sizes (Cohen, 1969, p.38), two subscales produced small effect sizes (DD and AH), three subscales produced medium effect sizes (TA, VA, and FI). The Confusion-Bewilderment (CB) subscale produced a large effect size. The percentages of non-overlap of the two groups on certain subscales suggests that membership of the category 'homosexual' may have some differential effect upon scores obtained in some of the POMS subscales. Thus, the HIV-seronegative homosexual male weighted means and pooled standard deviations are hereafter utilised as the basis for effect-size calculations with respect to HIV-seropositive and ARC subjects.

The pooled standard deviations and weighted means for HIV-seropositive asymptomatic (CDC Groups II and III) subjects (N = 45) in the study by Blaney *et al.* (1930) and those in the study by Atkinson *et al.* (1938) (N = 17), along with the effect sizes (d) and percentage of overlap per subscale, are tabulated in Table 4.

As the standard deviations of the two groups do not differ substantially, it was considered appropriate to assume that the two groups are sampled from the same population, thus enabling the use of the homosexual male normative standard deviation as the basis for calculating the effect size. Although the sample sizes are unequal, this does not affect the validity of the calculated effect sizes and percentages of non-overlap (Cohen, 1969, p.40).

**TABLE 4** 

# POMS: EFFECT SIZES FOR SERONEGATIVE VERSUS SEROPOSITIVE (ASYMPTOMATIC) HOMOSEXUAL MALES

	HIV-seroneg. Homosexual Males N = 24	HIV-seropos. Asymptomatic Homosexual Males N = 62	Effect Size
	Mean StDev	Mean StDev(p)	(d) %Non-overlap
TA DD AH VA FI CB	9.8 6.2 10.5 10.7 9.1 7.9 19.0 6.2 6.8 5.6 5.2 6.2	13.2 7.5 11.5 9.6 8.5 6.9 17.1 7.6 7.0 5.3 8.3 5.5	0.6 38.2 % 0.1 7.7 % 0.1 7.7 % 0.3 21.3 % 0.0 0.0 % 0.4 27.4 %

With the exception of the Tension-Anxiety (TA) subscale, there appears to be very weak to small effect sizes for the various subscales. In order to obtain an adequate sample size for alpha = 0.05 and to attain power of 0.80 in a one-tailed test of significance, a sample size of 35 subjects per group would be required for a moderate effect size of 0.60. Effect sizes smaller than 0.50 will not be considered for further analysis, as 50+ subjects per group would be required, which is not practically feasible in the current study. Therefore, it would be more useful to ascertain samples sizes necessary to attain statistical power for differences between seronegative and symptomatic subjects (refer Table 5), as well as between asymptomatic and symptomatic subjects (refer Table 6).

In this regard, the study by Blaney *et al.* (1990) does not include subjects with ARC, and the following analysis is based upon statistics provided by Atkinson *et al.* (1988), who included 13 subjects with ARC. As with the previous analysis, the seronegative homosexual male norms are used as a basis for comparison.

TABLE 5

## POMS: EFFECT SIZES FOR SERONEGATIVE VERSUS SYMPTOMATIC HOMOSEXUAL MALES

	HIV-seroneg. Homosexual Males N = 24		HIV-seropos. ARC Homosexual Males N = 13		Effect Size	
	Mean	StDev	Mean	StDev	(d) <sup>s</sup>	%Non-Overlap
TA DD AH VA FI CB	10.5 1 9.1 19.0 6.8	6.2 0.7 7.9 6.2 5.6 6.2	16.5 17.6 14.8 11.4 14.7 11.5	8.6 12.7 11.1 6.9 6.9 7.0	1.1 0.7 0.7 1.2 1.4 1.1	58.9 % 43.0 % 43.0 % 62.2 % 68.1 % 58.9 %

It is apparent that there are substantial effect sizes for the construct measured by most of the POMS scales. In order to attain power of 0.80 in 1-tailed comparisons at alpha = 0.05, the necessary sample sizes per group would need to range from approximately 8 (for d = 1.40) and 26 subjects (for effect size of 0.70). For alpha set at 5% and beta at 5% (power of 0.95), samples sizes would need to range from 12 to 44 subjects. In order to obtain a general compromise, a mean effect size was calculated (d = 1.03), which would require a sample size of ranging between 13 subjects (alpha = 5%, beta = 20%, power = 0.80) and 22 subjects (both alpha and beta = 5%, power = 0.95), to produce significant differences in 1-tailed t-tests.

When the HIV-seropositive asymptomatic and ARC subjects are compared (refer Table 6), the effect sizes per subscale are as following, utilising the asymptomatic subject's standard deviation:

POMS: EFFECT SIZES FOR
ASYMPTOMATIC VERSUS SYMPTOMATIC (ARC)

**HOMOSEXUAL MALES** 

TABLE 6

	Asymptomatic Homosexual Males (N = 62)	Symptomatic Homosexual Males (N = 13)	Effect Size	Sample Size Required
	Mean StDev	Mean StDev	(d) %Overlap	N
TA DD AH VA FI CB	13.2 7.5 11.5 9.6 8.5 6.9 17.1 7.6 7.0 5.3 8.3 5.5	6.5 8.6 17.6 12.7 14.8 11.1 11.4 6.9 14.7 6.9 11.5 7.0	0.4 27.4 % 0.6 38.2 % 0.9 51.6 % 0.8 47.4 % 1.5 70.7 % 0.6 38.2 %	78 35 18 20 < 8 35

A mean effect size (d = 0.8) would require a sample size ranging from 20 (alpha = 5% and beta = 20%) to 34 subjects (alpha = 5% and beta = 5%) to provide statistically significant differences in 1-tailed t-tests.

#### 4.10.3 CONCLUSIONS REGARDING SAMPLE SIZE

In order to attain statistical interpretation validity regarding the effects of coping methods, a sample size greater than 20 (probability of Type II error = 20%) or greater than 28 subjects (Type II error probability = 5%) would be required in 1-tailed t-tests, if alpha (probability for Type I error) is set at 5%. For descriptive applications of the POMS, sample size ranging between 13 and 20 subjects per symptom category would seem to be adequate to produce meaningful differences at alpha = 0.05 and power = 0.80, in 1-tailed tests of differences between means. For more stringent tests, sample sizes per category would need to range between 22 and 34 subjects, in order to attain power of 0.95 at alpha of 5% in 1-tailed t-tests. It also appears than consideration needs to be given to possible differences between seronegative homosexual males and college male norms, as there are indications that being a homosexual male produces moderate to large effect sizes in both instruments, when compared to college students. With respect to the POMS, these differences are lower mean scores in Tension-Anxiety, higher Vigour-Activity, lower Fatiguethe COPE, homosexual and lower Confusion-Bewilderment. In Inertia. seronegative appear to have lower mean scores on males Denial/Disengagement factor, compared to college students. However, the small size of the sample of homosexual males suggests that such differences may be spurious, and that, unless plausible reasons for such differences can be posited, the use of college male norms would be more advisable.

#### 4.11 CONCLUSIONS

Several important methodological issues inherent in a study of this nature have been discussed, including the problem of establishing causal direction in cross-sectional associations, as well as issues related to the accuracy of retrospective measurements of times of infection.

With regard to establishing causal directions, it was concluded that, with respect to psychosocial variables assumed to be stable over time, the direction of causality needs to be viewed within the context of existing empirical research which supports the plausibility of specific causal directions, but that prospective studies would be required to substantiate the causal direction of any emergent associations between psychosocial and immunological measurements.

The research design also incorporates methods of cross-validation for selfreported information regarding the time of infection and symptom development, thus reducing problems inherent in retrospective measurements.

In a quasi-experimental study of this nature, it is not possible to eliminate all sources of error, nor is it possible to exert control over all extraneous variables. Furthermore, this study is essentially opportunistic, in that it utilises a sample which is already participating in a clinical drug-trial, which may in itself affect psychosocial measurements such as hopelessness.

Therefore, despite the limitations inherent in the methodology employed, the use of standardised instruments and methods of cross-validation would suggest that the design of the study would provide a fair estimate of the true state of affairs regarding associations between psychosocial variables and immune functioning across the spectrum of HIV-infection, and provide a valuable basis for further investigation.

### **CHAPTER 5**

## STATISTICAL ANALYSIS AND RESULTS

#### 5.1 INTRODUCTION

The previous four chapters have provided the empirical and theoretical basis for the present investigation, culminating in the construction of a research instrument designed to measure a variety of psychosocial and immunological constructs in 27 HIV-infected homosexual men.

In this chapter, specific *a priori* hypotheses and research objectives are presented, based upon a summation of expectations raised in the review of empirical research, taking into consideration the limitations of the research instrument utilized.

Due to the opportunistic nature of the study, certain measurements, such as immune measures, could not be simultaneously measured at the time of the psychosocial measurements. Therefore, prior to the presentation of the results from this study, it is necessary to elucidate the statistical methods which would reduce error variance for these measurements.

Finally, the various hypotheses are statistically tested, and the results of these analyses are presented. These results are discussed in chapter 6.

#### 5.2 HYPOTHESES & RESEARCH OBJECTIVES

As stated in section 4.2 of chapter 4, one of the objectives of this study is to obtain a general psychosocial, demographic and clinical profile of the HIV-infected subjects included in this study. The obtained psychosocial measurements will then be compared to existing norms in order to determine whether this sample is similar to samples utilized in prior research, and whether the obtained sample adequately represents a broad cross-section of HIV-infected homosexual men.

A second methodological area of interest is the stability of measures of coping methods over time, as well as measurements of hopelessness, joy, and loneliness. This will be ascertained by determining the associations of such measurements with the time since infection and age of the subjects. In addition, the stability of such psychosocial constructs in terms of the effects of life changes will be ascertained by analyzing associations with the two measured life-changes, namely unemployment and the presence of symptoms, both of which have been demonstrated to be associated with higher levels of emotional distress (eg., Arnetz et al., 1987; Kessler et al., 1988; Catalan, 1988; Atkinson et al., 1990; Perry et al., 1992; Rabkin et al., 1991; Ostrow et al., 1989).

Unlike coping methods, loneliness, joy and hopelessness, the Profile of Mood States (POMS; McNair et al., 1971) measures recent short-term emotional states. It is expected that levels of emotional distress will be different for the various symptom categories, as well as for employed and unemployed subjects. Furthermore, associations between the various POMS scales and the period of time since infection will be examined in order to ascertain whether the various emotional states are more prevalent at different times after infection, such as soon after notification of seropositive status and the diagnosis of AIDS.

Although short-term emotional states are not generally regarded as having effects on long-term immunological functioning, there are indications that scales measuring anger may prove to be predictive of subsequent immune measures, particularly when levels of anger interact with levels of social desirability (Ostrow, 1988). Furthermore, the same study found significant associations between most of the POMS scales and total lymphocyte counts. Therefore, all POMS scales will be analyzed in order to determine whether associations with immune measures exist.

Regarding coping strategies, loneliness, hopelessness and joy, linear associations between the various psychosocial constructs and immune measures will be examined, as well as multi-factorial analyses of the effects of coping strategies which emerge as predictive of ten percent or more of the variance of the immune measures, as such predictive power equates to substantial clinical effects.

Linear associations between immune measures and composite scores for problem-focused, emotion-focused, and avoidance coping, will also be examined. It is expected that problem-focused coping will be less efficacious for long-term immune functioning compared to emotion-focused coping, as well as compared to avoidance coping. The repression of emotions is also expected to be associated with lower immune function measures.

A final objective of this study is to survey the needs of HIV-infected individuals regarding emotional and instrumental problems, as well as determining knowledge of, and receptivity towards, programs or methods of coping.

# 5.3 DESCRIPTION OF THE SAMPLE

Thirty-one participants were recruited at the Cape Town branch of ASET (AIDS Support and Education Trust), and are all HIV-seropositive homosexual males. Most of the subjects are residents of urban cities or suburbs (96.7%), of which 90% (n=27) live in Cape Town urban and suburban areas. The racial composition of the sample is largely urbanized white and mixed-race males, and is consistent with so-called 'First World' infection, namely HIV-1 infection. Although the number of subjects who participate in active coping programmes such as Body Positive could not be determined due to the confidentiality inherent in such programmes, it can be assumed that the majority of participants living in Cape Town are affiliated or participate in this programme. Similarly, many participants receive ongoing counselling from ASET counsellors.

Of these 31 participants, two were excluded because CD4-cell immune measures were not obtainable, and a further two were excluded due to fact that they did not report a time of infection. Neither medical or personal history files could shed light on these times of infection and immune measures. Therefore, the final sample consisted of 27 participants, with only one of these 27 not having CD8-cell counts on record. The obtained raw data is tabulated in Appendix 5, tables 5-A through 5-J.

This sample of 27 participants thus adequately complies with the previously estimated sample size needed to reveal true effects (refer Chapter 4, section 4.10), namely between 20 and 34 subjects.

### 5.3.1 DEMOGRAPHIC DESCRIPTION OF THE SAMPLE

The following demographic information regarding home language, highest educational level attained, age, and employment status was obtained (refer Appendix 5, Table 5-A for raw data):

TABLE 7
---------

## **DEMOGRAPHIC DESCRIPTIVE DATA**

(N = 27)

1. Home Language	f	%
English	: 16	(59.3 %)
Afrikaans	: 8	(29.6 %)
Both English & Afrikaans	: 3	(11.1 %)

### 2. Employment:

Unemployed	: 13	(48.1 %)
Employed	: 14	(51.9 %)

## 3. Monthly income (Those employed):

Median monthly income : R 2100 p.m.

Range : R 1250 - R 16250 p.m.

### 4. Highest Educational Level:

Less than Matric	: 2	( 7.4 %)
Matric	: 14	(51.9 %)
Post-Matric	: 9	(33.3 %)
Unknown	: 2	(7.4%)

#### 5. Age

Range: 20.3y - 50.8y Mean: 33.2y Std = 7.5y

It appears that the sample is predominantly English-speaking, with approximately 30% being Afrikaans-speaking. There is also a high rate of unemployment, with almost half of the sample being unemployed.

The sample also appears to be fairly well educated, with approximately half being high-school graduates, and a further third having tertiary education.

# 5.3.2 CLINICAL DESCRIPTION OF THE SAMPLE

The clinical profile of the sample is presented in Table 8 (refer Appendix 5, Table 5-B for raw data). Not reflected in this table or the previous one, is the fact that almost half of those who are unemployed (n = 6; 46.2%) are symptomatic. Several of these subjects indicated that they live on disability pensions.

TABLE 8 CLINICAL PROFILE OF THE SAMPLE					
CDC Sta	ge Classification (N = 27):				
11	Asymptomatic	10	(37.0 %)		
111	Asymptomatic, with PGL	5	(18.5 %)		
IVa	Asymptomatic, CD4 below 200	1	( 3.7 %)		
IVa	Non life-threatening infections	4	(14.8 %)		
IVc-1	Secondary infections, Type 1	4	(14.8 %)		
IVc-2	Secondary infections, Type 2	3	(11.1 %)		
IVd .	Lymphomas eg Kaposi's Sarcoma	0	( 0.0 %)		
IVe	Other: Co-existent condition	0	( 0.0 %)		
SUMMAR	Y:				
Asympto	matic (II,III, Asympt IVa)	16	(59.3 %)		
Sympton	natic:				
Mil	d symptoms (IVa)	4	(14.8 %)		
Sed	condary Infections (IVc-1&2,IVe)	7	(25.9 %)		

Due to the small number of subjects in CDC stages IVa and other stage IV (AIDS) symptom categories, all stage IV(a-e) data are grouped together. Thus, there are 16 asymptomatic subjects (including one stage IVa subject with no symptoms), and 11 symptomatic subjects. The reason for including this one asymptomatic stage IVa subject with the stages II and III subjects in subsequent analyses, is that the presence of symptoms is considered a major source of distress. Therefore, analyses of emotional correlates of symptom-status needs to distinguish between those who are symptomatic stage IVa, and those who are not, regardless of CD4-cell counts.

### 5.3.3 IMMUNOLOGICAL PROFILE OF THE SAMPLE

In figures 3 and 4, CD4 and CD8-cell counts are plotted against the mean time since infection in order to provide an overview of the immunological status of the sample, utilizing the raw unadjusted data obtained (refer Appendix 5, Tables 5-B and 5-C).

There are several cautionary aspects to these descriptive figures: First of all, it should be noted that the mean of the reported dates between which infection is considered most likely to have occurred is used as the time of infection. A more detailed evaluation of this method of ascertaining the time of infection is discussed later, in section 5.3.5.

Secondly, the CD4-cell counts indicated in figure 3 are not adjusted for antiretroviral and experimental drug effects, which subsequent analyses will take into consideration.

Finally, included in both figures 3 and 4 are five subjects who reported that they had been infected within two years of the immunological measurements reflected in these figures. As both CD4 and CD8-cell counts fluctuate dramatically within this early period of infection, subsequent analyses will examine this aspect in greater detail, in order to evaluate the necessity for dealing with such subjects as a separate grouping.

FIGURE 3

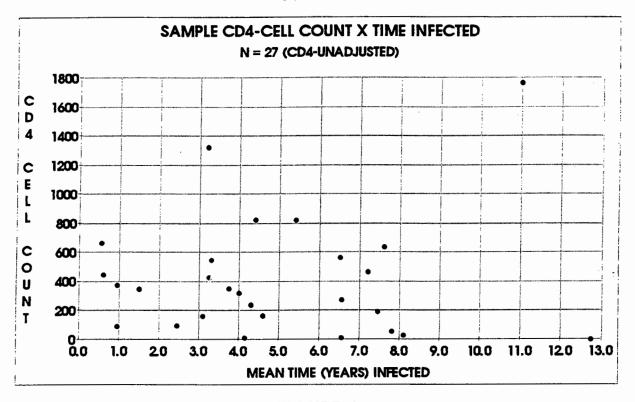
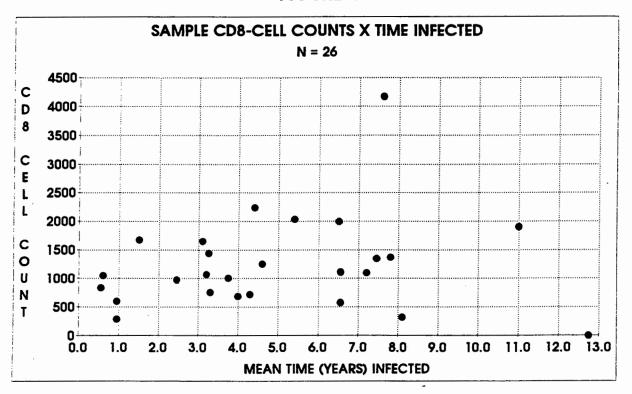


FIGURE 4



The following descriptive statistics are applicable to figures 3 and 4:

TABLE 9

CD4-CELL, CD8-CELL & MEAN LENGTH OF INFECTION

	Mean	StDev	N
CD4-cell counts (per microlitre)	413.5	402.0	27
CD8-cell counts (per microlitre)	1237.2	808.1	26
Mean length of infection	4.9yrs	3. 1yrs	27

When the obtained means are compared to the normative data provided by Lang et al. (1989) (refer figure 1a and 2, pp.25 and 26), it is apparent that the obtained sample conforms to these normative means. For example, Lang et al. (1989) report CD4-cell counts of approximately 361 to 446 for the period spanning 4.5 and 5.0 years post-infection (refer figure 1a, p.25), while the sample obtained has a mean of 413 (std = 402; n = 27) at 4.9 years post-infection. Similarly, CD8-cell counts appear to be within normative parameters, with Lang et al. (1989) reporting a mean of approximately 1060 to 1134 (refer figure 2, p.26), while the sample has a mean of 1237.2 (std = 808: n = 26).

Immunologically, the sample appears to be a fair representation of the norms obtained for CD4 and CD8-cell counts during HIV-infection. This is further suggested by the fact that CD4-cell counts range from zero to 1764, CD8-cell counts range from zero to 4172, and the time since infection includes several recent seroconverters (eg., 0.6 and 1.0 years post-infection), as well as a few who have been infected for lengthy periods of time (eg., 12.8 and 11.0 years).

# 5.3.4 ANTIRETROVIRAL & EXPERIMENTAL DRUG EFFECTS

One-third (n = 9) of the sample have used, or are currently using, antiretrovirals, such as AZT (Zidovudine), ddl, ddC, and d4T. In addition, two-thirds of the sample have been participating in the experimental drug-trial being conducted at ASET (i.e., the immunoenhancing natural compound) for up to five months prior to immune measurements (mean usage = 2.5 months, st.dev = 1.2 months; n = 18).

Six of the subjects who used antiretrovirals also used the experimental drug, which is a naturally-occurring (non-antiretroviral) compound with immunoenhancing effects. The periods of usage are tabulated in Table 5-D of Appendix 5, and range from zero to five months.

In order to minimize these variations and to separate the effects of such drug treatments from possible effects of psychosocial variables, each subject's CD4-cell count needs to be adjusted for their specific drug treatment profile. CD8-cell counts are largely unaffected by the drug treatments which the sample have been exposed to, and are therefore not adjusted.

Usage of antiretrovirals conforms largely to the American protocols, namely commencing treatment (usually AZT, followed by DDI, DDC or D4T later) when CD4-cell counts reach between 450 and 500. According to the recently published results of the Concorde Trial involving 1762 subjects followed up for three years, AZT produces no significant differences between those who started treatment early or later, regarding survival rates and the onset of symptoms. Early onset of treatment produced an average increase of 20 CD4-cells over the first three months, and a decrease of 10 CD4-cells in those who commenced treatment later. This difference was maintained during the entire 3-year follow-up period (Medical Research Council (UK) Press Notice, 2 April 1993).

Therefore, for those subjects who had used antiretrovirals for three months and longer, CD4-cell counts for all subjects who commenced treatment with AZT prior to development of symptoms were reduced by 20, while those who commenced AZT treatment after development of symptoms were increased by 10 (refer Appendix 5, Table 5-E).

Four subjects have received convergent therapy (i.e., simultaneous treatment with AZT, ddl, ddC or d4T). However, recent reports by influential research evaluation groups, such as the Treatment Action Group (TAG, in 'Report slams government AIDS Research', 1994) suggest that additional adjustments to CD4-cell counts are not warranted.

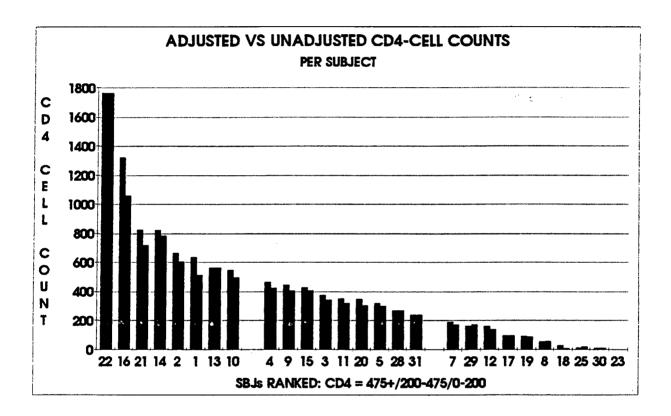
It has been mentioned that the majority of the subjects are drawn from participants in an experimental immunoenhancing (as opposed to antiretroviral) drug trial. Preliminary results indicate a consistent average monthly increase of approximately five percent in CD4-cell counts ascribed to the experimental drug in CD4-cell counts, but not for CD8-cell counts. Therefore, the mean CD4-cell count change per month ascribed to the drug was subtracted from the most recent CD4-cell count according to months of usage prior to the immune measure, in order to produce a final corrected CD4-cell count. The adjustments made are based upon two years of experimental trials, and the effects are considered to be reliable and consistent. Only one subject in the sample did not respond to the experimental drug, and his CD4-cell count was thus not adjusted.

In cases where adjustments reduced CD4-cell counts to levels below zero, the adjusted count was considered to be zero, as negative cell counts are not realistically possible.

Figure 5 contains a comparison between the unadjusted CD4-cell counts (solid bars) and the adjusted CD4-cell counts (shaded bars). Subjects were ranked according to initial unadjusted CD4-cell counts, and divided into three groups.

These three groups consisted of those subjects with cell counts above 475 cells per microlitre (which is usually the level at which antiretroviral usage is commenced; n = 8), those between 475 and above 200 (n = 9), and those with cell-counts below 200 (n = 10), which is one of the CDC criteria for AIDS diagnosis (stage IVa-e).

FIGURE 5



From figure 5, it is apparent that there were no significant changes in the ranking of subjects due to the adjustments for drug usage for CD4-cell counts. There were also no subjects who changed from one of the three CD4-cell categories to another as a result of the adjustments.

From Table 5-E in Appendix 5, the following data is obtained:

TABLE 10

ADJUSTED VS UNADJUSTED CD4-CELL COUNTS
FOR THREE GROUPS:
0-200, 201-475, AND 475+
CD4-CELLS/MICROLITRE

	CD4-CELL	LDANU	CD4	ADJUST	ED CD4	N	r	T
	RANGE	MEAN	STD	MEAN	STD			(1-Tailed)
GROUP A:	475+	891.6	403.5	811.4	398.9	8	0.980	2.630 p < 0.025
GROUP B:	201-475	359.1	072.8	333.2	061.4	9	0.985	4.544 p < 0.005
GROUP C:	0-200	079.9	066.9	075.4	063.8	10	0.986	1.193 p < 0.300
TOTAL		413.5	402.0	379.4	374.4	27	0.992	3.150 p < 0.005

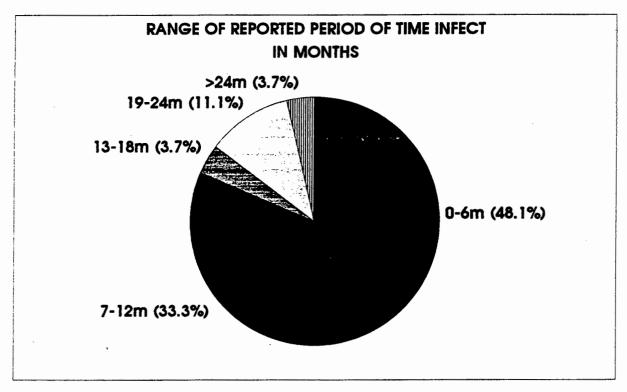
From table 10 it appears that the adjustments to CD4-cell counts were significant for those subjects with cell-counts between 200 and 475 (p < 0.005) as well as those subjects with counts above 475 (p < 0.025). However, the adjustments were not significant for those with counts below 200, which comprises the immunological CDC criteria for AIDS diagnosis (p < 0.30). However, correlations between all three sets of adjusted and unadjusted CD4-cell counts are very high (between 0.980 and 0.992), indicating that the adjusted and unadjusted sets of data covary very highly. Therefore, for the purposes of subsequent analyses, only the CD4-cell counts which have been corrected to drug usage (CD4Adj) are used, as these cell-counts reduce variance due to medical drug usage, and the adjustments made are unlikely to alter covariation between CD4-cell counts and other factors.

## 5.3.5 TIME SINCE INFECTION

Prior mention has been made of the methodological issues surrounding the reporting of the time of infection. Subjects were provided with the option to indicate either a specific month and year of infection, or to indicate a period of months or years between which infection was most likely to have occurred. As the time of infection is an important component of analyses involving progression of HIV-infection over time, a closer examination of the data pertaining to the time of infection is appropriate.

Table 5-c of Appendix 5 indicates that the mean range of the reported period of likely infection is 0.685 years (approx. 8 months), with a standard deviation of 0.725 years (approx. 9 months).

FIGURE 6



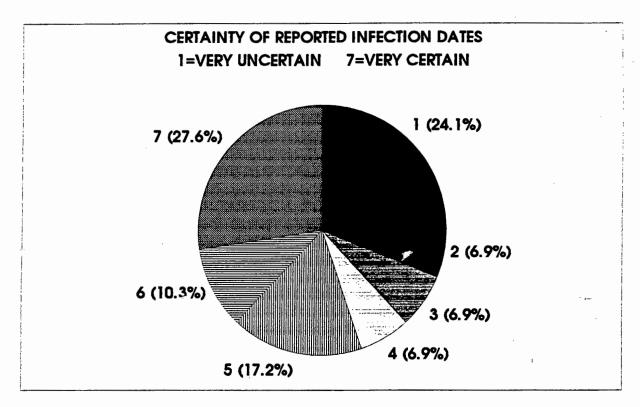
From figure 6 it appears that the majority of the sample (84.4 %) reported dates of most likely infection that ranged from zero to 1 year apart. A further 14.8 percent provided infection dates that were between one and two years apart, and only one subject provided a time range greater than two years (3 years). The median reported range of the dates between which infection occurred is 0.6 years (7 months).

In general, a mean between the two dates provided would be adequate for statistical purposes, as such a mean would potentially contain a low level of error, which would range from zero to six months for the large majority of the sample.

However, despite the relatively narrow time range in which infection was considered most likely to have occurred, this does not directly indicate the subject's confidence in this reported time of infection. Therefore, a brief examination of the accompanying reported degree of certainty regarding these times of infection is appropriate.

From Table 5-C of Appendix 5, it appears that the mean level of confidence regarding the reported most likely times of infection is 4.1 (st.dev = 2.4). This mean confidence score is approximately midway on the seven-point likert-type scale, where a score of seven indicates very high certainty regarding the reported dates of infection.

FIGURE 7



The above figure 7 indicates that, of the 27 subjects, 55.1 percent indicated that they were fairly confident to very confident (scores 5 to 7) regarding the reported dates of infection, while 37.9 percent were a little uncertain to very uncertain (scores 1 to 3). However, it should be noted that information obtained from medical and personal records were utilized to verify all reported periods of infection, thus increasing the overall accuracy of all dates of infection.

In general, the verification process revealed few discrepancies between the dates reported and those apparent from medical and other records. Apart from two subjects of the original 31 subjects who did not report a time of infection, and where additional records could not be found to locate such a date, only four reported dates of infection were modified due to additional medical and other information (refer Appendix 5, Table 5-C), while the remainder appeared to be congruent with such records.

## 5.3.6 RATE OF CD4-CELL DECLINE OVER TIME

Although HIV infection progresses over a number of years, individual variability regarding the rate of CD4-cell decline over time varies widely, as evidenced from figure 3. As this aspect of HIV infection is a major focus of the present study, a careful examination of the samples' CD4-decline rate is necessary.

There are several methodological advantages for utilizing the rate of CD4-cell decline as the most prominent immunological parameter for analyzing associations with psychosocial variables in HIV infection. First of all, the rate of CD4-cell decline is considered to be approximately linear (Lang *et al.*, 1989), with the exception of the first 12 to 18 months post-infection, where the rate of CD4-cell loss is both highly variable and rapid. Thereafter, the rate of decline tends to stabilize (refer figures 1a and 1b, p.25), and then increases again upon development of AIDS.

Furthermore, the study is opportunistic in terms of entering an existing drug-trial in which immune measures were done at times which often differed from the psychosocial measurements, often by several months. Therefore, the rate of CD4-cell decline (calculated at the time of immune measurement, and assumed to reflect the rate at the time of psychosocial measurement) will serve as a more accurate measurement of each individual's overall immunological functioning, compared to simple adjusted CD4-cell counts. This is due to the fact that CD4-cell counts tend to fluctuate over short periods of time, and the rate of CD4-cell decline would tend to provide a more stable indication of CD4-cell levels, as such variations are divided by the length of time since infection. However, caution would be appropriate in calculating rates of decline for those who have been infected for short periods of time, as the error variance contained within the CD4-cell counts would be higher than for those who have been infected for much longer periods of time.

The time difference between immunological and psychosocial measurement (refer Appendix 5, Tables 5-A and 5-B), was quite small (mean difference = 0.16y, i.e., 2 months; std. = 0.11y., i.e., 1.3 months). Three subjects (11.1 %) had concurrent measurements, and there were differences of one month (n=12; 44.4 %), two months (n=5; 18.5 %), three months (n=6; 22.2 %), and four months (n=1; 3.7 %).

In order to calculate the rate of CD4-cell decline since infection, the following procedure was applied:

According to Lang *et al.* (1989), the mean pre-infection CD4-cell count is approximately 1119. Therefore, in order to calculate the mean rate of CD4-cell decline over time (hereafter referred to as **CD4-rate**) per individual, the subject's most recent CD4-cell count was first adjusted for drug usage (hereafter **CD4-adjusted**), then subtracted from 1119, and finally divided by the time from infection (hereafter referred to as **time-infected**) until immune measurement. The latter was calculated by obtaining the mean between the two dates reported as reflecting the most likely period of infection, and obtaining the difference between this mean and the date of the immune measurement. These calculations are tabulated in Appendix 5, Table 5-F.

A cursory view of the calculated CD4-rates for the sample (refer Appendix 5, Table 5-F) indicates that five subjects have very high CD4-rates of decline. All five of these subjects have reported times of infection equal to or less than 18 months (mean CD4-adj = 347.4, std = 1861; mean years infected = 0.94 years, std = 0.37; mean CD4-rate = 881.1 cells per year of infection, std = 246.1 years). As previously mentioned, the first 18 months after initial infection is characterized by a rapid drop in CD4-cell counts, as well as high variability of such counts. Therefore, the inclusion of these subjects' CD4-cell counts and CD4-rates would inflate error variance, and these CD4-cell counts and CD4-rates are excluded from all following statistical analyses involving immunological measures. These subjects are, however, retained in other types of analyses, such as descriptive profiles related to time since infection.

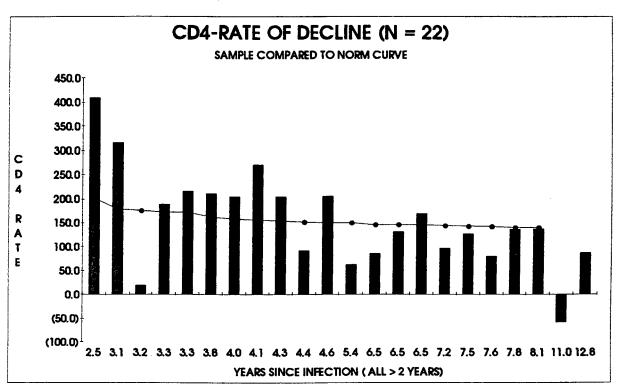
Table 11 and figure 8 provide a descriptive overview of the rates of CD4-cell decline:

TABLE 11

CD4-RATES OF DECLINE FOR SUBJECTS
INFECTED LONGER THAN 18 MONTHS (N = 22)

	MEAN	STDev
CD4-ADJUSTED:	379.4	381.5
YEARS INFECTED:	5.795	2.657
CD4-RATE:	154.2	101.7

FIGURE 8



The norm curve in figure 8 (solid horizontal line extending from approx. CD4-rate of 220 at 2.5 years to approx. 140 at 8.0 years) is based upon the norms provided by Lang et al. (1989) for HIV infection until AIDS diagnosis at approximately six years post-infection, and projected forward to reflect an absolute zero CD4-cell count at approximately eight years post-infection. Each vertical bar represents one of the 22 subjects CD4-rate plotted against the time since infection.

Table 11 indicates that the mean sample CD4-rate (154.2 CD4-cells decline per year, std. = 101.7; at a mean period of infection of 5.8 years) is almost identical to the norm cumulative rate of 155 CD4-cells decline per year at 6.0 years reported by Lang et al. (1989; refer figure 1b, p.25). In addition, figure 8 reveals that ten subjects fall below this mean curve, ten fall above it, and two subjects are positioned very close to the norm CD4-rate at the relevant times since infection.

It thus appears that the sample contains a fairly representative cross-section of CD4-cell rates of decline when compared to existing norms.

## 5.3.7 PSYCHOSOCIAL COMPARISONS WITH NORMS

In the following three tables, the mean scores for the entire sample (N = 27) for the COPE, POMS, and UCLA Loneliness survey are compared to existing (college students, assumed to be seronegative) norms. No norms are available for the scales for measuring joy and hopelessness.

TABLE 12

# PSYCHOSOCIAL MEASUREMENTS: COMPARISON OF SAMPLE (N = 27) WITH NORMS

# (PROFILE OF MOOD STATES)

	SAMF HIV+ HOM MALE N = 2	OSEXUAL ES	MALE COLL STUDI N = 3	EGE Ents *		
		, N STD		N STD	T(2-tailed) df=365	р
TA	14.9	10.9	12.9	06.8	+1.395	< 0.30 (ns)
DD	20.4	17.2	13.1	10.5	+3.286	< 0.01
AH	16.4	13.2	10.1	07.8	+3.795	< 0.001
VA	17.4	07.5	15.6	06.0	+1.471	< 0.20 (ns)
FI	13.1	08.2	10.4	06.2	+2.122	< 0.05
СВ	11.4	07.7	10.2	05.2	+1.108	< 0.30 (ns)

From: McNair, Lorr & Droppleman (1971). Refer Appendix 3, Table 3-F.

#### **ABBREVIATIONS:**

TA = Tension-Anxiety
AH = Anger-Hostility
FI = Fatigue-Inertia

DD = Depression-Dejection

VA = Vigour-Activity

CB = Confusion-Bewilderment

# TABLE 13

# PSYCHOSOCIAL MEASUREMENTS: COMPARISON OF SAMPLE (N = 27) WITH NORMS

# (UCLA LONELINESS SURVEY)

SAMPLE	ADULTS	
HIV+	AGED 31-40 *	
HOMOSEXUAL		
MALES		
N = 27	N = 94	
MEAN STD	MEAN STD	T (2-tailed)
		df=119 p
9.74 3.31	8.17 1.97	+3.087 < 0.01

<sup>\*</sup> From: Russell (1982). Norms for age group 31-40 yrs. Refer Appendix 2, Table 2-J.

## TABLE 14

# **PSYCHOSOCIAL MEASUREMENTS: COMPARISON OF SAMPLE (N = 27) WITH NORMS**

# (COPING ORIENTATIONS TO PROBLEMS EXPERIENCED)

	SAMPLE			
	HIV+ HOMOSEXUAL	COLLEGE STUDENTS *		
	MALES N = 27	N = 1030		
	MEAN STD	MEAN STD	T(2-tailed)	
			df=1055	P
Active	11.30 3.06	11.89 2.26	-1.326	< 0.20 (ns)
Plan	12.00 3.04	12.58 2.66	-1.114	< 0.30 (ns)
Suppr	10.00 2.50	09.92 2.42	+0.169	> 0.50 (ns
Restr	10.56 2.49	10.28 2.53	+0.568	> 0.50 (ns)
SSSI	10.44 2.80	11.50 2.88	-1.889	< 0.10 (ns)
SSSE	09.59 3.21	11.01 3.46	-2.109	< 0.05
Pos	12.63 3.08	12.40 2.42	+0.484	> 0.50 (ns)
Relig	08.41 4.09	08.82 4.10	-0.513	> 0.50 (ns)
/ent	10.04 2.70	10.17 3.08	-0.217	> 0.50 (ns)
Accept	14.00 2.08	11.84 2.56	+4.346	< 0.001
Deny	07.22 3.62	06.07 2.37	+2.449	< 0.02
3Dis	07.93 3.14	06.11 2.07	+4.439	< 0.001
MDis	10.82 2.53	09.66 2.46	+2.417	< 0.02
AlcDis	01.78 1.05	01.38 0.75	+2.704	< 0.01

<sup>\*</sup> From: Carver, Scheier & Weintraub (1989). Refer appendix 2, table 2-D.

#### **ABBREVIATIONS:**

Active = Active coping

Plan = Planning

Suppr = Suppression of competing activities

Restr = Restraint coping

SSSI = Seeking social support for instrumental reasons

SSSE = Seeking social support for emotional reasons

Pos = Positive reinterpretation and growth

Accept = Acceptance Vent = Focusing and venting of emotions

Relig = Turning to religion Deny = Denial

BDis = Behavioural Disengagement

MDis = Mental Disengagement

AlcDis = Alcohoi and drug Disengagement

From table 12, it appears that the sample of HIV-infected homosexual men at various stages of HIV infection (N = 27) is significantly more *depressed*, *hostile* and *fatiguea* than the college student norms (t = +3.286, p < 0.01; t = +3.795, p < 0.001; and t = +2.122, p < 0.05, respectively, df = 365).

Table 13 reveals that the sample is significantly *lonelier* than the norm group for ages 31 to 40 years old (t = +3.087, p < 0.01, df = 119).

Table 14, which compares the sample with college student norms, indicates that the sample scored significantly lower in the COPE measure of seeking social support for emotional reasons (t = -2.109, p < 0.05, df = 1055), and significantly higher in all COPE scales related to acceptance-avoidance coping (Acceptance: t = +4.346, p < 0.001; Denial: t = +2.449, p < 0.02; Behavioural disengagement: t = +4.439, p < 0.001; Mental disengagement: t = +2.417, p < 0.02; Alcohol and drug disengagement: t = +2.704, p < 0.01; df = 1055 for all 2-tailed t-tests, with alpha = 5%).

There were no significant differences for problem-focused coping, including active coping (t = -1.326, p < 0.20, df = 21), planning (t = -1.114, p < 0.30, df = 21), suppression of competing activities (t = 0.169, p > 0.50, df = 21), and restraint coping (t = 0.568, p > 0.50, df = 21). The COPE scale of seeking social support for instrumental reasons approached significance (t = -1.889, p < 0.10, df = 21), with the sample having lower mean scores than the college student groups for this scale.

There were also no significant differences between the sample and the student norm group regarding coping strategies of positive reinterpretation and growth (t = 0.484, p > 0.50, df = 21), turning to religion (t = -0.513, p > 0.50, df = 21), and focusing upon and venting of emotions (t = -0.217, p > 0.50, df = 21).

In order to establish how emotional distress related to symptom-status in the present study compares to those obtained in equivalent studies, the POMS scale scores of asymptomatic and symptomatic subjects were compared to the data obtained from two such studied, namely Blaney *et al.* (1990) and Atkinson *et al.* (1988).

Table 15 compares the asymptomatic subjects (N = 16) with norms obtained from pooling the data from Blaney *et al.* (1990) and Atkinson *et al.* (1988), which thus provides a comparison group of 62 homosexual HIV-seropositive asymptomatic (CDC II and III) males.

Table 16 compares the symptomatic subjects (N = 11) with the data obtained from Atkinson *et al.* (1988). It should be noted that the data obtained from latter study consisted only of subjects with ARC (CDC IVa), and is a relatively small comparative sample (N=13). However, alternative data for a broader spectrum of symptom categories was not available for a more satisfactory comparison.

TABLE 15

POMS: COMPARISON OF ASYMPTOMATIC SUBJECTS (N = 16)
WITH ASYMPTOMATIC NORMS (N = 62)\*

	HIV+ A <b>Samp</b> i N = 16	е	IATIC HOMO Norm N = 69		EN
	Mean	StD	Mear	stD	T(df=76) p
TA	16.6	11.7	13.2	07.5	+1.427 p < 0.20
DD	21.9	19.1	11.5	09.6	+3.070 p < 0.01
AH	16.7	13.0	08.5	06.9	+3.457 p < 0.001
VA	17.8	09.2	17.1	07.6	+0.314 p > 0.50
FI	14.2	09.1	07.0	05.3	+4.117 p < 0.001
CB	12.3	08.3	08.3	05.5	+2.318 p < 0.05

<sup>\*</sup> Sample data obtained from Blaney *et al.* (1990) and Atkinson *et al.* (1988); Pooled standard deviations & weighted means used..

TABLE 16

POMS: COMPARISON OF SYMPTOMATIC SUBJECTS (N = 11)

WITH SYMPTOMATIC NORMS (N = 13)\*

	SYMPTOMATIC H Sample IVa-e N = 11		Norm (ARC) N = 1	)			
	Mean	StD	Mear	n StD	T(df=22) p		
TA	12.4	09.5	16.5	08.6	- 1.110 p < 0.30		
DD	18.3	14.9	17.6	12.7	+0.124 p > 0.50		
AH	15.9	14.1	14.8	11.1	+0.214 p > 0.50		
VA	16.8	04.4	11.4	06.9	+2.235 p < 0.05		
FI	11.5	06.7	14.7	06.9	- 1.147 p < 0.30		
CB	10.1	06.9	11.5	07.0	-0.491 p > 0.50		

<sup>\*</sup> Sample data obtained from Atkinson et al. (1988); 'ARC' subjects

From table 15, it appears that the asymptomatic sample subjects are significantly more distressed than the comparative norm group, on all POMS scales, with the exception of *vigour-activity* (t = +0.314, p > 0.50, df = 76; 2-tailed, alpha = 5 %) and *anxiety-tension* (t = +1.427, p < 0.20, df = 76; 2-tailed, alpha = 5 %). The sample of asymptomatic subjects are more *depressea* (t = +3.070, p < 0.01, df = 76; 2-tailed, alpha = 5 %), *angry-hostile* (t = +3.347, p < 0.001, df = 76; 2-tailed, alpha = 5 %), *fatiguea* (t = +4.117, p < 0.001, df = 76; 2-tailed, alpha = 5 %), and more *confusea* (t = +2.318, p < 0.05, df = 76; 2-tailed, alpha = 5 %), compared to the normative group.

Symptomatic subjects do not appear to be more distressed than the comparative group, with insignificant differences (t = -1.110, p < 0.30; t = +0.124, p > 0.50; t = +0.214, p > 0.50; t = -1.147, p < 0.30; t = -0.491, p > 0.50; all 2-tailed, alpha = 5%; for tension-anxiety, depression, anger, fatigue, and confusion, respectively) for all scales except vigour-activity. In the latter, the sample scored significantly higher than the comparison group (t = +2.235, p < 0.05, t = 22; 2-tailed, alpha = 5%).

# 5.4 STABILITY OF COPING MEASURES

A critical factor in determining the plausibility of directions of causality between coping, attitudinal factors and immunological measures, concerns the stability of these psychosocial factors under different circumstances. For example, the development of symptoms, unemployment, maturation with age, and coming to terms with HIV-positive diagnosis over time, may affect the specific coping style used in a specific context. In order to assess this possibility, linear correlations between these life-change, temporal factors, and all COPE scales, loneliness scores, as well as scores for joy and hopelessness, were calculated. The results are tabulated in table 17.

From table 17, it appears that none of the psychosocial coping and attitudinal measures are significantly correlated with the time since infection, nor with the ages of the subjects. However, whether a person is employed or not (with employment scored as 1, and unemployment scored as 0), appears to be significantly positively associated with the seeking of social support for emotional reasons (r = +0.393, p < 0.05, df = 26), and significantly negatively associated with denial coping (r = -.420, p < 0.05, df = 26), behavioural disengagement (r = -.433, p < 0.05, df = 26), as well as the composite score for avoidance coping (r = -.442, p < 0.05). Thus, it appears that unemployment is associated with a decreased likelihood for seeking emotional social support, as well as increased levels of denial coping, behavioural disengagement, and overall avoidance coping.

Symptom-status (with symptomatic status scored as 1 and asymptomatic status scored as 0), does not appear to be significantly associated with any of the coping strategies measured, nor with levels of *loneliness*, *joy* and *hopelessness*.

Therefore, in assessing associations between coping measures and immune functions, caution would be appropriate for interpreting associations with seeking social emotional support, denial, behavioural disengagement and avoidance coping, as employment status may possibly act as a third variable. However, there is no evidence to suggest that the coping constructs measured are susceptible to changes over time, symptom-development, nor aging.

TABLE 17

# CORRELATIONS BETWEEN CLINICAL, DEMOGRAPHIC, COPING, & ATTITUDINAL MEASURES (N = 27)

Time 1 Age<sup>2</sup> Empl<sup>3</sup> Sympt<sup>4</sup> COPING SCALE Α +.098 -. 183 +.218 +.270 Active coping В +.124 -.074 +.000 +.101 **Planning** C -.068 -.283 +.121 +.215 Suppression of competing activities D +.275 -.160 -.054 +.275 Restraint coping +.240 +.031 Ε +.076 -.089 Seeking social support for instrum, reasons F +.150 +.153 -.224 +.251 \* PROBLEM-FOCUSED COPING (A+B+C+D+E) G +.324 -.109 +.393\* +.107 Seeking social support for emot, reasons Н -.203 -. 167 -.020 +.351Positive reinterpretation & growth +.269 -.179 +.205 +.085Turning to religion +.215 -.012 +.109 +.111 Acceptance J +.190 +.056 K +.039 +.218 \* EMOTION-FOCUSED COPING (G+H+I+J) +.353 -.216 \* FOCUS ON & VENTING OF EMOTIONS +.182 +.131 L -, 126 -.021 -.420\* M -.094 Denial coping +.032 +.088 -.433\* Behavioural disengagement N -.029 -.102 0 -.049 +.004 +.001Mental disengagement -.280 Ρ -.288 -. 178 -.041 Alcohol & drug disengagement Q -, 101 +.003 -.442° -.061 \* AVOIDANCE COPING (M+N+O+P) R -.021 +.244 -. 168 -.096 LONELINESS S +. 107 -.216 +.083 +,127JOY T -.041 +.087 -.287 -.025**HOPELESSNESS** 

#### SUPERCRIPTS:

4.896

3.069

27

33.22

07.66

27

.519

.509

27

"" r(crit) = 0.486 for df = 26; = T(crit) = 2.779 (2-tailed) for p = 0.01, alpha = 5 %

.407

.501

27

- " r(crit) = 0.444 for df = 26; = T(crit) = 2.479 (2-tailed) for p = 0.02, alpha = 5%
- \* r(crit) = 0.381 for af = 26; = T(crit) = 2.056 (2-tailed) for p = 0.05, alpha = 5%
- 1 TIME FROM INFECTION TILL IMMUNE MEASUREMENT (IN YEARS)
- 2 AGE IN YEARS
- 3 EMPLOYED (1 = YES; 2 = NO)
- 4. SYMPTOM STATUS (1 = SYMPTOMATIC; 0 = ASYMPTOMATIC)

Mean

Ν

Standard Deviation

# 5.5 SHORT-TERM EMOTIONAL CHANGES

In order to assess whether levels of anxiety, depression, anger, vigour, fatigue and confusion are more prevalent at different periods or stages of HIV infection, as well as whether life-changes such as unemployment and symptom-development result in higher levels of distress, two methods of analysis were conducted, using the POMS scales as indicators of these emotional states. In the first analysis, linear correlations between these mood states and the time since infection, age of respondent, symptom-status, and employment status were conducted (refer table 18). In the second analysis, a series of one-way anovas were conducted (refer table 19), utilizing four categories of HIV-infection, namely asymptomatic infection within two years of infection (early infection; n = 5), asymptomatic infection longer than two years (n = 11), the presence of non-life-threatening illness (CDC IVa; early symptomatic stage; n = 4), and AIDS (CDC IVc-e; late symptom stage; n = 7).

TABLE 18

CORRELATIONS BETWEEN CLINICAL, DEMOGRAPHIC,
& MOOD STATES
(N = 27)

Time <sup>1</sup>	Age <sup>2</sup>	Empl <sup>3</sup>	Sympt <sup>4</sup>	PROFILE OF MOOD STATES SCALE
002	+.193	146	194	TA (Tension-Anxiety)
179	102	215	106	DD (Depression-Dejection)
+.017	+.082	110	030	AH (Anger-Hostility)
+. 181	224	007	066	VA (Vigour-Activity)
028	+.324	098	162	Fl (Fatigue-Inertia)
178	+.281	106	145	CB (Confusion-Bewilderment)
4.896	33.22	.519	.407	Mean
3.069	07.66	.509	.501	Standard Deviation
27	27	27	27	N

#### **SUPERCRIPTS:**

- r(crit) = 0.486 for df = 26; = T(crit) = 2.779 (2-tailed) for p = 0.01, alpha = 5%
- " r(crit) = 0.444 for df = 26; = T(crit) = 2.479 (2-tailed) for p = 0.02, alpha = 5%
- r(crit) = 0.381 for df = 26; = T(crit) = 2.056 (2-tailed) for p = 0.05, alpha = 5%
- 1 TIME FROM INFECTION TILL IMMUNE MEASUREMENT (IN YEARS)
- 2 AGE IN YEARS
- 3 EMPLOYED (1 = YES; 2 = NO)
- SYMPTOM STATUS (1 = SYMPTOMATIC; 0 = ASYMPTOMATIC)

## TABLE 19

# LEVELS OF PROFILE OF MOOD STATES FOR SYMPTOM-CATEGORIES

### 1-WAY ANOVAS

#### 4 SYMPTOM-CATEGORIES:

- (1) ASYMPTOMATIC, INFECTED 0 TO 2 YEARS (EARLY ASYMPTOMATIC; n = 5)
- (2) ASYMPTOMATIC, INFECTED 2+ YEARS (LATE ASYMPTOMATIC; n = 11)
- (3) SYMPTOMATIC, STAGE IVa (EARLY SYMPTOMATIC; n = 4)
- (4) SYMPTOMATIC, STAGE IVb-e (LATE SYMPTOMATIC: AIDS; n = 7)

#### X POMS SCALES

	-	SS	DF	Ms	. ~ F ·	р
TENSION-ANXIETY	TA ERROR	0219.234 2848.173	3 23	073.078 123.834	0.590	p > 0.05
DEPRESSION-DEJECTION	DD ERROR	0390.464 7376.203	3 23	130.155 320.705	0.406	p > 0.05
ANGER-HOSTILITY	AH ERROR	0289.873 4214.424	3 23	096.624 183.236	0.527	p > 0.05
VIGOUR-ACTIVITY	VA ERROR	0038.127 1424.392	3 23	012.709 061.930	0.205	p > 0.05
FATIGUE-INERTIA	FI ERROR	0071.425 1661.242	3 23	023.808 072.228	0.330	p > 0.05

From tables 18 and 19, it appears that none of the emotional states measured in the various POMS scales are significantly associated with the time since infection, age of the subject, employment status, and symptom-status (i.e.,  $r < r_{crit} = 0.374$  for all associations, thus p > 0.05, alpha = 5 %, df = 26). The latter finding is replicated by the absence of any significant differences between the four symptom categories, in terms of all POMS scale scores (i.e.,  $F < F_{crit} = 3.03$  for all analyses, thus p > 0.05, alpha = 5 %, df = 3.23)

# 5.6 ASSOCIATIONS WITH IMMUNE MEASURES

In the following analyses, all subjects who have been infected 18 months or less at the time of immune measurement are excluded, as the high variability of immune functioning during this early post-infection time may introduce undue error variance into the associations between psychosocial constructs and immune levels. Therefore, the sample size for CD4-cell based analyses is 22. As one of these 22 subjects did not have CD8-cell counts recorded, the sample size for CD8-cell analyses is 21.

It should be noted that interaction effects on immune levels between psychosocial and demographic constructs could unfortunately not be determined, as the sample size proved to be too small, resulting in cells-sizes less than five for several interaction categories, thus making the resulting analyses statistically unreliable.

For all linear associations (r), significance levels (p) were determined by determining the equivalent t-test value, for 2-tailed tests of significant differences, at alpha of five percent.

Furthermore, although a correlation of 0.316 (i.e.,  $r^2 = 0.100$ ) is not significant at p = 0.05 (alpha = 5 % and df = 21 and df = 20), all associations of  $r^2$  equal to, or greater than, 10 % of CD8, CD4-cell and CD4-rate of decline variance (r approx. > 0.316 and < -0.316) are considered to be of interest, as this represents a substantial clinical effect size on immune measures.

## 5.6.1 CD8-CELL COUNTS: LINEAR ASSOCIATIONS

Linear associations between all the psychosocial constructs and CD8-cell counts were conducted. The results are tabulated in table 20.

From table 20, it is clear that none of the demographic (including time since infection and symptom-status) and psychosocial measurements are significantly correlated with CD8-cell counts (i.e.,  $r < r_{crit} = 0.432$ , df = 20, alpha = 5 %. Thus, p > 0.05 for all associations), indicating an almost complete absence of any associations between the psychosocial constructs measured and CD8-cell counts.

No demographic or psychosocial factor predicted ten percent or more of CD8-cell count variance.

# TABLE 20

# LINEAR ASSOCIATIONS BETWEEN CD8-CELL COUNTS, DEMOGRAPHIC, AND PSYCHOSOCIAL FACTORS

# x CD8-cell Counts (N=21)

	DEMOGRAPHIC:					
- 0.012	Time since infection (Years)					
- 0.045	Age of subject					
- 0.098	Symptom-Status (Sympt = 1; Asympt. = 0)					
+0.188	Employment (Employed = 1; Unempl. = 0)					
	COPE SCALES					
- 0.065	Active coping					
- 0.095	Planning					
- 0.226	Suppression of competing activities					
- 0.097	Restraint coping					
- 0.192	Seeking social support for instrum, reasons					
- 0.187	* PROBLEM-FOCUSED COPING (A+B+C+D+E)					
- 0.134	Seeking social support for emot. reasons					
+0.034	Positive reinterpretation & growth					
- 0.138	Turning to religion					
+0.003	Acceptance					
- 0.122	* EMOTION-FOCUSED COPING (G+H+I+J)					
- 0.156	* FOCUS ON & VENTING OF EMOTIONS					
+0.150	Denial coping					
- 0.012	Behavioural disengagement					
+0.138	Mental disengagement					
+0.014	Alcohol & drug disengagement					
+0.111	* AVOIDANCE COPING (M+N+O+P)					
- 0.003	UCLA LONELINESS					
+0.059	JOY					
- 0.003	HOPELESSNESS					
	POMS:					
+0.100	TA: Tension-Anxiety					
0.039	DD: Depression-Dejection					
0.053	AH: Anger-Hostility					
+0.054	VA: Vigour-Activity					
+0.019	FI: Fatigue-Inertia					
+0.050	CB: Confusion-Bewilderment					
319.7	Mean CD8-Cell count					
870.5	Standard Deviation					
21	N					
	r(crit) = 0.547 for df = 20; = $T(crit)$ = 2.845 (2-tailed) for p = 0.01, alpha = 5%					

r(crit) = 0.547 for df = 20; = T(crit) = 2.845 (2-tailed) for p = 0.01, alpha = 5%

r(crit) = 0.502 for df = 20; = T(crit) = 2.528 (2-tailed) for p = 0.02, alpha = 5 % r(crit) = 0.432 for df = 20; = T(crit) = 2.086 (2-tailed) for p = 0.05, alpha = 5 %

# 5.6.2 CD4-CELL COUNTS & CD4-RATE OF DECLINE: LINEAR ASSOCIATIONS

Linear associations between all the psychosocial constructs and adjusted CD4-cell counts and CD4-rates of decline (N=22) were conducted. The results are tabulated in table 21 (CD4-cell counts) and table 22 (CD4-rates of decline), on the following two pages.

Only one psychosocial factor, namely **mental disengagement coping**, attained significance in association with adjusted CD4-cell counts (r = -0.488,  $r^2 = 0.238$  CD4-cell variance, df = 21, p < 0.03, alpha = 5 %), while the POMS **vigouractivity** scale approached significance (r = +0.395,  $r^2 = 0.156$ , p = 0.07, df = 21, alpha = 5 %). Only one other factor, **suppression of competing activities**, predicted 10 percent or more of CD4-cell counts (r = 0.332,  $r^2 = 0.110$ , df = 21, p = 0.14, alpha = 5 %).

Of the demographic factors, only symptom status was significantly correlated with CD4-cell counts (r = -0.648, p < 0.002, df = 21). The time since infection was not significantly associated with CD4-cell counts (r = +0.128, df = 21, p > 0.50).

CD4-Rate is significantly negatively correlated with the *time since infection* (r = -0.626,  $r^2=0.392$ , df = 21, p < 0.01, alpha = 5 %), and significantly positively correlated with *symptom-status* (r = +0.445,  $r^2=0.198$ , df = 21, p < 0.05, alpha = 5%). Neither the age of subjects (r = -0.045, p > 0.50, df = 21, alpha = 5 %), nor employment-status (r = +0.188, p > 0.40, df = 21, alpha = 5 %), are significantly correlated with CD4-rate of decline, nor are they predictive of ten percent or more of CD4-rate variance.

Of the psychosocial factors, only the COPE scale of *focusing on and venting of emotions* was significant at the p < 0.05 level, and was significantly negatively associated with CD4-rate of decline (r = -0.433, r = 0.187, df = 21, p < 0.05, alpha = 5%). Only two other psychosocial factors predicted ten percent or more of CD4-rate variance, namely *mental disengagement coping* (r = +0.314, r = 0.10, df = 21, p = 0.16, alpha = 5%), and POMS *vigour-activity* (r = -0.336, r = 0.113, df = 21, p = .13, alpha = 5%).

### TABLE 21

# LINEAR ASSOCIATIONS BETWEEN ADJUSTED CD4-CELL COUNTS, DEMOGRAPHIC, AND PSYCHOSOCIAL FACTORS

r

+0.014

+0.009

-0.084

-0.168

+0.395 p = 0.07

x CD4-cell Counts (N=22)

**DEMOGRAPHIC:** Time since infection (Years) +0.128 -0.247Age of subject - 0.648 p < 0.002 Symptom-Status (Sympt = 1; Asympt. = 0) +0.018 Employment (Employed = 1; Unempl. = 0) **COPE SCALES** +0.182Active coping +0.251 Planning -0.332 p = 0.14Suppression of competing activities -0.172Restraint coping Seeking social support for instrum. reasons +0.069 \* PROBLEM-FOCUSED COPING (A+B+C+D+E) +0.024 Seeking social support for emot, reasons -0.032+0.224Positive reinterpretation & growth -0.199Turning to religion -0.052Acceptance \* EMOTION-FOCUSED COPING (G+H+I+J) +0.031 \* FOCUS ON & VENTING OF EMOTIONS +0.220 +0.084 Denial copina -0.130Behavioural disengagement Mental disengagement - 0.488 p < 0.03\* +0.175 Alcohol & drug disengagement \* AVOIDANCE COPING (M+N+O+P) - 0.150 - 0.157 **UCLA LONELINESS** +0.177 YOL -0.126**HOPELESSNESS** POMS: - 0.056 TA: Tension-Anxiety

386.7	Mean Adjusted CD4-Cell count	
416.3	Standard Deviation	
22	N	

DD: Depression-Dejection

**CB:** Confusion-Bewilderment

AH: Anger-Hostility

VA: Vigour-Activity

FI: Fatique-Inertia

r(crit) = 0.535 for df = 21; = T(crit) = 2.831 (2-tailed) for p = 0.01, alpha = 5%

r(crit) = 0.491 for df = 21; = T(crit) = 2.518 (2-tailed) for p = 0.02, alpha = 5% r(crit) = 0.422 for df = 21; = T(crit) = 2.080 (2-tailed) for p = 0.05, alpha = 5%

# **TABLE 22**

# LINEAR ASSOCIATIONS BETWEEN CD4-RATE OF DECLINE, DEMOGRAPHIC, AND PSYCHOSOCIAL FACTORS

x CD4-Rate (N=22)

	DEMOGRAPHIC:
- 0.626 p < .01	Time since infection (Years)
+0.085	Age of subject
+0.445 p < .05	
+0.080	Employment (Employed = 1; Unempl. = 0)
+0,000	COPE SCALES
- 0.146	Active coping
- 0.272	Planning
+0.284	Suppression of competing activities
+0.082	Restraint coping
- 0.012	Seeking social support for instrum, reasons
- 0.044	* PROBLEM-FOCUSED COPING (A+B+C+D+E)
- 0.013	Seeking social support for emot, reasons
+0.072	Positive reinterpretation & growth
+0.193	Turning to religion
- 0.022	Acceptance
- 0.026	* EMOTION-FOCUSED COPING (G+H+I+J)
- 0.433 p < .05	* FOCUS ON & VENTING OF EMOTIONS
+0.053	Denial coping
+0.085	Behavioural disengagement
+0.314 p = .16	Mental disengagement
- 0.243	Alcohol & drug disengagement
+0.130	* AVOIDANCE COPING (M+N+O+P)
+0.055	UCLA LONELINESS
	JOY
	HOPELESSNESS
	POMS:
- 0.094	TA: Tension-Anxiety
- 0.033	DD: Depression-Dejection
- 0.184	AH: Anger-Hostility
-0.336 p = .13	VA: Vigour-Activity
- 0.139	FI: Fatigue-Inertia
+0.064	CB: Confusion-Bewilderment
154.3	Mean cumulative CD4-Rate of decline
101.7	Standard Deviation
22	N
	" r(crit) = 0.535 for df = 21; = T(crit) = 2.831 (2-tailed) for $p = 0.01$ , dipha = 5%
• -	" $r(crit) = 0.491$ for $df = 21$ ; = $T(crit) = 2.518$ (2-tailed) for $p = 0.02$ , $alpha = 5\%$
	$r(crit) = 0.422$ for df = 21; = $T(crit) = 2.080$ (2-tailed) for $\hat{p} = 0.05$ , alpha = 5%

## 5.6.3 FACTORIAL ANALYSIS OF CD4-CELL COUNTS

Only three factors have emerged as predicting ten percent or more of CD4-cell counts, namely the COPE scales of *suppression of competing activities* coping and *mental disengagement coping*, and the POMS scale of *vigouractivity*.

From table 23, it appears that none of these factors are significantly correlated with the time since infection or symptom-status (p > 0.05, df = 21, alpha = 5 %). Furthermore, none of the inter-factor associations between the three psychosocial factors are significant, indicating that they are relatively independent. It is notable that the time that a person has been infected is not significantly correlated with CD4-cell counts (r = +0.128, p > 0.50, df = 21, alpha = 5 %, 2-tailed), indicating the high variability of CD4-cell counts in HIV infection.

TABLE 23

CORRELATION MATRIX OF ASSOCIATES

OF CD4-CELL COUNTS (N = 22)

	ADJ-CD4 COUNT	TIME	SYMPT	SUPPR	MDIS	VA
TIME SINCE INFECTION	+.128	•				
SYMPTOM-STATUS	171	369	•			
COPE: SUPPR	332	+.049	+.232	•		
COPE: MDIS	488 <b>a</b>	173	056	+.051	•	
POMS: VA	+.395	+.354	183	+.105	396	•
MEAN STD	386.7 416.3	5.795y 2.657y	0.409 0.503	9.818 2.702	10.955 02.681	17.136 07.344

<sup>&#</sup>x27;a': p < 0.05 (r(crit) = 0.422 for df = 21, p = 0.05, alpha = 5 % (2-tailed))

When these three psychosocial factors are entered into a multifactorial stepwise regression, the model emerged as significant (F = 4.042, df = 3.18, p < 0.05), as described in table 24. From table 24, it appears that the model predicts 40.2 percent of CD4-cell counts. Furthermore, the standard error of estimate (S.E.E. = 329.9) is lower than the standard deviation of CD4-cell counts (Std = 416.3), indicating that the model is a superior predictor of CD4-cell counts than knowledge of the mean and standard deviation of CD4-cell counts.

## TABLE 24

# MULTIFACTORIAL MODEL FOR PREDICTING CD4-CELL COUNTS: THREE-PREDICTOR MODEL

man man ga gamam aspang (a a a a a a a a a								
Suppr = Suppression of competing activities coping (COPE scale)								
VA = Vigour-Activity (POMS scale)								
Multip	ie R <sup>2</sup> = 0.402	Multiple R = 0.634			$R^2$ (shrunk) = 0.340			
•								
Beta v	alues:	Coeffi	cients	StDev	T (df = 18)			
MDis	355	- 55.19	71	30.969	-1.782 p < 0.10			
Suppr	344	- 53.05	57	28.373	- 1.870 p < 0.10			
VA	+.290	+16.46	2	11.353	+1.450 p < 0.20			
Constant = 1230.143								
Source	SS	df	MS		F			
Regr	Regr 1464839 3		488279.8		4.042 (p < 0.05)			
Resid 2174580		18	120810					

MDis = Mental disengagement coping (COPE scale)

Predictors (3 predictors)

However, when the *vigour-activity* factor is deleted from the above model, there is only a four percent decrease in the conservative  $R^2(\text{shrunk})$ , to 29.9 percent. Thus, although deleting this factor reduces the overall predictive power of the model to 33.3 percent ( $R^2$ ), the standard error of estimate is not markedly changed (S.E.E. = 348.1). Therefore, the final model consists of only the two coping factors, as described in table 25.

#### TABLE 25

# MULTIFACTORIAL MODEL FOR PREDICTING CD4-CELL COUNTS: TWO-PREDICTOR MODEL

Predictors (2 predictors)

MDis = Mental disengagement coping (COPE scale)

Suppr = Suppression of competing activities coping (COPE scale)

Multiple  $R^2 = 0.333$  Multiple R = 0.577  $R^2(\text{shrunk}) = 0.299$ 

Beta values: Coefficients StDev T (df = 19)

MDis - .472 - 73.337 29.138 - 2.517 p < 0.05

Suppr - .308 - 47.440 28.911 - 1.641 p < 0.15

Constant = 1656.246

Source SS df MS F

Regr 1210863 2 605431.6 4.737 (p < 0.05)

Resid 24285578 19 127818.8

#### 5.6.4 FACTORIAL ANALYSIS OF CD4-RATE OF DECLINE

Unlike CD4-cell counts, CD4-rates of decline are significantly negatively correlated with the time since infection (r = -0.626, p < 0.01, df = 21, alpha = 5 %, 2-tailed), as well as significantly positively correlated with the presence of symptoms (r = +0.445, p < 0.05, df = 21, alpha = 5 %, 2-tailed). As the CD4-rate is calculated in terms of the cumulative loss of finite number of CD4-cells over time, both these associations are to be expected. However, in assessing correlates of the CD4-rate of decline, the issue of time since infection becomes somewhat problematic, as any associations with CD4-rates may be ascribed to third variables, such as either the progression of time itself (eg., aging), or the development of symptoms (eg., changes in coping strategies due to increased distress).

Alternatively, associations with CD4-rates may simply be due to the fact that these factors (e.g., specific coping strategies) are somehow causally involved in immunological functioning, thus either elevating or slowing down the rate at which CD4-cells are lost over time. In this way, it is possible that co-variation of a specific factor with both CD4-rates and the time since infection, may simply be due to the fact that this factor is associated with slow immunological decline, thus being found to be higher (or lower) in those who have survived longer than others, hence the association with time since infection. Conversely, the absence of high (or low) levels of a specific factor in those who have been infected for short periods of time may be biased due to higher mortality rates for those with rapid CD4-cell declines.

Clearly, a factorial analysis of associates with CD4-rates needs to be preceded by a careful evaluation of possible directions of causality, in order to explicate concurrent associations with the time since infection.

The three psychosocial factors found to be associated with CD4-rates of decline were the coping strategies of *focusing upon and venting of emotions* and *mental disengagement*, and the *vigour-activity* POMS scale. Table 26 tabulates the associations between these factors, symptom-status, time since infection, and CD4-rates of decline over time.

TABLE 26

#### CORRELATION MATRIX OF ASSOCIATES OF CD4-RATES OF DECLINE (N = 22)

	CD4 RATE	TIME	SYMPT	AGE	VENT	MDIS	VA
TIME SINCE INFECTION	626a	•			· · · · · · · · · · · · · · · · · · ·		
SYMPTOM-STATUS	+.445 <b>a</b>	369	•				
AGE	+.085	+.088	417	•			
COPE: VENT	433a	+.536@	+.070	137	•		
COPE: MDIS	+.314	173	056	009	+.013	•	
POMS: VA	336	+.354	183	+.013	+.208	396	•
MEAN STD	154.3 101.7	5.795y 2.657y	0.4 <b>09</b> 0.503	34.25 07.14	10.00 2.708	10.96 2.681	17.14 7.344

<sup>&#</sup>x27;a': p < 0.05 (r(crit) = 0.422 for df = 21, p = 0.05, alpha = 5 % (2-tailed))

From table 26 is appears that none of the three psychosocial factors (venting of emotions, mental disengagement, and vigour-activity) are significantly associated with symptom-status (r = +0.070, p > 0.50, df = 21; r = -0.056, p > 0.50, df = 21; r = -0.183, p > 0.40, df = 21; respectively, all 2-tailed, alpha=5 %), nor with age (r = -0.137, p > 0.50; r = -0.009, p > 0.50, df = 21; r = +0.013, p > 0.50, df = 21; respectively, all 2-tailed, alpha=5%).

Both mental disengagement and vigour-activity are not significantly associated with the time since infection (r = +0.314, p < 0.10, df = 21; r = -0.336, p < 0.10, df = 21; respectively, both 2-tailed, alpha = 5%).

However, the COPE scale of focusing upon and venting of emotions is significantly positively correlated with the time since infection (r = +0.536, p < 0.01, alpha = 5 %, 2-tailed). As this factor is not significantly associated with symptom-status nor age, the association with time cannot be reasonably ascribed to maturation or distress due to the presence of symptoms.

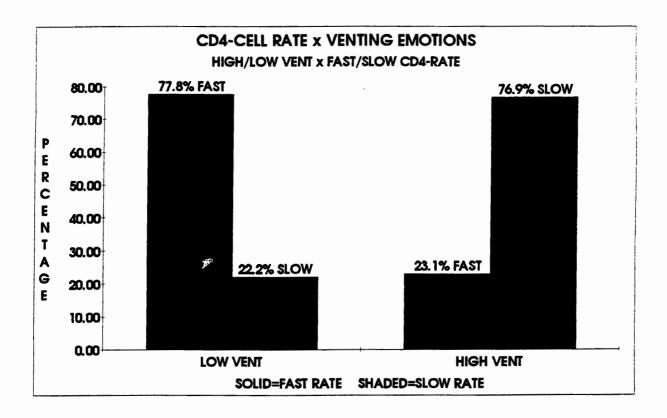
As all subjects who have been infected for less than 18 months have been excluded from these analyses, thus excluding one of the two accelerated periods of infection, only subjects in the more stable asymptomatic period which follows, and in the later stage of infection (AIDS), remain. As the latter is characterized by an acceleration of CD4-cell decline, one would expect that longer periods of infection would be associated with higher rates of CD4-cell decline. This does not appear to be the case, as the association between CD4-cell decline and time since infection is strongly negative (r = -0.626, p < 0.01, df = 21, alpha = 5 %, 2-tailed), indicating that the slower rates of decline are indeed representative of longer-surviving subjects.

A median-split of CD4-rates (Slow group = rates less than 155 CD4-cells lost per year, n = 12, slow decline; Fast group = rates greater than 155 CD4-cells lost per year of infection, n = 10), and time since infection (group 1 = infected for five years or less, n = 11; group 2 = infected for more than five years, n = 11), illustrate this possibility: Of those infected for five years or less, 81.8 percent (n = 9) have 'Fast' CD4-rates, while 90.9 percent (n = 10) of those infected for more than five years have 'Slow' CD4-rates. Furthermore, the difference in mean venting of emotion scores for this two groups is significant (t = 3.082, p < 0.01, df = 17, alpha = 5 %, 2-tailed test), with the 'Fast' group having significantly lower scores on this coping scale (mean = 8.111, std = 2.147, n = 9) compared to the 'Slow' group (mean = 11.4, std = 2.503, n = 12).

All 22 subjects were thus median-split in terms of scores on the *focusing and* venting of emotion scale (median = 10, scores < 10, n = 9; scores => 10, n = 13), and plotted against Slow/Fast CD4-rate categories, as depicted in figure 9, overleaf.

From this figure, it is clear that a slow decline of CD4-cells is strongly associated with high scores on the *focus on and venting of emotions* coping scale, with 77 percent of 'Slow' decliners scoring above the median on this scale, and 78 percent of 'Fast' decliners scoring below the median.

FIGURE 9



Therefore, this coping scale concerning the *venting of emotions* was entered into a multiple stepwise regression model, along with the coping scale of *mental disengagement* and the POMS scale of *vigour-activity*. When all three factors were entered into the model, the model was not significant. However, when the *vigour-activity* factor was deleted from the model, it attained significance at the p < 0.05 level. The results of this final (two-factor) regression model are tabulated in table 27.

TABLE 27

# MULTIFACTORIAL MODEL FOR PREDICTING CD4-RATES OF DECLINE (TWO PREDICTORS)

Multipl	le R <sup>2</sup> =	0.290	Multipi	le R = 0	.538	$R^2(\text{shrunk}) = 0.254$
Beta values:		Coefficients		StDev	T (df = 19)	
Vent	437		- 16.41	8	7.262	- 2.261 p < 0.05
MDis	+.320		+12.12	7	7.335	+1.653 p < 0.15
		Constc	int = 18	5.567		
Source	•	SS		df	MS	F
Regr		62916.3	3	2	31458.	15 3.874 (p < 0.05)
Resid		154284	.4	19	8120.23	31

#### Predictors:

Vent = Focus on and venting of emotions (COPE scale)

MDis = Mental disengagement coping (COPE scale)

The two-factor model of coping by means of *focusing upon and venting of emotions* and *mental disengagement coping*, predicts 29 percent of CD4-rate of decline, with a conservative prediction of 25.4 percent. Furthermore, the standard error of estimate is 87.8, which is lower than the standard deviation of 101.7 for CD4-rate.

Three psychosocial factors - all coping strategies - have emerged as significant predictors of either CD4-cell counts or CD4-rates of decline: **Suppression of competing activities, focusing upon and venting of emotions,** and **mental disengagement.** For descriptive purposes, the associations between these three factors and all other psychosocial factors are tabulated in table 28.

TABLE 28

## PSYCHOSOCIAL ASSOCIATES WITH PREDICTORS OF CD4-CELL COUNT AND CD4-RATE (N = 22)

SUPPR	VENT	MDIS	•
(r)	(r)	(r)	
	· · · · · · · · · · · · · · · · · · ·	CO	PE SCALES
+.573***	+.437*	436* Active coping	
+.566***	+.405	459*	Planning
	+.124	+.051	Suppression of competing activities
+.040	+.166	+.035	Restraint coping
+.523**	+. 197	261	Seeking social support for instrum. reasons
+.765***	+.391	336	* PROBLEM-FOCUSED COPING (A+B+C+D+E)
+.098	+.362	051	Seeking social support for emot. reasons
+. 159	+. 180	268	Positive reinterpretation & growth
143	211	+.066	Turning to religion
+.358	+.391	+.043	Acceptance
+.142	+.475*	068	* EMOTION-FOCUSED COPING (G+H+I+J)
+.124		+.013	* FOCUS ON & VENTING OF EMOTIONS
.580***	322	043	Denial coping
⊦.085	215	+.248	Behavioural disengagement
+.051	+.013		Mental disengagement
.500**	+.000	060	Alcohol & drug disengagement
.498**	226	+.563***	* AVOIDANCE COPING (M+N+O+P)
.441*	035	+. 187 <b>UC</b> L	.A LONELINESS
.296	+.060	+,444* JOY	1
⊦. 137	+.511**	040 HOI	PELESSNESS
		PON	
.317	249	+.298	TA: Tension-Anxiety
.334	354	+.208	DD: Depression-Dejection
.169	+.047	+.170	AH: Anger-Hostility
105	+.208	396	VA: Vigour-Activity
.262	022	+.181	FI: Fatigue-Inertia
.310	305	+.330	CB: Confusion-Bewilderment

r(crit) = 0.535 for df = 21; = T(crit) = 2.831 (2-tailed) for p = 0.01, alpha = 5%

r(crit) = 0.491 for df = 21; = T(crit) = 2.518 (2-tailed) for p = 0.02, alpha = 5%

r(crit) = 0.422 for df = 21; = T(crit) = 2.080 (2-tailed) for p = 0.05, dipha = 5%

From table 28, is appears that none of the three coping strategy predictors are significantly associated with any of POMS scales, indicating no associations with levels of *anxiety*, *depression*, *anger*, *vigour*, *fatigue*, and *confusion*.

The independence of the three coping strategies is also apparent, with few common significantly associated factors. The coping strategies of *suppression* of competing activities and mental disengagement, both of which are negatively associated with CD4-cell counts, appear to be conceptually disparate factors, as the *suppression* of competing activities is significantly positively correlated with active coping (r = +0.573, p < 0.01, df = 21) and planning (r = +0.566, p < 0.01, df = 21), while the mental disengagement coping strategy is significantly negatively associated with both active coping (r = -0.436, p < 0.05, df = 21) and planning (r = -0.459, p < 0.05, df = 21). Furthermore, the suppression of competing activities is significantly negatively associated with the avoidance coping composite score (r = -0.498, p < 0.02, df = 21), while mental disengagement is significantly positively correlated with the same factor (r = +0.563, p < 0.01, df = 21).

The suppression of competing activities coping factor is also significantly positively correlated with the seeking of emotional support for instrumental reasons (r = +0.523, p < 0.02, df = 21) and the problem-focusea coping composite score (r = +0.765, p < 0.01, df = 21), while it is significantly negatively correlated with denial coping (r = -0.580, p < 0.01, df = 21), alcohol-drug disengagement (r = -0.500, p < 0.02, df = 21), and loneliness (r = -0.441, p < 0.05, df = 21). This factor is not significantly associated with any psychosocial factor not mentioned thusfar, including the emotion-focused composite score (r = +0.142, p > 0.05, df = 21).

Apart from the significant associations with active coping, planning, and the avoidance composite score previously mentioned, the **mental disengagement** factor is significantly correlated only with one other factor, namely joy (r = +0.444, p < 0.05, df = 21).

The coping strategy of *focusing upon and venting of emotions*, which is positively associated with CD4-rates of decline, has comparatively few significant psychosocial associates. This coping factor, like the *suppression of competing activities factor*, is significantly positively associated with the *active coping* scale. However, unlike both the *suppression of competing activities* and *mental disengagement*, the *focus upon and venting of emotions* factor is significantly correlated with the *emotion-focused composite score* (r = +0.475, p < 0.05, df = 21). This factor is not significantly associated with either the *problem-focused* and *avoidance coping composite scores* (r = +0.391, p > 0.05; r = -0.226, p > 0.05; df = 21; respectively), nor with the *seeking of social support for instrumental reasons, denial coping, alcohol-drug disengagement, loneliness* or *joy* (r = +0.197, p > 0.05; r = -0.322, p > 0.05; r = 0.000, p > 0.05; r = -0.35, p > 0.05; r = +0.060, p > 0.05; r = 21; respectively). However, the *focusing upon and venting of emotions* is significantly positively correlated with *hopelessness* (r = +0.511, p < 0.02, df = 21).

All three predictor factors were not significantly correlated with restraint coping, / seeking social support for emotional reasons, positive reinterpretation and growth, turning to religion, acceptance, and behavioural disengagement coping.

#### 5.7 SURVEY OF NEEDS

In the survey of needs and attitudes towards group coping programmes, only questions related to awareness of methods of coping with HIV and the desire for information regarding coping methods, are closed-ended. The remaining items are all open-ended, and will thus be summarized thematically. All respondents (N = 31) are included in this survey of needs, although there are only a few items which were completed by all these respondents. Therefore, the frequency of specific responses are relative to the number of subjects who replied to each specific questions.

Of the 28 subjects who responded to the items regarding awareness of coping with HIV by means of methods such as relaxation, visualization and healthy diets, 60.7 percent (n = 17) indicated that they were aware of such coping methods, and also indicated that they would like more information and/or assistance regarding these methods. Another 21.4 percent (n = 6) said that they were not aware of such coping methods, but would like information and/or assistance regarding such methods. Only 17.9 percent (n = 5) indicated that they were aware of such methods, but did not want further information or assistance regarding these coping methods. There were no respondents who said that they were not aware of the various coping methods, and who said that they did not want further information or assistance.

In response to the question regarding which methods of coping the respondent would specifically like to obtain more information about, the most frequent response concerned relaxation techniques, followed by healthy diets, meditation and visualization. Other less frequently-mentioned methods mentioned were aromatherapy, emotional coping methods, methods to assist in spiritual strength, and how to deal with stress.

Two-thirds (66.7 %; n = 16) of those who responded (N = 24) to the questions regarding reservations concerning group programmes specifically designed for coping with HIV, expressed no reservations regarding their willingness to participate in such programmes.

Reasons for being unwilling to participate in such programmes (16.7 %; n = 4) included the fear of revealing HIV-positive status in a group context, and fear of losing employment if HIV-status becomes known. Three respondents (12.5 %) expressed a willingness to participate in such programmes, on condition that confidentiality was ensured. Only one person (4.2 %) stated that he felt that he would not benefit from such a programme.

....

Of the 22 people who responded to the question regarding the type of programme they felt they might benefit from (eg., group support for emotional adjustment or problem-focused methods of coping), 45.5 percent (n = 10) stated a preference for a programme involving group support for assisting in emotional adjustment, while 18.2 percent expressed a preference for a programme concerning problem-focused coping methods. A further 27.3 percent stated that they would like a programme which included both emotional support and problem-focused coping methods. One person stated a preference for a programme concerning coping with health problems, while another said that he would prefer one-to-one counselling rather than participating in a group programme.

The major concerns regarding assistance were financial, such as the cost of treatment, the inability to obtain medical aid coverage for medication, employment discrimination and the fear of losing a job as a result of the employer becoming aware of the respondent's HIV-status, and the inability to find work, life insurance, and the means to pay hospital bills.

There was also concern regarding the refusal of government hospitals to supply medication, as well as the absence of government assistance and programmes for dealing with HIV-related problems.

Several respondents indicated a need for assistance regarding how to deal with their HIV-status within relationships, such as how to let their parents know about it, how to assist HIV-negative sexual partners cope with the knowledge of their partner's HIV-status, and safe-sex practices. Fear of sexual intimacy, as well as the lack of such sexual contact, appear to be of great concern.

Several respondents expressed a desire to speak to someone who is HIV-positive, and who is coping actively. The need for finding other people in the same situation, appears to be prevalent.

Quite a number of respondents expressed appreciation for the information made available by ASET, and several people stated that they got all the information they needed from this agency. It was also mentioned that the drug trials are morale-boosting. The majority of respondents stated that ASET provided them with important services, such as serving as an invaluable first step in dealing with their HIV-status, assisting in self-empowerment, providing access to information about all aspects of the disease, providing advise and assistance regarding medication, job and accommodation placements, keeping track of immune levels, and emotional support and counselling. Regarding the latter, several respondents stated that they experienced great relief by being in an environment with people in the same situation.

Needs for factual information included: A newsletter concerning the latest trends in treatment, drug-trials, alternative treatments, safe-sex practices, and how to live with HIV in a relationship context. There was also a need expressed for explanations of the diverse HIV/AIDS terminology, such as 'AZT' and 'T-cells'.

Emotional anxieties were often related to financial and medical problems, specifically the difficulties of obtaining affordable treatment, and fear of losing employment, and the consequences of this potential loss. Several respondents expressed anxiety concerning the possibility of dying, and the uncertainty and fear of what is going to happen to their body. Loss of direction and purpose, loneliness, lack of sex and intimacy, anger, sadness, and preoccupation with being HIV-seropositive were also mentioned as being causes of anxiety. Interestingly, only one person directly mentioned an existing symptom as a source of anxiety, and it seems as if the anxiety surrounding symptoms and illness are centred around the implications of these symptoms, rather than the symptoms themselves.

### **CHAPTER 6**

### **DISCUSSION OF RESULTS**

#### 6.1 INTRODUCTION

In chapter 5, a number of descriptive and analytic statistics were presented, regarding the nature of the sample, the stability of coping constructs over time, emotional states for different categories of subjects, and associations between psychosocial and immunological measures.

In this chapter, these results will be examined and discussed, in order to draw conclusions regarding the various hypotheses and research objectives previously established. In addition, an evaluation of the methodology employed will be discussed in order to ascertain the validity and reliability of the obtained results, in terms of the criteria proposed by Cook and Campbell (1976) for quasi-experimental field research.

The implications of the findings for psychosocial programmes are discussed, as well as emergent areas requiring clarification in future research.

#### 6.2 METHODOLOGICAL CONSIDERATIONS

In a quasi-experimental study of this nature, there are several important methodological considerations pertaining to reliability and validity that need to be carefully considered before the statistical conclusion validity of the results can be assessed and meaningfully interpreted.

The reliability (test-retest reliability and internal consistency) of the main psychosocial measurements (COPE, POMS, and UCLA Loneliness Scale) have already been elaborated upon in chapter 4, section 4.6, and have been found to be satisfactory. The method in which the immune assays were conducted is also considered to be standard for such measurements, and can thus be considered reliable.

Methodological issues pertaining to the representativeness of the sample, measurement of the time of infection, the temporal gap between the time of psychosocial and immunological measurements, as well as issues regarding the stability of the coping constructs, need to addressed. These issues pertain to establishing causal direction in associations between these psychosocial measurements and the immune levels, and which subsequently affect the validity of any emergent associations.

#### 6.2.1 REPRESENTATIVENESS OF THE SAMPLE

An important methodological consideration concerns the external validity of the study, which is determined by the nature of the sample and the sampling procedures. As random sampling to disease-category or immune levels is not possible, increasing the external validity of the results of the study would need to rely upon a heterogeneous sample in order to maximize representativeness (Cook & Campbell, 1976). To a large extent (as detailed below), this objective was achieved.

The sample is largely English-speaking, white and mixed-race urban homosexual men, half of whom are employed. Ages range from 20 to 51 years, with an average age of 33 years (std = 7.5 years; refer table 7, p.109). The majority of these men (93 %) are high-school graduates, of which a third have tertiary education. It can thus be assumed that the sample is well-educated, and well-informed regarding HIV and AIDS. The latter is concluded from the survey of needs, in which a substantial number of subjects indicated that they receive a great deal of information from ASET regarding the latest trends and developments in treatments related to HIV infection. Thus, in terms of demographic characteristics, the sample appears to be representative of largely urbanized, well-educated homosexual HIV-infected males.

Approximately 60 percent of the sample are asymptomatic (CDC classification II and III), while the remainder are symptomatic, either with mild symptoms (CDC IV = 15%), or with secondary infections (CDC categories IVc1 and IVc2 = 26 %; refer table 8, p.110). There were no subjects with HIV-related neurological symptoms (CDC category IVb), as such potential participants were purposefully excluded from the study. The exclusion of this sub-set of HIV-symptomatology was deemed necessary, as the inclusion of such subjects would make the interpretation of associations between clinical and psychosocial factors difficult, in terms of differentiating between functional cognitive conditions and organic neuropsychiatric conditions.

It is also notable that there were no stage IVd (HIV-related lymphomas such as Kaposi's Sarcoma) subjects in the sample. Enquiry into the absence of such symptoms from the sample revealed that HIV-lymphomas such as Kaposi's Sarcoma are a rarity in the South African (homosexual as well as heterosexual) HIV-infected population.

One possible reason for this situation, is that there is little access to the antiretrovirals and sophisticated treatments which would prolong a person's life beyond the first few bouts of opportunistic infections, such as *pneumocystis carinii*. HIV-related lymphomas usually occur after a lengthy period of infection, and are generally regarded as late-stage disease manifestations, a disease-progression situation which few South African HIV-infected people reach. An alternative reason for the rarity of Kaposi's Sarcoma (KS) in South Africa, is that "...KS is caused by a sexually transmitted cofactor that has remained more prevalent in the original epidemic centers (San Francisco, Los Angeles and New York)..." (Archibald *et al.*, 1992, cited by The AIDS Coalition To Unleash Power (ACT UP), 1994, p.1), as the incidence of KS has been found to be independently associated with residence in these disease epicentres.

The absence of stage IVe subjects (representing symptomatology not easily classified under any other category) is not unexpected either, as this category is not often utilized, and is used for conditions such as concurrent diabetes. There was only one of the original 31 subjects who was classified as CDC IVe. However, this subjects was excluded due to unrelated reasons, namely the absence of a time of infection.

Therefore, in terms of clinical symptom-status, the sample is fairly representative of asymptomatic HIV-infected individuals. However, in terms of symptomatic HIV-infected participants, the sample is restricted only to CDC stages IVa, IVc1 and IVc2.

Immunologically, the sample appears to represent a wide range of CD4-cell levels (range = zero to 1764; refer figure 3, p.112, and table 9, p.113) and CD8-cell levels (range = zero to 4172; refer figure 4, p.112, and table 9, p.113), as well as a wide range of the time that subjects have been infected, varying from less than a year post-infection to more than 12 years post-infection. However, as CD4-cell counts are influenced by antiretroviral and the experimental drug, the validity of the raw CD4-cell counts require further inquiry, as there is an absence of uniform length of usage for these drugs. The validity of immune measures and adjustments for medical drug usage are discussed in greater detail in the following section.

An additional constraint upon the representativeness of symptomatic subjects concerns mortality. By the very nature of the disease, subjects who have rapidly declined and died are less likely to be represented in the sample, although the sample does include a wide range of rates of decline in CD4-cells, which is considered to be the primary disease-progression marker in HIV-infection. The problem of mortality is more apparent when it is considered that disease-progression in HIV-infection is highly diverse, and that symptom-formation and death can occur from as early as within the first year of infection, to more than a decade after infection, with some people apparently remaining healthy for the entire duration of their infection, while others decline either rapidly or more slowly. Therefore, subjects who decline rapidly and die would logically be found exclusively within the shorter times of infection.

This situation could possibly restrict the representativeness of a sample regarding the immunological and clinical spectrum of HIV progression by excluding those subjects with the most rapid immunological decline. Even if such rapidly-declining subjects are represented in the sample, the mortality of those who most rapidly decline would inject a distinct bias in associations between immunological measures and associated psychosocial factors, as the association between the rate of CD4-cell decline and the time infected would confound the possible direction of associative causality between rates of decline and other factors. This issue is discussed in greater detail in section 6.2.2.

Further threats to both external and internal validity concern the fact that an unknown portion of the sample utilize either counselling services, or programmes designed to assist in coping with HIV-infection, such as Body Positive. If such differential access to services have a significant effect upon coping and emotional scores, this will become apparent in comparisons with the levels of such constructs in other similar groups, such as those used as normative groups in chapter 4, namely the samples presented by Blaney et al. (1990) and Goodkin, Blaney et al. (1990).

#### **6.2.2 VALIDITY OF IMMUNE MEASURES**

As mentioned in the previous section, an important methodological concern relates to the reliability of the immune measures, as the study is opportunistic in terms of utilizing existing immunological data obtained for the purposes of the drug-trial in which most subjects were participating at the time that the present study was conducted. Three particular immune-related issues are of importance, namely that CD4-cell counts were adjusted for drug usage, that the psychosocial and immunological measurements were not conducted at the same time, and the issue of mortality on rates of CD4-cell decline.

#### (a) ADJUSTMENT FOR MEDICAL DRUG USAGE

In order to reduce the immunological variance due to medical drugs, each subject's drug usage was individually assessed, and their CD4-cell counts were corrected for such usage. CD8-cell counts are not affected by such medication, and were thus not adjusted. The adjustments to CD4-cell counts were significant for subjects with unadjusted CD4-cell counts above 200 (refer table 10, p.117), but not for those with CD4-cell counts below 200, probably because the major adjustment involved a percentage adjustment for the experimental drug, which becomes negligible for lower cell-count levels

The method used for adjusting CD4-cell counts for antiretroviral drug usage was based upon the findings of the Concorde Trial (MRC, UK, 1993; NIAID, 1993), which is considered to be the most conclusive study to date of the effects of AZT upon CD4-cell counts. Although individuals vary in their immunological response to this antiretroviral drug, thus decreasing the reliability of uniform adjustments for the effect of this drug, the adjustments for this drug are very small, ranging from an increase of 10 cells to a decrease of 20 CD4-cells. When it is considered that these adjustments were conducted for only eight subjects, and represent a mean adjustment of less than 4 CD4-cells for the entire sample (refer appendix 5, table 5-E), it appears unlikely that this adjustment would have any major affect upon the overall immunological profile of the sample.

The adjustment for the experimental drug is more substantial, and affects 63 percent of the sample (refer appendix 5, table 5-E). The mean adjustment for the sample (N=27) was a decrease of 32 CD4-cells per, and the adjustments ranged from 20 percent to zero percent. As the effects of the experimental drug have been closely monitored for each subject, these adjustments may be considered to be fairly reliable. Consequently, such adjustments would markedly increase the reliability of the final adjusted CD4-cell count for each subject, as representing CD4-cell counts which exclude the effects of primary medical drugs. Naturally, the effects of other medication, such as prophylactic drugs and treatments for specific infections, can not be controlled for, thus leaving a degree of variance that can be ascribed to medical drugs.

However, the final adjusted CD4-cell count is the closest approximation of CD4-cell counts under the circumstances. A comparison between adjusted and unadjusted CD4-cell counts (refer figure 5, p.116) indicates that the adjustments made did not significantly affect the ranking of CD4-cell levels, and the correlation between adjusted and unadjusted counts are very high (refer table 10. p.117), indicating a minimal alteration of covariation.

#### (b) TIME BETWEEN IMMUNE AND PSYCHOSOCIAL MEASUREMENT

The temporal difference between the immunological and psychosocial measurements ranged from zero to four months, with a mean difference of 2 months (std = 1.3 months). As the rate of CD4-cell decline was calculated at the time of immune measurement for each subject, and the rate is considered to be fairly linear (Lang *et al.*, 1989), this temporal difference can not be viewed as having any significant effect upon the validity of analyses involving this immune measurement.

However, CD4-cell and CD8-cell counts are affected by time, although changes in these cell counts are generally not marked over such short periods of time. Therefore, such cell-counts may be considered to be fair approximations of cell-counts at the time of psychosocial measurement, although differences in rates of decline, as well as the normal fluctuations in these immune measures, would indicate that an element of caution be used in assuming that such cell-counts are indeed very reliable.

Comparatively speaking, the rate of CD4-cell decline could thus be considered to be a more reliable indication of immune functioning than the CD4-cell and CD8-cell counts.

It should also be mentioned that all analyses involving immunological measures (including CD4-cell and CD8-cell counts) exclude those subjects who have been infected for less that two years, which represents a segment of the infection period which is characterized by high levels of immunological fluctuations, which tend to stabilize about two years post-infection. By excluding these early-infection subjects, the sample size (and thus statistical power) of these analyses are also reduced. However, the error variance due to normal random fluctuations is also substantially reduced, which was considered to outweigh the loss of statical power due to a reduced sample size.

An indication of the reliability of the adjusted CD4-cell counts, and the rate of CD4-cell decline (calculated for the time from reported infection until immunological measurement), is obtained by comparing the data with the norms provided by Lang et al. (1989), for mean CD4-cell counts from infection until the development of AIDS. From this comparison, the obtained rates of CD4-cell decline are consistent with the norms provided by Lang et al. (1989; refer figure 8, p.123). For example, the mean rate of CD4-cell decline of 154 cells per year, with a mean period of infection of 5.8 years (refer figure 8, p.123, and table 11, p.123), appears to be congruent with existing statistical norms (mean CD4-cell rate of decline at six years post-infection = 155 cells per year) obtained from Lang et al. (1989). Thus, from an immunological perspective, the sample appears to be a fairly reliable representation of HIV-infected immune functioning from early infection to AIDS.

#### (c) MORTALITY

In section 6.2.1, the problem of mortality of subjects was mentioned, and the potential bias this would have upon associations between immune measures and other factors. For example, if a specific psychosocial factor is hypothetically causally associated with a slower rate of immunological decline, then this factor may also emerge as being significantly associated with the length of time that the person has been infected, simply due to the fact that those individuals with the lowest (or highest) levels of this psychosocial factor would also be the same people who die before most other HIV-infected people.

Therefore, if a broad spectrum of HIV-infected people are sampled, such rapidly-declining individuals (with, for example, the lowest levels of the hypothetical causal psychosocial factor) would be absent from that segment of the infection-period spectrum which contains median to slow declining individuals (with, for example, the highest levels of the hypothetical causal psychosocial factor). This would have the effect of creating a spurious association between the hypothetical causal psychosocial factor and the time since infection.

Therefore, great care would be required in interpreting emergent associations between rates of CD4-cell decline and psychosocial factors, as a dual association between a specific psychosocial factor and both the rate of immunological decline and the length of infection, would necessitate secondary analyses to eliminate the possibility that levels of the psychosocial factor are causally influenced by factors associated with time, such as age and symptom-formation.

#### 6.2.3 RELIABILITY OF SELF-REPORTED DATA

#### (a) RELIABILITY OF REPORTED PERIOD OF INFECTION

A central issue related to analyses involving the progression of HIV infection over time, including the rate of CD4-cell decline, is the accuracy of self-reported dates or periods in which infection occurred. As discussed in section 5.3.5 (p.118), it appears that the method used to elicit the time of infection was effective in obtaining fairly reliable data, which was verified by consulting clinical and personal history files of each subject. In addition, as has been previously indicated in section 6.2.2 (b), the rates of CD4-cell decline calculated from these reported dates of infection are consistent with norms provided by Lang *et al* (1989).

There is thus little reason to question the reliability or validity of the obtained time of infection.

#### (b) RELIABILITY OF REPORTED SYMPTOM-STATUS

Symptomatology reported by all subjects were verified by consulting medical files. In general, subjects were less accurate regarding symptoms than the time of infection, although the present sample appeared to be more accurate than the sample described by Kessler et al. (1988), who found that the correlation between self-reported symptoms and examination-detected symptoms were very low. In the present study, possible reasons for inconsistencies included exaggeration of minor symptoms and reporting of transient symptoms (despite being requested to indicate only those symptoms present for at least two weeks in the last six months). Although the accuracy of self-reported symptoms (compared to medically-verified symptoms) were not statistically analyzed, there were few cases of failure to report symptoms. Instead, there appeared to be a tendency to over-report symptoms.

#### 6.2.4 STABILITY OF COPING CONSTRUCTS

As stated in sections 4.5 (p.62) and 5.4 (p.131), establishing causal direction can only established in cross-sectional studies if (a) temporal antecedence can be established, and (b) alternative explanations can be plausibly dismissed (Cook & Campbell, 1976). Therefore, in a study of this nature, it is imperative that the presumably stable psychosocial constructs being measured, including coping methods, loneliness, joy and hopelessness, are stable over time (eg., they are independent from the period of infection and the age of subjects), and do not vary with the presence and absence of distressing events such as symptomformation and unemployment. If this can be established, then it would be reasonable to conclude that such psychosocial constructs are relatively stable trait-like characteristics, and that it is plausible that these constructs precede HIV infection in terms of time. On condition that such stability over time is determined, analyses may focus upon the task of assessing alternative explanations for the hypothesis that such psychosocial factors are causally associated with immunological measures.

Table 17 (p.132) summarizes the associations between all coping constructs, loneliness, joy, and hopelessness, with the time from infection until immune measurements, the age of the subject, whether the subjects are employed or not, and the presence or absence of symptoms. From this table, it appears that none of the psychosocial constructs are significantly associated with the time since infection and the age of subjects. The only psychosocial factor exceeding significance at the ten percent level with the time from infection until immune measurement, was the COPE scale of focusing upon and venting of emotions. There were no psychosocial factors which approached significance with the age of respondents at p = 0.10.

Similarly, only one association with *symptom-status* approached significance, namely the COPE scale of *positive reinterpretation and growth*, although it did not attain significance at the five percent level. No other factor approached significance at the ten percent probability level.

Therefore, in terms of the length of time that a person has been infected, the age of the respondent, and symptoms-status, coping methods, loneliness, joy and hopelessness appear to be relatively enduring and stable constructs. This supports the notion that such constructs are trait-like, and do not change despite the passage of time and the development of symptoms, as found by Billings and Moos (1981) and Goodkin, Fuchs *et al.* (1992) regarding coping methods, Rabkin *et al.* (1991) regarding hopelessness, and Rabkin *et al.* (1990) and Longo *et al.* (1988) regarding loneliness.

However, four coping constructs - seeking social support for emotional reasons, denial coping, behavioural disengagement, and the avoidance coping composite score - are significantly correlated with employment status (i.e., whether a person is employed or not). Clearly, unemployment appears to be a significant disrupting factor in habitual coping style, more so than symptom-status.

As employment-status was scored positively (i.e., employed = 1; unemployed = 0), this indicates that *unemployment* is significantly associated with *decreased* coping by means of seeking social support for emotional reasons, and increased coping by means of denial, behavioural disengagement, and general avoidance coping. Of interest, is that the three avoidance coping scores - denial coping, behavioural disengagement, and the avoidance coping composite score - are all highly inter-correlated, with correlations of r = +0.883 and r = +0.867 (both p < 0.001, df = 26; 2-tailed, alpha = 5 %, beta < 1 %) with the avoidance coping composite score, for denial coping and behavioural disengagement respectively.

Unlike the other two active avoidance-related coping scales (mental disengagement and alcohol-drug disengagement, containing items such as "I turn to work or other substitute activities" and "I drink more..."), the denial and behavioural disengagement coping scales reflect cognitive avoidance strategies (eg., "I refuse to believe that it has happened" and "I give up the attempt to get what I want"). The latter may thus be considered as representing a cohesive avoidance construct related to cognitive avoidance which may result in decreased behavioural activity, as opposed to active avoidance (eg., avoidance through engagement in other activities which serve as distractions).

Neither denial coping, behavioural disengagement or the avoidance coping composite scores are significantly correlated with coping by means of seeking social support for emotional reasons (r = -0.266; r = -0.221; r = -0.236; respectively; all p > 0.10; df = 26; 2-tailed, alpha = 5 %, beta > 66 %).

Thus, it appears as if unemployment is associated with increased levels of coping by means of passive cognitive avoidance (and not associated with active coping or active avoidance coping), and that unemployment is also associated with a decreased (active) seeking of emotional social support.

Perhaps of greater interest than the nature of the changes in coping methods associated with unemployment, is that employment status appears to have such effects, while symptom-status does not. This would suggest that the effects of unemployment are experienced as being more profoundly emotionally distressing than the presence of symptoms.

This explanation is supported by the observation noted in the summary of the survey of needs (section 5.7, p.152), namely that the most prevalent issue mentioned as being anxiety-provoking, is the financial implications of being HIV-infected.

For example, there was near unanimity that the cost of medication, inability to obtain medical aid, life insurance, possible loss of employment due to exposure of HIV-status, loss of self-esteem due to unemployment, fear of losing accommodation, and a multitude of other financial issues, are the primary source of anxiety, whether these feared consequences had actually occurred, or whether the person feared that they might occur. It was also noted that only one person mentioned the nature of a specific symptom as being a source of anxiety, while all other symptom-related issues concerned the financial implications of symptomatology.

Thusfar, the focus has been upon those coping constructs which appear to be susceptible to changes over time, age, symptom-status, and employment. Apart from associations with employment-status, there were no significant associations between coping constructs and these factors. Furthermore, all active coping scales and emotion-focusea coping methods (except seeking social support for emotional reasons), loneliness, joy, and hopelessness were unaffected by the time since infection, the age of respondents, and both symptom-status and employment-status. There is also an absence of significant associations between these four factors and the coping methods of mental disengagement and alcohol-drug disengagement. This is consistent with the findings of Blaney et al. (1990), namely that coping styles were not significantly disrupted by event-related stresses in a group of 45 asymptomatic seropositive gay males.

Therefore, these coping methods may be assumed to be stable over time, and associations between these psychosocial constructs and immune measures would merit the consideration of the plausibility of these coping constructs being causally implicated in such associations with immune levels. Naturally, there may be factors which have not been measured, such as bereavement, which may affect such apparently stable psychosocial constructs. However, the cumulative evidence from this study, and previous studies (eg., Martin & Dean, 1993), suggests that this is unlikely, as the effects of bereavement do not appear to persist over time, although bereavement does result in high levels of distress.

#### 6.2.5 GENERAL METHODOLOGICAL CONCLUSIONS

Careful consideration of issues of validity and reliability are essential in quasiexperimental, opportunistic studies of this nature. In addition, the study
incorporates retrospective measurement, specifically related to the time of
infection. The results from such a study cannot be assumed to be as reliable as
those obtained from more controlled research conditions, nor can they assume
to have the clarity regarding causal directions that a prospective study would
have. However, a number of methodological devices have been included in
the present research design in order to take these potentially disadvantageous
methodological issues into account. From the comparison of the obtained
data with similar studies, it appears that the obtained data is consistent with
these studies, indicating that the methodological devices employed were
successful in keeping error variation to an acceptable level, thus strengthening
the reliability and validity of the findings.

In the evaluation of the stability of the various coping constructs, including loneliness, joy and hopelessness, it was found that all these constructs appear stable and trait-like, and unaffected by distressing events such as symptom-status, as well as the age of the respondent and the length of the time that the person has been infected. Furthermore, with a few exceptions, these constructs appears unaffected by the distress of unemployment. It was concluded that analyses of associations between these apparently stable constructs would merit consideration of their possible causal involvement in immune levels and changes over time.

It is with this in mind that we turn to the various hypotheses that were tested.

#### 6.3 PSYCHOSOCIAL DESCRIPTIVE ANALYSES

Two research objectives are discussed in this section: An overview of the psychosocial profile of the sample, including comparisons with HIV-seropositive and HIV-seronegative norm groups, and comparisons between different groupings of subjects regarding levels of emotional distress.

#### 6.3.1 SHORT-TERM MOOD STATES

A comparison of the sample's (POMS) emotional states with college student norms (refer table 12, p.125) reveals that the sample is significantly more *depressed, angry-hostile*, and *fatigued* than college norms, which is congruent with expectations raised by previous studies (eg., Blaney *et al.*, 1990; Atkinson *et al.*, 1988; Ostrow *et al.*, 1989; Catalan, 1988).

Further investigation reveals that these emotional states are not significantly associated with the time since infection, age of the respondents, employment-status, nor symptom-status (refer table 18, p.133). From table 19, it is also apparent that the HIV illness categories of early asymptomatic infection (infected less than two years), asymptomatic infection longer than two years, early symptomatic infection (CDC category IVa), and the presence of life-threatening secondary infections (CDC categories IVc1 and IVc2), did not differ significantly regarding any of the POMS scales.

Clearly, symptom-status does not seem to affect levels of emotional distress, which is contrary to expectations raised by previous studies (eg., Atkinson *et al.*, 1988; Kessler *et al.* 1988). As the sample consists of both asymptomatic (N = 16) and symptomatic subjects (N = 11), these two groups were compared to data obtained from similar groups (Blaney *et al.*, 1990; Atkinson *et al.*, 1988; refer tables 15 and 16, pp.129-130).

These analyses indicate that it is the asymptomatic group which is more distressed than expected, while the symptomatic group does not appear to differ significantly for most of the POMS scales (with the exception of *vigouractivity*) with the comparable data obtained for 13 ARC subjects reported by Blaney *et al.* (1988).

The asymptomatic subjects appear to be more *depressed, angry, fatigued*, and more *confused* than comparable HIV-infected asymptomatic homosexual men in Blaney *et al.* 's USA sample.. As previously indicated, these levels of distress are not associated with symptom-status, employment, age or length of infection. Therefore, the evident distress experienced by asymptomatic subjects - comparable to symptomatic subjects - can not be explained in terms of these clinical and demographic factors. Although there are a multitude of potential factors which may result in such levels of distress, there are few that explain why asymptomatic subjects are more distressed than comparable (USA) asymptomatic HIV-infected homosexual men. It is possible to hypothesize that extraneous factors, such as the social and economic situation experienced by South African HIV-infected asymptomatic subjects may partially explain these high levels of distress.

#### 6.3.2 LONELINESS

Table 13 also reveals that the sample is significantly *lonelier* than the norms for 31 to 40 year olds (Russell, *et al.*, 1982).

This result is inconsistent with the finding of Blaney *et al.* (1990), who found no significant difference between 45 asymptomatic seropositive subjects and the college norms provided by Russell *et al.* (1980), utilizing the full (20-item) Revised UCLA Loneliness Scale. This suggests that the sample is lonelier than comparable HIV-seropositive individuals.

As with the high levels of distress evident in asymptomatic subjects, possible reasons for the difference found between Blaney et al.'s (1990) USA (Miami) sample, and the South African sample, both of which comprise homosexual HIV-seropositive males, may be the absence of a cohesive homosexual subculture in South Africa, a dearth of social, medical, and political services for this group of HIV infected men, and the comparatively higher levels of social stigma associated with HIV-seropositive status, possibly due to much lower levels of media education regarding both homosexuality and HIV. Although the association between these factors and loneliness are purely speculative, it is notable that there is a distinct difference in the social attitudes and awareness of HIV between the two countries, which may result in differential feelings of isolation (and thus loneliness) in homosexual HIV-infected men.

#### 6.3.3 COPING METHODS

Table 14 (p. 127) indicates that the sample scored significantly higher on levels of all avoidance-related coping scales, (acceptance coping, denial, behavioural disengagement, mental disengagement, and alcohol-drug disengagement), and significantly lower than college students on scores for coping by means of seeking of social support for emotional reasons.

The latter results are particularly interesting in the light of the significant associations previously found between several avoidance-related coping scales, as well as the seeking of social support for emotional reasons, with employment-status (refer section 6.2.4). It is notable that unemployment was the only demographic and clinical factor significantly associated with most of these coping methods. Furthermore, the directions of the differences between all the differences found between the sample and the college student norms, are uniformly congruent with the directions of the effects found for unemployment on these coping constructs.

This suggests that unemployment and economic factors may be an important causal factor involved in all these highly significant differences.

#### 6.3.4 OVERVIEW OF THE SAMPLE'S PSYCHOSOCIAL PROFILE

Four main findings are apparent from the descriptive analysis of the psychosocial factors measured: (1) Although the sample appears more distressed than (presumably HIV-seronegative) college student norms, there is an unexpected absence of significant differences between asymptomatic and symptomatic, as well as employed versus unemployed, groups regarding levels of emotional distress; (2) Closer examination reveals that it is the asymptomatic subjects who are more distressed than comparable (USA) asymptomatic HIV-infected groups, while the symptomatic group appears to be largely equally distressed compared to USA ARC subjects; (3) The entire sample is lonelier than HIV-seronegative norms, which is inconsistent with previous studies measuring loneliness in HIV-infected individuals; (4) Those coping methods which were previously found to be affected by employment-status, are also largely the same coping methods that are significantly different to college norms, and these differences are in the same direction as those associated with unemployment.

One possible reason posited for the unexpectedly higher levels of distress of asymptomatic subjects, and loneliness in the total sample, was social and economic factors unique to South Africa, and which detrimentally affect homosexual HIV-infected males. This possible explanation is generally supported by the prevalence of comments in the survey of needs regarding economic and social obstacles, as well as the fact that employment-status, and not symptom-status, appears to be the major life-change event affecting relatively stable psychosocial constructs such as specific coping methods.

However, this does not necessarily explain why symptomatic subjects are not more distressed than their USA comparison group, unless the symptomatic subjects included in this study have access to similar facilities, such as economic and medical assistance.

In this regard, it is worth noting that ASET does indeed provide various such services for symptomatic HIV-infected homosexual men. If the availability of such services does indeed make such a noticeable difference in levels of emotional distress, then this would indicate that such services are inadequate for the asymptomatic HIV-infected men, but not for the symptomatic HIV-infected men.

With respect to the evident differences regarding avoidant coping methods and the seeking of social support for emotional reasons, this appears to be consistent with the effects of unemployment.

in general, the absence of economic resources to the subjects included in this sample appears to be a pervasive source of distress which seems to distinguish this sample from comparable USA HIV-infected homosexual men. According to Tross and Hirsch (1988), the major sources of distress for HIV-infected homosexual men are the loss of a job, eviction, denial of insurance, denial of public services, denial or delay of health-care facilities, life-style changes, medical expenses, adaptation to diagnoses, physical disability, the reality of possible death, and legislation affecting homosexuality and HIV-infected people. Clearly, the issues are common to both the USA and South Africa. However, it appears that many of these issues are exacerbated in the South African context, particularly those related to economic and health-care resources.

#### 6.4 ASSOCIATIONS WITH IMMUNE MEASURES

Two methodological devices have been employed in the testing of hypotheses related to associations between psychosocial factors and the immune functions measured (CD4-cell and CD8-cell counts, and the cumulative rate of CD4-cell decline from infection until the time of immune measurement. These are: (a) the exclusion of subjects (n = 5) who have been infected for less than two years; (b) the inclusion of associations with psychosocial factors which predict ten percent or more of the variance of the immune measures, for the purposes of further investigation.

It should also be noted that, for all analyses involving CD4-cell counts, the adjusted CD4-cell counts are utilized, as these represent levels of CD4-cell after the effects of antiretroviral and experimental drugs have been controlled for.

#### 6.4.1 ASSOCIATIONS WITH CD8-CELL COUNTS

There appears to be a total absence of significant associations between CD8-cell counts and all psychosocial measurements, clinical measures (including time since infection and symptom-status), and demographic data (refer table 20, p. 137).

Furthermore, CD8-cell counts were not significantly associated with adjusted CD4-cell counts (r = +0.411, p < 0.10, df = 20; 2-tailed), nor with the CD4-rate of decline (r = -0.280, p < 0.30, df = 20; 2-tailed).

According to Levy (1993), the antiviral activity of CD8-cells may be an important factor in the control of viral proliferation. However, CD8-cell antiviral activity is not correlated with the number of CD8-cells. Therefore, CD8-cell counts are not significantly associated with disease progression.

In this light, the obtained absence of associations between clinical and immunologic factors with CD8-cell counts is consistent with the expected associations between these factors.

As the absence of associations between CD8-cells and psychosocial fà would apparently shed little light upon possible associations between consignificant associations with CD8-cell counts are not discussed at greater length.

A characteristic of research concerning HIV and AIDS is the rapidity of developments and new findings concerning immunological structures and functions which appear to be central to disease progression. The importance of CD8-cell counts is but one of these immunological parameters which initially appeared to be important in disease progression, but which later proved to be of lesser value, and has thus been superseded by more detailed analyses of immune functions, such as CD8-cell antiviral activity.

Similarly, the importance of CD4:CD8 ratios and percentages of CD4 cells (in terms of total lymphocyte counts), which have been utilized as significant disease-progression indicators in many earlier PNI studies, have also subsequently been superseded by new developments, such as the discovery of the importance of immunological factors such as gamma-globulins, interleukins, and natural killer cell stimulatory factors.

## 6.4.2 CD4-CELL COUNTS & RATES OF DECLINE: ASSOCIATIONS WITH CLINICAL AND DEMOGRAPHIC VARIABLES

Despite ongoing immunological research developments concerning HIV infection, the importance of CD4-cell counts remains as a central marker for disease-progression in HIV infection.

Therefore, a central focus of the present investigation concerns the high variability of CD4-cell counts in HIV-infected individuals, a prominent (and perplexing) characteristic of HIV infection which has been noted by numerous authors (eg., Sheppard *et al.*, 1993; Glasner & Kaslow, 1990; Haney, 1994; Nielsen, 1993).

In the present study, this variability is evidenced by the absence of significant association between the **length of time that a person has been infected** and **CD4-cell counts** (refer table 21, p.139). CD4-cell counts were however significantly associated with **symptom-status**, but not with the **age** of the respondent, nor with **employment status**.

The obtained data is consistent with previous studies which have indicated that CD4-cell counts are highly variable during HIV infection. Furthermore, the significant association between CD4-cell counts and symptom-status, the latter defined simply as the presence or absence of CDC-classified symptoms which have persisted for more than two weeks within the last six months, indicates the value of using CD4-cell counts as indicators of disease progression, as symptom-formation is highly significantly associated with lower CD4-cell levels. It is also apparent that the age of HIV-infected individuals is not associated with CD4-cell levels.

Of particular interest, in the light of the apparent distress associated with unemployment in the sample, is that CD4-cell counts appear to have a very low level of association with whether the person is employed or not, thus obviating the need to investigate whether this distressing factor is potentially causally associated with immune levels.

**CD4-rates of decline** are highly significantly associated with the **time since infection** (refer table 22, p.140), as well as with **CD4-cell counts**. As CD4-cell counts and the time since infection have been previously found to be independent from each other, these highly significant associations with the rate of CD4-cell decline indicates the usefulness of the CD4-rate construct as an indicator of the general disease progression over time, as opposed to the cross-sectional nature of CD4-cell counts.

For example, the usefulness of the CD4-rate construct becomes apparent when it is considered that two individuals with similar CD4-cell counts, but with different periods of infection, would have different rates of CD4-cell decline, and thus different levels of this indicator of disease progression. Thus, associations between psychosocial factors with the (relatively individually linear) CD4-rate of decline would provide greater insight into the effects of chronic emotional states, attitudes and coping methods, as the longitudinal effects would be more apparent than associations with cross-sectional CD4-cell counts.

CD4-rates of decline are also significantly associated with **symptom-status** (refer table 22, p.140). This is probably due to the association of the CD4-rate of decline with CD4-cell counts, and that higher rates of decline would logically result in more rapid depletion of CD4-cell counts, an apparent prerequisite for symptom-formation. The CD4-rate of decline is not significantly correlated with the **age** of the subject, nor with **employment status**, which is consistent with the findings regarding these factors' associations with CD4-cell count levels.

## 6.4.3 CD4-CELL COUNTS AND RATES OF DECLINE: ASSOCIATIONS WITH PSYCHOSOCIAL FACTORS

Several hypothesized associations between the psychosocial constructs and CD4-cell counts and CD4-rates of decline were found to be insignificant (refer tables 20 and 21, pp.139 and 140; for CD4-cell counts and CD4-rate of decline, respectively; all tests were 2-tailed, df = 21).

#### (a) ASSOCIATIONS WITH MOOD STATES

Although the sample has been found to be significantly more distressed than college student norms (refer table 12, p.125; discussed in section 6.3.1), there are no significant associations between the POMS scales of *depression*, *anxiety*, *anger*, *vigour*, *fatigue or confusion*, with respect to CD4-cell counts and CD4-rates of decline (refer tables 21 and 22, pp.139-140). As the POMS scales measure short-term emotional states, which have been found to be unrelated to enduring immunological changes (eg., Knapp *et al.*, 1992; Moss *et al.*, 1989), these results are consistent with expectations, although some studies have found associations between these scales and total lymphocyte counts (Ostrow, 1988).

It is of course possible that some of these emotional states are chronic, particularly as the scores for *anger*, *depression* and *fatigue* appear to be significantly higher for the entire sample. Regardless of the potential chronicity of these emotions, they do not appear to be significantly associated with CD4-cell counts or the rate of CD4-cell decline.

#### (b) LONELINESS, JOY, AND HOPELESSNESS

From tables 21 and 22 (pp.139-140), it is apparent that none of these three factors - *loneliness*, enjoyment of present life-conditions (*joy*), and future-related *hopelessness* - are significantly associated with either CD4-cell counts or CD4-rates of decline.

Several studies have found significant associations between *loneliness* and various immunological functions in non-HIV samples (eg., Kiecolt-Glaser, Garner *et al.*, 1984; Kiecolt-Glaser *et al.*, 1987; 1988; Kennedy *et al.*, 1988), but not in HIV-infected samples (eg., Solano *et al.*, 1992). The present study is consistent with the latter study, as there was no significant association between *loneliness* and CD4-cell counts, as well as with CD4-rates of decline. Although the mean level of *loneliness* has been found to be significantly higher in the sample (refer table 13, p.126), it does not appear as if this factor has any significant association with CD4-related immune functioning.

The finding concerning the association between *hopelessness* and CD4-cell counts is consistent with those of Rabkin *et al.* (1990), who also found no significant association between *hopelessness* and CD4-cell counts. Furthermore, the present study also found no significant association between *hopelessness* and symptom status (r = -0.096), which is also consistent with Rabkin *et al.*'s findings, but contrary to the findings of Ostrow *et al.* (1989), who found significant associations between the severity of symptoms and *hopelessness* and demoralization. However, the latter associations were with *perceived* (as opposed to medically-diagnosed) physical symptoms.

Perry et al. (1992) also found no significant cross-sectional association between hopelessness and CD4-cell counts, but did find a significant association between hopelessness and CD4-cell counts 12 months later. However, the latter association was small (r = -0.220, p < 0.05, df = 87). Therefore, although it is possible that hopelessness may prove to be significantly predictive of CD4-cell variance over lengthy periods of time, the effect size is small, and can be expected to predict less than five percent of CD4-cell counts over time.

Enjoyment of current living conditions (*joy*) also appears to be unrelated to both CD4-cell counts and rates. Interestingly, this construct is also unrelated to symptom and employment status (refer table 17, p. 132). Unfortunately, there are no similar constructs to which *joy* could be compared in order to establish construct validity, and it is consequently not possible to draw conclusions regarding the absence of associations with immunological, symptom-related and employment measures.

#### (c) PROBLEM-FOCUSED COPING

CD4-cell counts and CD4-rates of decline were not significantly associated with all the *problem-focused coping scales*, including *active coping* and the *seeking of social support for instrumental reasons coping* scale. Only one coping factor, the *suppression of competing activities* scale, predicted ten percent or more of the variance of CD4-cell counts. The same factor predicted only eight percent of variance for the CD4-cell rate of decline.

As none of these *problem-focused coping* factors were significantly different from college norms (refer table 14, p.127), nor significantly associated with either the period of time infected, age, employment status, and symptom-status (refer table 17, p.132), there is little evidence to suggest that these coping methods are significantly affected by factors related to HIV infection. There is also little evidence to justify the plausibility of causal associations of these *problem-focused coping* methods with immunological functioning in HIV infected individuals.

These results are consistent with the findings reported by Goodkin, Fuchs *et al.* (1992), who found no significant association between life-change scores and *active coping* styles in a group of 11 asymptomatic HIV-infected homosexual men.

Goodkin, Fuchs *et al.* (1992) also found significant negative associations between life-change scores and CD4-cell counts, as well as a significant positive correlation between *active coping* and CD4-cell counts. However, the present study did not find any significant association between the only non-clinical life-change (i.e., unemployment) and CD4-cell counts, nor that *active coping* was related to CD4-cell counts.

It should be noted that the sample of asymptomatic HIV-infected individuals included in the study by Goodkin, Fuchs *et al.* (1992) had a high mean CD4-cell count (mean = 800, std = 121), which they acknowledge as indicating that the sample consists largely of people who have been very recently infected. As this specific category of HIV-infected individuals have been purposefully excluded from the analyses included in the present study, the results from the two studies are not necessarily comparable, with the present study focusing upon a much wider spectrum of the HIV disease spectrum.

At best, it may be concluded from these two studies that, although active coping may be significantly associated with CD4-cell counts during the first few months after infection, no evidence emerged in the present study that this association persists for the period after early infection. Specifically, there is no evidence to suggest that active coping (or any related COPE problem-focused coping style) has any long-term immunological benefits in HIV-infected individuals.

As the only life-change event measured in the present study was unemployment, no conclusions can be made regarding the association between a more comprehensive life-change inventory and cross-sectional measurements of CD4-cell counts for periods following early infection.

#### (d) EMOTION-FOCUSED COPING

Regarding the *emotion-focused coping scales*, the total *composite score* proved to be not significantly associated with both CD4-cell counts and CD4-rates of decline (refer tables 21 and 22, pp.139-140), including the *seeking of social support for emotional reasons coping* scale, and the *positive reinterpretation and growth* scale.

With the exception of the **seeking of social support for emotional reasons** (refer section 6.3.3), none of these coping constructs were found to be significantly different from college student norms either (refer table 14, p.127), nor significantly associated with either the time since infection, age of subjects, employment status, or symptom-status (refer table 17, p.132).

Of these associations, the most notable absence of associations with CD4-cell counts and the CD4-rate of decline concern the coping constructs of **seeking social support for emotional reasons** and **positive reinterpretation and growth**, both of which were hypothesized to be associated with higher CD4-cell counts and slower rates of CD4-cell decline. Thus, contrary to expectations of such associations raised by various studies (eg., Namir *et al.*, 1989; Thomas *et al.*, 1985; Levy, Herberman, Whiteside *et al.*, 1990; Baron *et al.*, 1990; McNaughton *et al.*, 1990; Glaser *et al.*, 1992; Solomon *et al.*, 1987), such associations did not prove to be significant.

Instead, it seems that levels of both *emotion-focused and instrumental social support* (the latter having previously been shown to have similar absence of associations with CD4-cell counts and rates of decline), have no main effect upon CD4-cell counts and rates of decline over time.

However, it should be noted that interaction effects were not analyzed due to the small sample size, and previous studies have suggested that the effects of social support are more evident under conditions of higher life-change (eg., McNaughton *et al.*, 1990; Thomas *et al.*, 1985), or for subjects who are already immunocompromised (Solano *et al.*, 1993).

The effects of positive attitudes (*positive reinterpretation and growth*) also appears to have little association with CD4-cell counts and rates of decline. This seems to be contrary to expectations that "finding new meaning as a result of the disease" (Solomon et al., 1987, p.649), and "(the belief) that life has become more meaningful as a result of HIV infection" (Nielsen, 1993, p.9; "Longterm survivors", 1993, p.13) is characteristic of long-term survival and thus associated with higher levels of CD4-cells and slower rates of decline.

The issue concerning the effects of 'positive attitudes' on the immune system is highly problematic and controversial, and is typically characterized by ill-defined measurements of this construct. Personal observations and discussions with long-term survivors suggests that realistic appraisals of situations are more prominent attitudinal factors amongst long-term survivors, as opposed to so-called 'positive' reinterpretations and appraisals. It is notable that there is dearth of published research concerning these issues in HIV-related literature.

#### (e) EMOTIONAL REPRESSION AND AVOIDANCE COPING

Although the COPE scale of *focusing upon and venting of emotions* (which concerns the repression of emotions) was not found to be significantly associated with cross-sectional measurements of CD4-cell counts (refer section 5.6.4, p.144), this coping scale was significantly associated with levels of CD4-rates of decline. Subsequent median-split analyses indicated that 77 percent of 'slow' CD4-rates of decline subjects scored above the median on this scale, while 78 percent of those with 'fast' CD4-rates of decline scored below the median on this scale, indicating that the association with this method of coping is closely linked to the progression of time. Furthermore, it was concluded that the most plausible direction of causality is that higher levels of *focusing upon and venting of emotions* (i.e., lower levels of emotional repression) result in slower rates of CD4-cell decline, as there is substantial theoretical and empirical evidence to support this possibility (eg., Pennebaker & Beall, 1986; Pennebaker *et al.*, 1987; 1988; Pennebaker & Susman, 1988; Weinberger *et al.*, 1979; Esterling *et al.*, 1990; Solano *et al.*, 1993).

In the latter study by Solano et al. (1993) of 100 HIV-seropositive asymptomatic homosexual males, only denial/repression attitudes negatively affected the clinical evolution of CD4-cell counts over 12 months. In additional, this effect was not significantly evident at six months after initial psychosocial measurement, which supports the previously suggested notion that the effects of emotional repression become more evident over longer periods of time.

It is interesting to note that the only other factor besides emotional repression which emerged as a significant predictor of the clinical evolution of CD4-cell counts in the study by Solano et al. (1993), was the coping construct of denial. Although the present study did not find significant associations between CD4-cell counts, CD4-rates of decline, and the coping methods of denial, behavioural disengagement, alcohol-drug disengagement and the avoidance coping composite score (refer table 21 and 22, pp. 139-140), the coping method of mental disengagement did emerge as a significant correlate of CD4-cell counts.

Although **mental disengagement** coping was not significantly correlated with CD4-rates of decline, it did predict ten percent ( $r^2 = 9.9$  %) of the variance of CD4-rates of decline. Of all the coping constructs, only the two COPE scales of **focusing upon and venting of emotions** and **mental disengagement** predicted ten percent or more of CD4-cell rates of decline.

From table 28 (refer p.149), the *focusing upon and venting of emotions* construct is positively associated with *emotion-focused coping*, but not with either *problem-focused* or *avoidance coping*. In contrast, *mental disengagement* is not significantly associated with either *problem-focused* or *emotion-focused coping*, and is instead positively associated with *avoidance coping*.

From appendix 2 (table 2-C), it is apparent that higher scores for the *focusing* upon and venting of emotions coping construct are significantly positively associated with Type A personality, anxiety, and monitoring (information seeking), and negatively correlated with social desirability, control, internal locus of control, and optimism. Regarding optimism, it is interesting to note that the scores for this coping construct and hopelessness were significantly negatively correlated in the sample. In general, the construct appears to strongly resemble the repressor-sensitizer interpersonal style construct, with low levels of this coping method resembling the repressor style, with associated higher levels of social desirability and lower levels of reported anxiety.

Weinberger et al., 1979 (pp.369-370) describe repressors as having "heightened recognition thresholds for anxiety provoking stimuli ... (and) this style also correlates on projective measures of maladjustment and with inaccurate self-descriptions ... (Their) defensiveness and preoccupation with avoiding awareness of anxiety may interfere with effective coping and, paradoxically, promote behavioral and physiological responses indicative of high anxiety" (emphasis mine). It appears that a critical component of this psychological construct is that repressors' mental defences are of such a nature that such people are simply not usually aware of the emotional distress evident within their bodies. This emotional repression has been found to be associated independent from alcohol and drug usage, physical activity, sleep loss, medication usage, body mass, and calories consumed - with poorer control of latent Epstein-Barr virus, as well as with higher levels of viral antigen titres (Esterling et al., 1990).

From appendix 2 (table 2-C), it appears that higher scores for coping by means of *mental disengagement* are also associated with lower levels of optimism and control, as well as higher levels of anxiety. In these aspects, this method of coping is similar to the *focusing upon and venting of emotions* construct. However, it is not associated with Type A personality, monitoring, or social desirability. According to Suls and Fletcher (1985), *avoidant coping* has been found to be associated with lower levels of distress in the short-term, but is less efficacious in the long-term.

In the present study, it is interesting to note that the *mental disengagement* coping construct is significantly positively correlated with present-time pleasure (*joy*; refer table 28, p.149), and negatively correlated with the long-term immunological parameter in HIV-infection, namely CD4-rates of decline.

When these two coping factors were entered into a multifactorial analysis, the model emerged as being significant, and predicted 29 percent of the CD4-rate of decline (refer table 27, p.148). The standard error of the estimate was smaller than the standard deviation, indicating that the model is a more accurate predictor of CD4-rates of decline than knowledge of the mean and standard deviation of CD4-rates of decline. The two COPE scales are independent of each other (r = +0.013, p > 0.50; refer table 26, p.145), which further strengthens the stability of the multifactorial model.

Although the FOMS scale of *vigour-activity* predicted 15.6 percent of the variance of CD4-rates of decline, when this factor was entered into the factorial model, the model no longer attained significance at the five percent level, and this factor was thus deleted from the final model.

Only three psychosocial factors predicted ten percent or more of the cross-sectional CD4-cell counts, namely the COPE scales of *suppression of competing activities* and *mental disengagement*, and the POMS scale of *vigour-activity*. From table 28 (p.149) it is apparent that these three coping constructs are independent from each other.

From table 28 (refer p.149), it is apparent that the *suppression of competing activities* is highly positively associated with *problem-focused coping* and negatively associated with *avoidance coping*. It is not significantly associated with *emotion-focused coping*. From appendix 2 (table 2-C), it appears that this construct is positively correlated with Type A personality. It is theoretically unclear why higher scores for this method of coping are associated with lower levels of CD4-cell counts. However, an examination of the items contained in this scale (refer appendix 2, table 2-A) would suggest that this method of coping involves a form of preoccupation with the distressing event or situation, and thus implies avoidance of other activities and situations.

When these three factors were entered into a multifactorial regression model for predicting CD4-cell counts, the three-factor model emerged as being significant (F = 4.042, p < 0.05, refer table 24, p.142), and predicted 40.2 percent of CD4-cell count variance.

When the *vigour-activity* scale was deleted from the model, the model remained significant (F = 4.737, p < 0.05, refer table 25, p.143), and lost seven percent of it's predictive power ( $R^2 = 33.3$  percent). As with the CD4-rate of decline, the *vigour-activity* factor was associated with immunoenhancement, while the *suppression of competing activities* and *mental disengagement* coping methods were associated with immunosuppression.

#### 6.4.4 GENERAL CONCLUSIONS REGARDING CAUSALITY

It is notable than none of the factors entered into the multifactorial models for predicting either CD4-cell counts or CD4-rates of decline were significantly associated with the age of respondents, the time since infection, symptom status, or employment status (refer tables 17 and 18, pp. 132-133). Furthermore, only the scores for the coping scale of *mental disengagement* was significantly higher than college norms (refer table 14, p.127). Scores for *vigour-activity*, and coping by means of *suppression of competing activities* and the *focusing upon and venting of emotions* did not differ significantly from college norms (refer tables 12 and 14, pp.125 and 127), and is can be tentatively assumed that these factors (excepting the short-term POMS *vigour-activity* emotion scale) are relatively stable and trait-like, thus indicating that they are causally implicated in CD4-cell count and CD4-rate levels, as there is little evidence to support the alternative notion that changes in these immune levels cause changes in these psychosocial factors.

However, the comparatively higher levels of coping by means of **mental** disengagement in the sample prevents the formation of even tentative conclusions regarding whether this factor is causally associated with CD4-cell levels.

It is unclear whether higher levels for this measurement are due to a tendency to mentally disengage after notification of HIV-seropositive status, or whether such higher levels exist prior to such notification amongst homosexual males in South Africa. As the sample has been found to differ from American homosexual HIV-infected males in scores on this scale, a normative sample of South African seronegative homosexual males would be necessary to establish whether this difference is due to a general reaction to notification of being HIV-seropositive or not.

The establishment of causality in a cross-sectional study utilizing retrospective measurements of certain factors is problematic, and can not expect to attain the levels of certainty that a prospective design would provide.

However, there are several compelling reasons to provide support for the methodology employed in the present study, including the fact that the results appear largely consistent with all three prospective studies of HIV-infected individuals reviewed, namely Perry et al. (1992), Rabkin et al. (1991), and Solano et al. (1993).

Specifically, Perry et al. (1992) and Rabkin et al. (1991) found little evidence for the ability of a variety of emotional states to predict CD4-cell counts 12 months later, including state anxiety, depression, hopelessness, social support, and life events. All these results seem to be replicated in the present study.

Furthermore, Solano *et al.* (1993) found that loneliness, social support, psychological distress, and the time since diagnosis, were not significant predictors of CD4-cell levels, while coping methods of denial and emotional repression did indeed predict CD4-cell counts 12 months later. Once again, the present study appears to have replicated these findings.

Finally, it is significant to note that these convergent results were obtained utilizing different psychosocial measuring instruments, which suggests that the obtained effects are robust and consistent.

#### 6.4.5 SUGGESTIONS FOR FUTURE RESEARCH

From the results of this study and the review of prior studies, the cumulative evidence tends to support the conclusion that the amount of life changes and emotional distress associated with events are not the crucial factors associated with enduring changes in immunological functioning. Instead, it seems that the generalized or habitual method of responding to distressing events and chronic conditions are associated with immunological levels in HIV-infected individuals. There is also substantial evidence to support the notion that these trait-like psychosocial factors may be causally implicated in alterations of CD4-cell levels and rates of decline over time.

Generally speaking, the evidence tends to support Sub and Fletchers's (1985) conclusion that, whereas avoidant coping results in short-term reduction of emotional distress, this method of coping may have detrimental long-term consequences. Furthermore, although emotion-focused coping (specifically, the venting of emotions) may be more emotionally distressing in the short-term, such coping has positive emotional and physiological long-term benefits.

However, there are several unresolved questions regarding these apparently stable coping methods. These issues primarily concern the lack of more detailed and defined instruments for measuring the various aspects of these constructs. For example, it is unclear whether the association between CD4-rates of decline and the repression of emotion is due to sensory or emotion-focused repression. These issue need to be investigated and clarified before psychosocial programmes can begin to implement and test the efficacy of specific coping skills.

For example, would such a programme require the acquisition of skills related to the vocalization of opinions and emotions, or should there be greater emphasis upon increased awareness of the sensory aspects of emotions and a decrease in the maintenance of mental defences against such sensations? In other words, would such a programme need to focus upon teaching forms of expression of emotions, or would it emphasize increased conscious awareness of emotions and sensations?

The latter approach may prove to be more efficacious, when it is considered that coping by means of mental disengagement and denial appears to be have long-term immunosuppressive consequences. However, due to the evident short-term emotional benefits of avoidance coping, a programme designed to enhance immunological functioning in HIV-infected individuals would need to address the need to consider long-term consequences.

In a similar vein, there is insufficient research regarding the effects of attitudes upon immune functioning in HIV-infected individuals, which results in confusion regarding the benefits of ill-defined constructs such as 'positive thinking' and hopelessness. At present, the reviewed studies utilize a limited range of items to measure such constructs, resulting in ambiguous results.

Greater attention to developments regarding the immunological aspects of HIV disease progression is essential. However, this may prove to be a difficult task, as there is such a rapid pace evident in medical research regarding the nature of the interaction between the HIV and the immune system. Current trends seem to suggest that the emphasis upon structural immune measures, such as cell counts, is inadequate for the purposes of understanding the links between psychosocial factors and HIV disease progression, and that more attention is needed in the area of cellular activity levels and the chemical mediators (eg., Interleukin 2, 10, and 12, and gammaglobulins) involved in cell-mediated immune processes.

There is an evident tendency for associations between psychosocial factors and immune measures to emerge over relatively long periods of time, which suggests that prospective studies extending over periods of more than 12 months are required in order to gain clarity regarding psycho-immunological associations. However, HIV has reached epidemic proportions worldwide, which adds a tremendous sense of urgency for acquiring knowledge of such associations. Therefore, PNI research may benefit from the development of research methodologies that can provide data pertaining to longitudinal associations without resorting to time-consuming prospective studies. The use of the CD4-rate of decline appears to provide such data, and efforts should be made to strengthen the reliability of this measurement.

#### REFERENCES

Ader, R. (1981). Psychoneuroimmunology. Orlando, Fl.: Academic Press.

Ader, R., & Cohen, N. (1975). Behaviorally conditioned immunosuppression. Psychosomatic Medicine, 37(4), 333-340.

Ader, R., Felten, D., & Cohen, N. (1990). Interactions between the brain and the immune system. <u>Annual Review of Pharmacology and Toxicology</u>, <u>30</u>, 561-602.

Ader, R., Felten, D.L., & Cohen, N. (Eds.). (1991). <u>Psychoneuroimmunology</u> (2nd Edit.). San Diego, Ca.: Academic Press.

Aggleton, P., Homans, H., Mojsa, J., Watson, S., & Watney, S. (1989). <u>AIDS: Scientific and social issues</u>. New York: Churchill Livingstone.

Aids top killer of young US men. (1993). Argus, October 29.

Antoni, M.H., August, S., LaPerriere, A., Baggett, H.L., Klimas, N., Ironson, G., Schneiderman, N., & Fletcher, M.A. (1990). Psychological and neuroendocrine measures related to functional immune changes in anticipation of HIV-1 serostatus notification. <u>Psychosomatic Medicine</u>, <u>52</u>, 496-510.

Antoni, M.H., Baggett, L., Ironson, G., LaPerriere, A., August, S., Klimas, N., Schneiderman, N., & Fletcher, M.A. (1991). Cognitive-behavioral stress management intervention buffers distress responses and immunologic changes following notification of HIV-1 seropositivity. <u>Journal of Consulting and Clinical Psychology</u>, 59(6), 906-915.

Antoni, M.H., Schneiderman, N., Fletcher, M.A., Goldstein, D.A., Ironson, G., & LaPerriere, A. (1990). Psychoneuroimmunology and HIV-1. <u>Journal of Consulting and Clinical Psychology</u>, <u>58</u>(1), 38-49.

Arnetz, B.B., Wasserman, J., Petrini, B., Brenner, S.O., Levi, L., Eneroth, P., Salovaara, H., Hjelm, R., Salovaara, L., Theorell, T., & Petterson, I.L. (1987). Immune function in unemployed women. <u>Psychosomatic Medicine</u>, <u>49</u>(1), 3-12.

Aspinwall, L.G., Kemeny, M.E., Taylor, S.E., Schneider, S.G., & Dudley, J.P. (1991). Psychosocial predictors of gay men's AIDS risk-reduction behavior. <u>Health Psychology</u>, 10(6), 432-444.

Atkinson, J.H., Grant, I., Kennedy, C.J., Richman, D.D., Spector, S.A., & McCutchan, A. (1988). Prevalence of psychiatric disorders among men infected with Human Immunodeficiency Virus. <u>Archives of General Psychiatry</u>, <u>45</u>, 859-864.

Baker, G.H.B. (1987). Psychological factors and immunity. <u>Journal of Psychosomatic Research</u>, <u>31</u>(1), 1-10.

Ballieux, R.E. (1992). Bidirectional communication between the brain and the immune system. <u>European Journal of Clinical Investigation</u>, 22(S1), 6-9.

Baron, R.S., Cutrona, C.E., Hicklin, D., Russell, D.W., & Lubaroff, D.M. (1990). Social support and immune function among spouses of cancer patients. Journal of Personality and Social Psychology, 59(2), 344-353.

Bartrop, R.W., Luckhurst, E., Lazarus, L., & Kiloh, L.G. (1977). Depressed lymphocyte function after bereavement. <u>Lancet</u>, <u>1</u>, 834-836.

Batchelor, W.F. (1988). AIDS 1988: The science and limits of science. <u>American Psychologist</u>, <u>43</u>(11), 853-858.

Baum, A., Davidson, L.M., Singer, J.E., & Street, S.W. (1987). Stress as a psychophysiological process. In A. Baum & J.E. Singer (Eds.), <u>Handbook of psychology and health</u> (pp.1-24). Hillsdale, NJ: Lawrence Erlbaum Associates.

Baum, A., Fleming, R., Singer, J.E. (1983). Coping with victimization by technological disaster. <u>Journal of Social Issues</u>, 39, 117-138.

Baum, A., & Nesselhof, S.E.A (1988). Psychological research and the prevention, etiology, and treatment of AIDS. <u>American Psychologist</u>, <u>43</u>(11), 900-906.

Beck, A.T., Ward, C.H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. <u>Archives of General Psychiatry</u>, 4, 561-571.

Beck, A.T., Weissman, A., Lester, D., & Trexler, L. (1974). The measurement of pessimism: The Hopelessness Scale. <u>Journal of Consulting and Clinical Psychology</u>, 42(6), 861-865.

Beecher, H.K. (1955). The powerful placebo. <u>J.A.M.A.</u>, <u>159</u>(17), 1603-1606.

Beecher, H.K. (1961). Surgery as placebo. <u>J.A.M.A.</u>, <u>176</u>(13), 1103-1107.

Beutler, L.E., Engle, D., Oro'-Beutler, M.E., Daldrup, R., & Meredith, K. (1986). Inability to express intense affect: A common link between depression and pain? <u>Journal of Consulting and Clinical Psychology</u>, <u>54</u>(6), 752-759.

Billings, A.G., & Moos, R.H. (1981). The role of coping responses and social resources in attentuating the stress of life events. <u>Journal of Behavioral Medicine</u>, 4(2), 139-157.

Blaney, N.T., Millon, C., Morgan, R., Eisdorfer, C., & Szapocznik, J. (1990). Emotional distress, stress-related disruption and coping among healthy HIV-positive gay males. <u>Psychology and Health</u>, <u>4</u>, 259-273.

Bolen, J.S. (1985, March/April). William Calderon: Incredible triumph over AIDS brings new hope. New Realities, pp.8-15.

Boo, K. (1991, June). What Mother Teresa could learn in a leather bar. <u>The Washington Monthly</u>, pp.34-40.

Borysenko, M. (1987). Area review: Psychoneuroimmunology. <u>Annals of Behavioral Medicine</u>, 9(2), 3-10.

Braud, W. (1986). PSI and PNI: Exploring the interface between parapsychology and psychoneurology. <u>Parapsychology Review</u>, <u>17</u>(4), 1-5.

Buske-Kirschbaum, A., Kirschbaum, C., Stierle, H., Lehnert, H., & Hellhammer, D. (1992). Conditioned increase of natural killer cell activity (NKCA) in humans. <u>Psychosomatic Medicine</u>, <u>54</u>, 123-132.

Calabrese, J.R., Kling, M.A., & Gold, P.W. (1987). Alterations in immunocompetence during stress, bereavement, and depression: Focus on neuroendocrine regulation. <u>American Journal of Psychiatry</u>, <u>144</u>(9), 1123-1134.

Camara, E.G., & Danao, T.C. (1989). The brain and the immune system: A psychosomatic network. <u>Psychosomatics</u>, <u>30(2)</u>, 140-146.

Carver, C.S., Scheier, M.F., & Weintraub, J.K. (1989). Assessing coping strategies: A theoretically based approach. <u>Journal of Personality and Social Psychology</u>, <u>56</u>(2), 267-283.

Catalan, J. (1988). Psychosocial and neuropsychiatric aspects of HIV infection: Review of their extent and implications for psychiatry. <u>Journal of Psychosomatic Research</u>, <u>32</u>(3), 237-248.

Centers for Disease Control (1987). Revision of the CDC surveillance case definition for acquired immune deficiency syndrome. <u>Mortality and Morbidity Weekly Report</u>, <u>36</u> (Suppl. 1s). Atlanta, Ga.: U.S. Department of Health and Human Services, Public Health Service.

- Cioffi, D., & Holloway, J. (1993). Delayed costs of suppressed pain. <u>Journal of Personality and Social Psychology</u>, <u>64</u>(2), 274-282.
- Clerici, M., & Shearer, G.M. (1993). A Th1 to Th2 switch is a critical step in the etiology of HIV infection. <u>Immunology Today</u>, <u>14</u>, 107-111.
- Coates, T.J., McKusick, L., Kuno, R., & Stites, D.P. (1989). Stress reduction changed number of sexual partners but not immune function in men with HIV. <u>American Journal of Public Health</u>, <u>79</u>(7), 885-887.
- Cohan, G.R. (1994, February 8). AIDS Line: Can I stimulate my immune system? The Advocate, pp.33-34.
- Cohen, F. (1987). Measurement of coping. In S.V. Kasl and C.L. Cooper (Eds.), <u>Stress and health: Issues in research methodology</u>, pp.283-305. Chichester: John Wiley & Sons.
- Cohen, J. (1969). <u>Statistical power analysis for the behavioral sciences</u>. New York: Academic Press.
- Cohen, S.I. (1988). Voodoo death, the stress response, and AIDS. In T.P. Bridge, A.F. Mirsky, & F.K. Goodwin (Eds.), <u>Psychological</u>, <u>Neuropsychiatric</u>, and <u>substance abuse aspects of AIDS</u>, pp.95-109. New York: Raven Press.
- Cohen, S., Tyrrell, D.A.J., & Smith, A.P. (1993). Negative life events, perceived stress, negative affect, and susceptibility to the common cold. <u>Journal of Personality and Social Psychology</u>, <u>64</u>(1), 131-140.
- Contrada, R.J., & Krantz, D.S. (1987). Measurement bias in Health Psychology research designs. In S.V. Kasl and C.L. Cooper (Eds.), <u>Stress and health: Issues in research methodology</u>, pp.57-78.
- Cook, T.D., & Campbell, D.T. (1976). The design and conduct of quasi-experiments and true experiments in field settings. In M.D. Dunnette (Ed.), Handbook of industrial and organizational psychology, pp.223-326. Chicago: Rand McNally College Publishing.
- Coxon, A. (1990). Coping with the threat of death. In B. Almond (Ed.), <u>AIDS A moral issue</u>, chap. 10. New York: St. Martin's Press.
- Daruna, J.H. & Morgan, J.E. (1990). Psychosocial effects on immune function: Neuroendocrine pathways. <u>Psychosomatics</u>, <u>31</u>(1), 4-12.

Davidson, S., Dew, M.A., Penkower, L., Becker, J.T., Kingsley, L., & Sullivan, P.F. (1992). Substance use and sexual behavior among homosexual men at risk for HIV infection: Psychosocial moderators. <u>Psychology and Health</u>, <u>7</u>, 259-272.

Denney, D.R., Stephenson, L.A., Penick, E.C., & Weller, R.A. (1988). Lymphocyte subclasses and depression. <u>Journal of Abnormal Psychology</u>, <u>97</u>(4), 499-502.

Derogatis, L.R. & Melisaratos, N. (1983). The Brief Symptom Inventory: an introductory report. <u>Psychological Medicine</u>, <u>13</u>, 595-605.

Dillon, K.M., Minchoff, B., & Baker, K.H. (1985). Positive emotional states and enhancement of the immune system. <u>International Journal of Psychiatry in Medicine</u>, 15(1), 13-18.

Dilley, J.W., Pies, C., & Helquist, M. (Eds.). (1989). <u>Face to Face: A guide to AIDS cour.seling</u>. San Francisco, Ca.: AIDS Health Project, University of California.

Dowling, S.J. (1984). Lourdes cures and their medical assessment. <u>Journal of the Royal Society of Medicine</u>, <u>77</u>, 634-638.

Elliot, D.L., Goldberg, L., & Coodley, G.O. (1992). Comment on: Effects of exercise training on men seropositive for the human immunodeficiency virus-1. Medicine and Science in sports and exercise, 24(7), 838.

Esterling, B.A., Antoni, M.H., Kumar, M., & Schneiderman, N. (1990). Emotional repression, stress disclosure responses, and Epstein-Barr viral capsid antigen titers. <u>Psychosomatic Medicine</u>, <u>52</u>, 397-410.

Esterling, B.A., Antoni, M.H., Schneiderman, N., Carver, C.S., LaPerriere, A., Ironson, G., Klimas, N.G., & Fletcher, M.A. (1992). Psychosocial modulation of antibody to Epstein-Barr viral capsid antigen and human Herpesvirus type-6 in HIV-1 infected and at-risk gay men. <u>Psychosomatic Medicine</u>, <u>54</u>, 354-371.

Evans, D.L., Folds, J.D., Petitto, J.M., Golden, R.N., Pedersen, C.A., Corrigan, M., Gilmore, J.H., Silva, S.G., Quade, D., & Ozer, H. (1992). Circulating natural killer cell phenotypes in men and women with major depression. <u>Archives of General Psychiatry</u>, 49, 389-395.

Folkman, S., Chesney, M.A., Pollack, L., & Phillips, C. (1992). Stress, coping, and high-risk sexual behavior. <u>Health Psychology</u>, <u>11</u>(4), 218-222.

Geiser, D.S. (1989). Psychosocial influences on human immunity. <u>Clinical Psychology Review</u>, 9(6), 689-715.

- Gilder, S., & Hodgkin, K. (Eds.). (1987). <u>South African Family Medical Adviser</u> (3rd Edit.). Cape Town: Reader's Digest Association of South Africa.
- Glaser, R., Kiecolt-Glaser, J.K., Bonneau, R.H., Malarkey, W., Kennedy, S., & Hughes, J. (1992). Stress-induced modulation of the immune response to recombinant Hepatitis B vaccine. <u>Psychosomatic Medicine</u>, <u>54</u>, 22-29.
- Glaser, R., Kiecolt-Glaser, J.K., Speicher, C.E., & Holliday, J.E. (1985). Stress, loneliness, and changes in Herpesvirus latency. <u>Journal of Behavioral Medicine</u>, <u>8</u>(3), 249-260.
- Glaser, R., Kiecolt-Glaser, J.K., Stout, J.C., Tarr, K.L., Speicher, C.E., & Holliday, J.E. (1985). Stress-related impairments in cellular immunity. <u>Psychiatry Research</u>, 16, 233-239.
- Glasor, R., Rice, J., Speicher, C.E., Stout, J.C., & Kiecott-Glaser, J.K. (1986). Stress depresses interferon production by leucocytes concomitant with a decrease in natural killer cell activity. <u>Behavioral Neuroscience</u>, <u>100</u>(5), 675-678.
- Glasner, P.D., & Kaslow, R.A. (1990). The epidemiology of Human Immunodeficiency Virus infection. <u>Journal of Consulting and Clinical Psychology</u>, <u>58</u>(1), 13-21.
- Goodkin, K., Blaney, N.T., Feaster, D., Fletcher, M.A., Baum, M.K., Mantero-Atienza, E., Klimas, N.G., Millon, C., Szapocznik, J., & Eisdorfer, C. (1992). Active coping style is associated with natural killer cell cytotoxicity in asymptomatic HIV-1 seropositive homosexual men. <u>Journal of Psychosomatic Research</u>, <u>36</u>(7), 635-650.
- Goodkin, K., Fuchs, I., Feaster, D., Leeka, J., Rishel, D.D. (1992). Life stressors and coping style are associated with immune measures in HIV-1 infection A preliminary report. <u>International Journal of Psychiatry in Medicine</u>, <u>22</u>(2), 155-172.
- Green, M.L., Green, R.G., & Santoro, W. (1988). Daily relaxation modifies serum and salivary immunoglobulins and psychophysiologic symptom severity. <u>Biofeedback and Self-Regulation</u>, <u>13</u>(3), 187-199.
- Guccione, B. (1994). The deadly sex scam. <u>Big Blue</u>, <u>4</u>(1), pp.9,12,13,58,59,72.
- Hall, N.R.S. (1988). The virology of AIDS. American Psychologist, 43(11), 907-913.
- Hall, N.R.S., & O'Grady, M.P. (1991). Psychosocial interventions and immune function. In R. Ader, D.L. Felten, & N. Cohen (Eds.). <u>Psychoneuroimmunology</u> (2nd Edit.), (pp. 1067-1080). San Diego, Ca.: Academic Press.

Halley, F.M. (1991). Self-regulation of the immune system through biobehavioral strategies. <u>Biofeedback and Self-Regulation</u>, <u>16</u>(1), 55-74.

Haney, D.Q. (1994, January 30). Long-term survivors may carry answers to AIDS virus. The Press Democrat, B6.

Hassan, N.F., & Douglas, S.D. (1990). Stress-related neuroimmunomodulation of monocyte-macrophage functions in HIV-1 infection. <u>Clinical Immunology and Immunopathology</u>, <u>54</u>, 220-227.

Herek, G.M., & Glunt, E.K. (1988). An epidemic of stigma: Public reactions to AIDS. <u>American Psychologist</u>, <u>43</u>(11), 886-891.

Horowitz, M., Wilner, N., & Alvarez, W. (1979). Impact of Event Scale: A measure of subjective stress. <u>Psychosomatic Medicine</u>, <u>41</u>(3), 209-218.

House, J.S., Landis, K.R., & Umberson, D. (1988). Social relationships and health. Science, 241, 540-545.

Howell, D.C. (1989). <u>Fundamental statistics for the behavioral sciences</u> (2nd Edit.). Boston: PWS-Kent.

Huber, J., & Schneider, B.E. (Eds.). (1992). <u>The social context of AIDS</u>. Newbury Park, Ca.: Sage Publications.

IL-12: Potential treatment restores immune response in laboratory test. (1993, December 30). <u>San Francisco Bay Times</u>, pp. 15, 17.

Ironson, G., LaPerriere, A., Antoni, M., O'Hearn, P., Schneiderman, N., Klimas, N., & Fletcher, M.A. (1990). Changes in immune and psychological measures as a function of anticipation and reaction to news of HIV-1 antibody status. <u>Psychosomatic Medicine</u>, <u>52</u>, 247-270.

Irwin, M., Daniels, M., Bloom, E.T., Smith, T.L., & Weiner, H. (1987). Life events, depressive symptoms, and immune function. <u>American Journal of Psychiatry</u>, 144(4), 437-441.

Irwin, M., Daniels, M., Risch, S.C., Bloom, E., & Weiner, H. (1988). Plasma cortisol and natural killer cell activity during bereavement. <u>Biological Psychiatry</u>, 24, 173-178.

Irwin, M., Daniels, M., & Weiner, H. (1987). Immune and neuroendocrine changes during bereavement. <u>Psychiatric Clinics of North America</u>, <u>10</u>(3), 449-465.

Irwin, M., Smith, T.L., & Gillin, J.C. (1992). Electroencephalographic sleep and natural killer activity in depressed patients and control subjects. <u>Psychosomatic Medicine</u>, <u>54</u>, 10-21.

Jemmott, J.B. (1985). Psychoneuroimmunology: The new frontier. <u>American</u> Behavioral Scientist, 28(4), 497-509.

Jemmott, J.B., Hellman, C., McClelland, D.C., Locke, S.E., Krause, L., Williams, R.M., & Valeri, C.R. (1990). Motivational syndromes associated with natural killer cell activity. <u>Journal of Behavioral Medicine</u>, <u>13</u>(1), 53-73.

Jennison, A. (1993). The HIV conspiracy. <u>Big Blue</u>, <u>3(1)</u>, 10-11.

Kaisch, K., & Anton-Culver, H. (1989). Psychological and social consequences of HIV exposure: Homosexuals in southern California. <u>Psychology and Health</u>, <u>3</u>, 63-75.

Kamen-Siegel, L., Rodin, J., Seligman, M.E.P., & Dwyer, J. (1991). Explanatory style and cell-mediated immunity in elderly men and women. <u>Health Psychology</u>, 10(4), 229-235.

Kasl, S.V., Evans, A.S., Niederman, J.C. (1979). Psychosocial risk factors in the development of infectious Mononucleosis. <u>Psychosomatic Medicine</u>, <u>41</u>(6), 445-466.

Kelly, J.A., & Murphy, D.A. (1992). Psychological interventions with AIDS and HIV: Prevention and treatment. <u>Journal of Consulting and Clinical Psychology</u>, <u>60</u>(4), 576-585.

Kelly, J.A., & St.Lawrence, J.S. (1989). <u>The AIDS health crisis</u> (2nd Edit.). New York: Plenum Press.

Kemeny, M.E., Cohen, F., Zegans, L.S., & Conant, M.A. (1989). Psychological and immunological predictors of genital Herpes recurrence. <u>Psychosomatic Medicine</u>, 51, 195-208.

Kemeny, M.E., Fahey, J.L., Schneider, S., Taylor, S.E., Weiner, H., & Visscher, B. (1989). Abstract: Psychosocial cofactors in HIV infection: Associations among bereavement, depression, and immunity in HIV+ and HIV- homosexual men. Psychosomatic Medicine, 51, 244-266.

Kennedy, S., Kiecolt-Glaser, J.K., & Glaser, R. (1988). Immunological consequences of acute and chronic stressors: Mediating role of interpersonal relationships. <u>British Journal of Medical Psychology</u>, <u>61</u>, 77-85.

Kent, J., Coates, T.J., Pelletier, K.R., & O'Regan, B. (1989). Unexpected recoveries: Spontaneous remission and immune functioning. <u>Advances</u>, <u>6</u>(2), 66-73.

Kessler, R.C., O'Brien, K., Joseph, J.G., Ostrow, D.G., Phair, J.P., Chmiel, J.S., Wortman, C.B., & Emmons, C.A. (1988). Effects of HIV infection, perceived health and clinical status on a cohort at risk for AIDS. <u>Social Science and Medicine</u>, <u>27</u>(6), 569-578.

Kiecolt-Glaser, J.K., Cacioppo, J.T., Malarkey, W.B., & Glaser, R. (1992). Editorial comment: Acute psychological stressors and short-term immune changes: What, why, for whom, and to what extent? <u>Psychosomatic Medicine</u>, <u>54</u>, 680-685.

Kiecolt-Glaser, J.K., Fisher, L.D., Ogrocki, P., Stout, J.C., Speicher, C.E., & Glaser, R. (1987). Marital quality, marital disruption, and immune function. Psychosomatic Medicine, 49(1), 13-34.

Kiecolt-Glaser, J.K., Garner, W., Speicher, C., Penn, G.M., Holliday, J., & Glaser, R. (1984). Psychosocial modifiers of immunocompetence in medical students. <u>Psychosomatic Medicine</u>, <u>46</u>(1), 7-14.

Kiecolt-Glaser, J.K., & Glaser, R. (1989). Psychoneuroimmunology: Past, present, and future. <u>Health Psychology</u>, <u>8</u>(6), 677-682.

Kiecolt-Glaser, J.K., & Glaser, R. (1991). Stress and immune function in humans. In R. Ader, D.L. Felten, & N. Cohen (Eds.). <u>Psychoneuroimmunology</u> (2nd Edit.), (pp.849-867). San Diego, Ca.: Academic Press.

Kiecolt-Glaser, J.K., & Glaser, R. (1992). Psychoneuroimmunology: Can psychological interventions modulate immunity? <u>Journal of Consulting and Clinical Psychology</u>, <u>60</u>(4), 569-575.

Kiecolt-Glaser, J.K., Glaser, R., Strain, E.C., Stout, J.C., Tarr, K.L., Holliday, J.E., & Speicher, C.E. (1986). Modulation of cellular immunity in medical students. Journal of Behavioral Medicine, 9(1), 5-21.

Kiecolt-Glaser, J.K., Glaser, R., Williger, D., Stout, J., Messick, G., Sheppard, S., Ricker, D., Romisher, S.C., Briner, W., Bonnell, G., & Donnerberg, R. (1985). Psychosocial enhancement of immunocompetence in a geriatric population. <u>Health Psychology</u>, 4(1), 25-41.

Kiecolt-Glaser, J.K., Kennedy, S., Malkoff, S., Fisher, L., Speicher, C.E., & Glaser, R. (1988). Marital discord and immunity in males. <u>Psychosomatic Medicine</u>, <u>50</u>, 213-229.

Kiecolt-Glaser, J.K., Ricker, D., George, J., Messick, G., Speicher, C.E., Garner, W., & Glaser, R. (1984). Urinary cortisol levels, cellular immunocompetency, and loneliness in psychiatric inpatients. <u>Psychosomatic Medicine</u>, <u>46</u>(1), 15-23.

Kiester, E. (Ed.). (1989). The new family medical guide. De Moines, Io.: Meredith.

King, E. (1993, August). HIV treatment is 'in its infancy', says gloomy experts. Gay Times, 30-31.

Kirkpatrick, R.A. (1981). Witchcraft and lupus erythematosus. <u>J.A.M.A.</u>, <u>245</u>(19), 1937.

Kirkwood, E.M., & Lewis, C.J. (1989). <u>Understanding medical immunology</u> (2nd Edit.), Chichester: John Wiley & Sons.

Knapp, P.H., Levy, E.M., Giorgi, R.G., Black, P.H., Fox, B.H., & Heeren, T.C. (1992). Short-term immunological effects of induced emotion. <u>Psychosomatic Medicine</u>, <u>54</u>, 133-148.

Korneva, E.A. (1989). Beginnings and main directions of psychoneuroimmunology. International Journal of Psychophysiology, 7, 1-18.

Kronful, Z., & House, J.D. (1984). Depression, cortisol, and immune function. Lancet, 1, 1026-1027.

Lang, W., Perkins, H., Anderson, R.E., Royce, R., Jewell, N., & Winkelstein, W. (1989). Patterns of T lymphocyte changes with human immunodeficiency virus infection: From seroconversion to the development of AIDS. <u>Journal of Acquired Immune Deficiency Syndromes</u>, 2, 63-69.

LaPerriere, A.R., Antoni, M.H., Schneiderman, N., Ironson, G., Klimas, N., Caralis, P., & Fletcher, M.A. (1990). Exercise intervention attenuates emotional distress and natural killer cell decrements following notification of positive serologic status for HIV-1. <u>Biofeedback and Self-Regulation</u>, <u>15(3)</u>, 229-242.

Laudenslager, M.L., & Reite, M.L. (1984). Losses and separations: Immunological consequences and health implications. <u>Review of Personality and Social Psychology</u>, 5, 285-312.

LeShan, L. (1989). Cancer as a turning point. Bath: Gateway Books.

Leventhal, H., & Tomarken, A. (1987). Stress and illness: Perspectives from Health Psychology. In S.V. Kasl and C.L. Cooper (Eds.), <u>Stress and health: Issues in research methodology</u>, pp.27-55.

Levenstein, S., Prantera, C., Varvo, V., Scribano, M.L., Berto, E., Luzi, C., & Andreoli, A. (1993). Development of the Perceived Stress Questionnaire: A new tool for psychosomatic research. <u>Journal of Psychosomatic Research</u>, <u>37</u>(1), 19-32.

Levine, J.D., Gordon, N.C., & Fields, H.L. (1978). The mechanism of placebo analgesia. The Lancet, 2, 654-657.

Levy, J.A. (1993). HIV pathogenesis and long-term survival. <u>AIDS</u>, <u>7</u>, 1401-1410.

Levy, S.M. (1988). Behavioral risk factors and host vulnerability. In T.P. Bridge, A.F. Mirsky, & F.K. Goodwin (Eds.), <u>Psychological</u>, <u>neuropsychiatric</u>, and <u>substance</u> abuse aspects of AIDS, pp.225-239. New York: Raven Press.

Levy, S.M., Herberman, R.B., Lee, J., Whiteside, T., Kirkwood, J., & McFeeley, S. (1990). Estrogen receptor concertiration and social factors as predictors of natural killer cell activity in early-stage breast cancer patients. Confirmation of a model. (Report No. 91172250). Pittsburgh, Pa.: Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh Cancer Institute. (MEDLINE Document Reproduction Service No. 91172250).

Levy, S., Herberman, R., Lippman, M., & d'Angelo, T. (1987). Correlation of stress factors with sustained depression of natural killer cell activity and predicted prognosis in patients with breast cancer. <u>Journal of Clinical Oncology</u>, <u>5</u>(3), 348-353.

Levy, S.M., Herberman, R.B., Whiteside, T., Sanzo, K., Lee, J., & Kirkwood, J. (1990). Perceived social support and tumor estrogen/progesterone receptor status as predictors of natural killer cell activity in breast cancer patients. Psychosomatic Medicine, 52, 73-85.

Locke, S.E., Kraus, L., Leserman, J., Hurst, M.W., Heisel, S., & Williams, R.M. (1984). Life change stress, psychiatric symptoms, and natural killer cell activity. Psychosomatic Medicine, 46(5), 441-453.

Longo, D.J., Clum, G.A., & Yaeger, N.J. (1988). Psychosocial treatment for recurrent genital herpes. <u>Journal of Consulting and Clinical Psychology</u>, <u>56</u>(1), 61-66.

Long-term survivors. (1993, October). Vancouver PWA, 72, 13-14.

Luborsky, L., Mintz, J., Brightman, V.J., & Katcher, A.H. (1976). Herpes simplex virus and moods: A longitudinal study. <u>Journal of Psychosomatic Research</u>, <u>20</u>, 543-548.

Marmot, M.G., & Madge, N. (1987). An epidemiological perspective on stress and health. In S.V. Kasl and C.L. Cooper (Eds.), <u>Stress and health: Issues in research methodology</u>, pp.3-26. Chichester: John Wiley & Sons.

Marshall, P.S. (1993). Allergy and depression: A neurochemical threshold model of the relation between illnesses. <u>Psychological Bulletin</u>, <u>113(1)</u>, 23-43.

Martin, J.L., & Dean, L. (1993). Effects of AIDS-related bereavement and HIV-related illness on psychological distress among gay men: A 7-year longitudinal study, 1985-1991. <u>Journal of Consulting and Clinical Psychology</u>, <u>61(1)</u>, 94-103.

Martin, R.A., Dobbin, J.P. (1988). Sense of humor, hassles, and immunoglobulin A: Evidence for a stress-moderating effect of humor. <u>International Journal of Psychiatry in Medicine</u>, <u>18</u>(2), 93-105.

McClelland, D.C., & Kirshnit, C. (1988). The effect of motivational arousal through films on salivary immunoglobulin A. <u>Psychology and Health</u>, 2, 31-52.

McClelland, D.C., Patel, V., Brown, D., & Kelner, S.P. (1991). The role of affiliative loss in the recruitment of helper cells among insulin-dependent diabetics. Behavioral Medicine, 17(1), 5-14.

McCutchan, J.A. (1990). Virology, immunology, and clinical course of HIV infection. <u>Journal of Consulting and Clinical Psychology</u>, <u>58</u>(1), 5-12.

McGrady, A., Conran, P., Dickey, D., Garman, D., Farris, E., & Schumann-Brzezinski, C. (1992). The effects of biofeedback-assisted relaxation on cell-mediated immunity, cortisol, and white blood cell count in healthy adult subjects. <u>Journal of Behavioral Medicine</u>, <u>15</u>(4), 343-354.

McKenzie, N.F. (Ed.). (1991). <u>The AIDS reader: Social, political, and ethical issues.</u> New York: Meridian.

McKinnon, W., Weisse, C.S., Reynolds, C.P., Bowles, C.A., & Baum, A. (1989). Chronic stress, leukocyte subpopulations, and humoral response to latent viruses. <u>Health Psychology</u>, <u>8</u>(4), 389-402.

McKirnan, D.J., & Peterson, P.L. (1989). AIDS-risk behavior among homosexual males: The role of attitudes and substance abuse. <u>Psychology and Health</u>, <u>3</u>, 161-171.

McKusick, L. (1988). The impact of AIDS on practitioner and client: Notes for the therapeutic relationship. <u>American Psychologist</u>, <u>43</u>(11), 935-940.

McNair, D.M., Lorr, M., & Droppleman, L.F. (1971). <u>EITS manual for the Profile of Mood States</u>. San Diego, CA.: Educational and Industrial Testing Service.

McNaughton, M.E., Smith, L.W., Patterson, T.L., & Grant, I. (1990). Stress, social support, coping resources, and immune status in elderly women. <u>Journal of Nervous and Mental Disease</u>, <u>178</u>(7), 460-461.

Medical Research Council UK. (1993, April 2). Results from the Concorde Trial. London: Medical Research Council Press Notice.

Mendolia, M., & Kleck, R.E. (1993). Effects of talking about a stressful event on arousal: Does what we talk about make a difference? <u>Journal of Personality and Social Psychology</u>, <u>64</u>(2), 283-292.

Moss, R.B., Moss, H.B., & Peterson, R. (1989). Microstress, mood, and natural killer-cell activity. <u>Psychosomatics</u>, <u>30(3)</u>, 279-283.

Namir, S., Alumbaugh, M.J., Fawzy, F.I., & Wolcott, D.L. (1989). The relationship of social support to physical and psychological aspects of AIDS. <u>Psychology and Health</u>, 3, 77-86.

Naor, S., Assael, M., Pecht, M., Trainin, N., & Samuel, D. (1983). Correlation between emotional reaction toloss of an unborn child and lymphocyte response to mitogenic stimulation in women. <u>Israeli Journal of Psychiatry Related Sciences</u>, 20(3), 231-239.

National Institute of Allergy and Infectious Diseases (1993, April 1). <u>Concorde Trial</u>. Bethesda, Md: National Institute of Allergy and infectious Diseases, U.S. Department of Health and Human Services.

Neveu, P.J., Crestani, F., & Le Moal, M. (1987). Conditioned immunosuppression: A new methodological approach. <u>Annals of the New York Academy of Sciences</u>, <u>496</u>, 595-601.

Nielsen, R. (1993, February). Positive attitudes and survival (Excerpt from Longterm Survival Skills, Seattle Treatment Education Project, Vol 5(1), February 1993). Vancouver PWA, 66, 9.

O'Leary, A. (1990). Stress, emotion, and human immune function. <u>Psychological</u> <u>Bulletin</u>, <u>108</u>(3), 363-382.

Ostrow, D.G. (1988). Models for understanding the psychiatric consequences of AIDS. In T.P. Bridge, A.F. Mirsky, & F.K. Goodwin (Eds.), <u>Psychological</u>, <u>neuropsychiatric</u>, <u>and substance abuse aspects of AIDS</u>, pp.85-94. New York: Rayen Press.

Ostrow, D.G., Monjan, A., Joseph, J., VanRaden, M., Fox, R., Kingsley, L., Dudley, J., & Phair, J. (1989). HIV-related symptoms and psychological functioning in a cohort of homosexual men. <u>American Journal of Psychiatry</u>, <u>146</u>(6), 737-742.

Pennebaker, J.W., & Beall, S.K. (1986). Confronting a traumatic event: Toward an understanding of inhibition and disease. <u>Journal of Abnormal Psychology</u>, <u>95</u>(3), 274-281.

Pennebaker, J.W., Hughes, C.F., & O'Heeron, R.C. (1987). The psychophysiology of confession: Linking inhibitory and psychosomatic processes. <u>Journal of Personality and Social Psychology</u>, <u>52</u>(4), 781-793.

Pennebaker, J.W., Kiecolt-Glaser, J.K., & Glaser, R. (1988). Disclosure of traumas and immune function: Health implications for psychotherapy. <u>Journal of Consulting and Clinical Psychology</u>, <u>56(2)</u>, 239-245.

Pennebaker, J.W., & Susman, J.R. (1988). Disclosure of traumas and psychosomatic processes. <u>Social Science and Medicine</u>, <u>26(3)</u>, 327-332.

Peplau, L.A., & Perlman, D. (1982). Perspectives on loneliness. In L.A. Peplau and D. Perlman (Eds.). (1982). <u>Loneliness: A Sourcebook of current theory</u> research and therapy, (chap. 1). New York: John Wiley & Sons.

Perry, S., Fishman, B., Jacobsberg, L., & Frances, A. (1992). Relationships over 1 year between lymphocyte subsets and psychosocial variables among adults with infection by human immunodeficiency virus. <u>Archives of General Psychiatry</u>, <u>49</u>, 396-401.

Perry, S., Fishman, B., Jacobsberg, L., Young, J., & Frances, A. (1991). Effectiveness of psychoeducational interventions in reducing emotional distress after Human Immunodeficiency Virus antibody testing. <u>Archives of General Psychiatry</u>, <u>48</u>, 143-147.

Phillips, K. (1991). The psychophysiology of health. In M. Pitts & K. Phillips (Eds.). The psychology of health (pp. 15-29). London: Routledge.

Polk, B.F., Fox, R., Brookmeyer, R., Kanchanaraksa, S., Kaslow, R., Visscher, B., Rinaldo, C., & Phair, J. (1987). Predictors of the acquired immunodeficiency syndrome developing in a cohort of seropositive homosexual men. <u>New England Journal of Medicine</u>, 316(2), 61-66.

Proto, L. (1990). <u>Self-healing: Use your mind to heal your body</u>. London: Judy Platkus.

Rabkin, J.G., Williams, J.B.W., Neugebauer, R., Remien, R.H., & Goetz, R. (1990). Maintenance of hope in HIV-spectrum homosexual men. <u>American Journal of Psychiatry</u>, 147(10), 1322-1326.

Rabkin, J.G., Williams, J.B.W., Remien, R.H., Goetz, R., Kertzner, R., & Gorman, J.M. (1991). Depression, distress, lymphocyte subsets, and human immunodeficiency virus symptoms on two occasions in HIV-positive homosexual men. <u>Archives of General Psychiatry</u>, 48, 111-119.

Ratliff-Crain, J., Temoshok, L., Kiecolt-Glaser, J.K., & Tamarkin, L. (1989). Issues in psychoneuroimmunology research. <u>Health Psychology</u>, <u>8</u>(6), 747-752.

Report slams government AIDS research (1994, January 27). <u>San Francisco Bay Times</u>, p.5.

Rider, M.S., Achterberg, J., Lawlis, G.F., Goven, A., Toledo, R., & Butler, J.R. (1990). Effect of immune system imagery on secretory IgA. <u>Biofeedback and Self-Regulation</u>, <u>15</u>(4), 317-333.

Rider, M.S., & Weldin, C. (1990). Imagery, improvisation, and immunity. <u>The Arts in Psychotherapy</u>, <u>17</u>, 211-216.

Rigsby, L.W., Dishman, R.K., Jackson, A.W., MacLean, G.S., & Raven, P.B. (1992a). Effects of exercise training on men seropositive for the human immunodeficiency virus-1. <u>Medicine and Science in sports and exercise</u>, 24(1), 6-12.

Rigsby, L.W., Dishman, R.K., Jackson, A.W., MacLean, G.S., & Raven, P.B. (1992b). Response to comments on: Effects of exercise training on men seropositive for the human immunodeficiency virus-1. <u>Medicine and Science in sports and exercise</u>, 24(7), 838-840.

Root-Bernstein, R.S. (1992). Rethinking AIDS. Frontier Perspectives, 3(1), 11-14;37.

Rosenthal, R., & Rosnow, R.L. (1991). <u>Essentials of behavioral research</u> (2nd Edit.). New York: McGraw-Hill.

Rundell, J.R., Paolucci, S.L., Beatty, D.C., & Boswell, R.N. (1988). Psychiatric illness at all stages of Human Immunodeficiency Virus infection. <u>American Journal of Psychiatry</u>, 145(5), 652-653.

Russell, D. (1982). The measurement of loneliness. In L.A. Peplau and D. Perlman (Eds.). (1982). <u>Loneliness: A Sourcebook of current theory, research and therapy, pp.81-104</u>. New York: John Wiley & Sons.

Russell, D., Peplau, L.A., & Cutrona, C.B. (1980). The revised UCLA Loneliness Scale: concurrent and discriminant validity evidence. <u>Journal of Personality and Social Psychology</u>, 39, 472-480.

Scheier, M.F., & Carver, C.S. (1985). Optimism, coping, and health: Assessment and implications of generalised outcome expectancies. <u>Health Psychology</u>, <u>4</u>(3), 219-247.

Schleifer, S.J., Keller, S.E., Bond, R.N., Cohen, J., & Stein, M. (1989). Major depressive disorder and immunity. <u>Archives of General Psychiatry</u>, <u>46</u>, 81-87.

Schleifer, S.J., Keller, S.E., Meyerson, A.T., Raskin, M.J., Davis, K.L., & Stein, M. (1984). Lymphocyte function in major depressive disorder. <u>Archives of General Psychiatry</u>, <u>41</u>, 484-486.

Schmale, A.H. (1958). Relationship of separation and depression to disease. <u>Psychosomatic Medicine</u>, <u>20</u>(4), 259-277.

Sheppard, H.W., Lang, W., Ascher, M.S., Vittinghoff, E., & Winkelstein, W. (1993). The characterization of non-progressors: Long-term HIV-1 infection with stable CD4+ T-cell levels. <u>AIDS</u>, 7, 1159-1166.

Shilts, R. (1987). And the band played on. New York: St. Martin's Press.

Siegel, B.S. (1986). Love, medicine & miracles. London: Arrow Books.

Simonton, O.C., Matthews-Simonton, S., & Creighton, J.L. (1978). <u>Getting well again</u>. New York: Bantam.

Sims, R., & Moss, V.A. (1991). <u>Terminal care for people with AIDS</u>. London: Edward Arnold.

Smith, G.R., & McDaniel, S.M. (1983). Psychologically mediated effect on the delayed hypersensitivity reaction to tuberculin in humans. <u>Psychosomatic Medicine</u>, 45(1), 65-70.

Solano, L., Costa, M., Salvati, S., Coda, R., Aiuti, F., Mezzaroma, I., & Bertini, M. (1993). Psychosocial factors and clinical evolution in HIV-1 infection: A longitudinal study. <u>Journal of Psychosomatic Research</u>, <u>37</u>(1), 39-51.

Solomon, G.F., Kemeny, M.E., & Temoshok, L. (1991). Psychoneuroimmunologic aspects of Human Immunodeficiency Virus infection. In R. Ader, D.L. Felten, & N. Cohen (Eds., <u>Psychoneuroimmunology</u> (2nd Edit.), pp.1081-1113. San Diego, Ca.: Academic Press.

Solomon, G.F., & Moos, R.H. (1964). Emotions, immunity, and disease. <u>Archives of General Psychiatry</u>, <u>11</u>,657-74.

Solomon, G.F., Temoshok, L., O'Leary, A., & Zich, J. (1987). An intensive psychoimmunologic study of long-surviving persons with AIDS: Pilot work, background studies, hypotheses, and methods. <u>Annals of the New York Academy of Sciences</u>, 496, 647-655.

Sontag, S. (1991). <u>Illness as metaphor and AIDS and its metaphors</u>. London: Pengion.

Spanos, N.P., Williams, V., & Gwynn, M.I. (1990). Effects of hypnotic, placebo, and salicyclic acid treatments on wart regression. <u>Psychosomatic Medicine</u>, <u>52</u>, 109-114.

Spencer, D. (1993). Book reviews. AIDS Bulletin, 2(2), 20.

Stall, R.D., Coates, T.J., & Hoff, C. (1988). Behavior risk reduction for HIV infection among gay and bisexual men: A review of results from the United States. <u>American Psychologist</u>, 43(11), 878-885.

Stein, M., Keller, S.E., & Schleifer, S.J. (1985). Stress and immunomodulation: The role of depression and neuroendocrine function. <u>The Journal of Immunology</u>, <u>135(2)</u>, 827s-833s.

Stein, M., Miller, A.H., & Trestman, R.L. (1991a). Depression, the immune system, and health and illness. <u>Archives of General Psychiatry</u>, <u>48</u>, 171-177.

Stein, M., Miller, A.H., & Trestman, R.L. (1991b). Depression and the immune system. In R. Ader, D.L. Felten, & N. Cohen (Eds., <u>Psychoneuroimmunology</u> (2nd Edit.), pp.897-930. San Diego, Ca.: Academic Press.

Steinberg, M. (1992). HIV infection in South Africa. AIDS Bulletin, 1(2), 19-20.

Stern, V.F. (1988). Psychoimmunology. <u>Psychotherapy Patient</u>, <u>4(2)</u>, 13-32.

Stice, E. (1992). The similarities between cognitive dissonance and guilt: Confession as a relief of dissonance. <u>Current Psychology: Research & Reviews</u>, 11(1), 69-77.

Suls, J., & Fletcher, B. (1985). The relative efficacy of avoidant and nonavoidant coping strategies: A meta-analysis. <u>Health Psychology</u>, <u>4</u>(3), 249-288.

Tecoma, E.S., & Huey, L.Y. (1985). Psychic distress and the immune response. The Life Sciences, 36, 1799-1812.

Temoshok, L. (1988). Psychoimmunology and AIDS. In T.P. Bridge, A.F. Mirsky, & F.K. Goodwin (Eds.), <u>Psychological</u>, <u>neuropsychiatric</u>, <u>and substance abuse aspects of AIDS</u>, pp. 187-197. New York: Raven Press.

The AIDS Coalition to Unleash Power (1994). <u>Committee to monitor poppers</u>. (Available from The AIDS Coalition to Unleash Power, 519 Castro Street, San Francisco, Ca., 94114).

Thomas, P.D., Goodwin, J.M., & Goodwin, J.S. (1985). Effect of social support on stress-related changes in cholesterol level, uric acid level, and immune function in an elderly sample. <u>American Journal of Psychiatry</u>, <u>142</u>(6), 735-737.

Tross, S., & Hirsch, D.A. (1988). Psychological and neuropsychological complications of HIV infection and AIDS. <u>American Psychologist</u>, <u>43</u>(11), 929-934.

Udelman, H.D., & Udelman, D.L. (1983). Current explorations in psychoimmunology. <u>American Journal of Psychotherapy</u>, <u>37(2)</u>, 210-221.

Vollhardt, L.T. (1991). Psychoneuroimmunology: A literature review. <u>American</u> <u>Journal of Orthopsychiatry</u>, 61(1), 35-47.

Weidenfeld, S.A., O'Leary, A., Bandura, A., Brown, S., Levine, S., & Raska, K. (1990). Impact of perceived self-efficacy in coping with stressors on components of the immune system. <u>Journal of Personality and Social Psychology</u>, <u>59</u>(5), 1082-1094.

Weinberger, D.A., Schwartz, G.E., & Davidson, R.J. (1979). Low-anxious, high-anxious, and repressive coping styles: Psychometric patterns and behavioral and physiological responses to stress. <u>Journal of Abnormal Psychology</u>, <u>88</u>(4), 369-380.

Weiner, H. (1991). Social and psychobiological factors in autoimmune disease. In R. Ader, D.L. Felten, & N. Cohen (Eds.), <u>Psychoneuroimmunology</u> (2nd Edit.) (pp.955-1011). San Diego, Ca.: Academic Press.

Weiss, R.S. (1982). Issues in the study of loneliness. In L.A. Peplau and D. Perlman (Eds.), <u>Loneliness: A sourcebook of current theory, research and therapy</u>, pp.71-80. New York: John Wiley & Sons.

Weisse, C.S. (1992). Depression and immunocompetence: A review of the literature. <u>Psychological Bulletin</u>, <u>111(3)</u>, 475-489.

Wilson, D. (1993). Community level HIV prevention among vulnerable groups in Zimbabwe. <u>AIDS Bulletin</u>, <u>2</u>(1), 8-10.

Wilson, J.F. (1981). Behavioral preparation for surgery: Benefit or harm? <u>Journal of Behavioral Medicine</u>, <u>4</u>, 79-102.

Zachariae, R., Kristensen, J.S., Hokland, P., Ellegaard, J., Metze, E., & Hokland, M. (1990). Effect of psychological intervention in the form of relaxation and guided imagery on cellular immune function in normal healthy subjects. Psychotherapy and Psychosomatics, 54, 32-39.

Zakowski, S.G., McAllister, C.G., Deal, M., & Baum, A. (1992). Stress, reactivity, and immune function in healthy men. <u>Health Psychology</u>, <u>11</u>(4), 223-232.

# APPENDIX 1 RESEARCH INSTRUMENT

Hi

This is a survey concerning coping strategies and HIV infection. Hold on! Before you dismiss this as 'just another project' and throw this away, please read the following, as there are a number of issues which may affect your decision to participate.

**The purpose of this survey** is to determine: (a) The specific needs of people who are HIV infected; (b) The various coping strategies people use, and whether this is in any way linked to the progression of HIV infection.

There are probably two main questions you may ask about this research: "How will it benefit me?", and "Will my anonymity be guaranteed?".

A major benefit of this study will be determining whether specific methods of coping are linked to the progression of HIV infection. This is based upon previous research concerning the effects of stress on the immune system. If such as link is established, then a program designed to improve coping skills could be initiated, with benefits for anyone who is HIV infected. It should be emphasised that specific coping strategies work better only in certain situations, and that it is unclear which specific coping strategy would be most beneficial for those who are HIV infected. Therefore, your cooperation and honesty is highly appreciated. There really are no correct or incorrect answers to any of the questions, as the answers to these issues will emerge from those who participate in the study.

A second benefit concerns the gaining of an understanding of the specific needs (emotional, information, legal, medical, etc.) of people who are HIV infected. This information will be made directly available to ASET.

Several safeguards to protect your identity have been included in this study. At no time is your name or address known to me: The selection of participants is done by ASET, and I merely supply the questionnaire and necessary stationary. Secondly, you are not required to write your name - or any other personally identifiable information - on the questionnaire itself. The results of the study will be available in a few months, and you will receive a copy of these results, using the same procedures to safeguard your identity.

I hope that the above has provided you with sufficient information to enable you to make an informed decision about participating or not. If you decide to complete the questionnaire (which should take about 20 to 30 minutes), please return the completed questionnaire in the envelope provided. Thank you.

#### **Neil Orr**

M.A. Post-graduate Research Department of Psychology, University of Cape Town

### **DEALING WITH STRESS GENERALLY**

The following section concerns the	many ways in which	n people respo	nd when they	confront							
difficult or stressful events in their l	ives. You are asked	l to indicate wi	hat you GENER	ALLY do							
and feel, when you experience stressful events.											
1 = Not at all	I usually don't do thi	is at all									

3 = Often 4 = Most of the time			I often do this, but not all the time I almost always do this							
- I	NO31 OI I	110 111110	•••••	i dilliosi (	uiwaj	3 <b>u</b> o 1	11113			
N GENERAL,	WHEN I	EXPERIE	NCE A D	IFFICULT C	OR STI	RESSFL	IL SITU	ATION		
. I talk to some									ings from	
1	2	3	4		inter with		vith my e	efforts at	dealing	
I make a pla	in of actio	n 3	4			1	2	3	4	
'	2	3	4	17	i thin	k hard	about w	hat stan	to take	
i talle ta cama					. Finin	k nara (	_		s to take.	
i talk to some concrete ab			somerning	<b>,</b>		•	2	3	4	
1	2	3	4	18	. I refu	se to b	elieve th	at it has	happened.	
						•	2	3	4	
I turn to work				10	1		4   - 4	41		
activities to t	rake my n	_	ngs.	19	. I get	upset c	_	ny emoti	ons out.	
1	2	3	4			1	2	3	4	
i. I just give up trying to re		each my goal.		20.	). I take aduitional action to try					
1	2	3	4		to ge	et rid of	the prot	olem.		
					_	1	2	3	4	
I go to movie		h TV,								- and
to think abou	ut it less.			21.	. I lean	n to live	with it.			
1	2	3	4			1	2	3	4	
l drink alcoh				22.	l restr	ain mys	elf from	doing a	nything	
in order to the	nink about	it less.			too c	uickly.		_	_	
1	2	3	4			1	2	3	4	
l keep myself	from get	ina distra	rted	23	l cet	uneet d	and I am	really a	ware of it.	1
by other thou			3164	20.	1961	ирэ <del>с</del> і, (	_	3	Wale of II.	
l l	ignis or do	3	4			'	2	3	4	
				. 24.	I try to	get er	motiona	support	•	
I think about t	how I mig	ht best			from	friends	or relativ	es.		
handle the p	roblem.					1	2	3	4	
1	2	3	4							
				25.	l ask p	people	who hav	e had si	milar	
I accept the	reality of	the fact tl	hat				what the			
it happened						1	2	3	4	
i	2	3	4							
				26.	I do w	hat ha	s to be d	done.		
I force myself	f to wait fo	or the righ	t time			tep at a				
to do someth		•				}	2	3	4	
1	Ž	3	4						-	
				27.	1 lear	somet	hing fror	n the ex	perience.	
I admit to my		can't dec	al			1	2	3	4	
with it, and a	lait italug.	2	4		14					
	2	3	4	28.		come to do.	up with	व शावां (	gy about	
I look for som	ething go	od in who	it				2	3	4	
is happening										
i	2	3	4	29.	l act d	s thou	ah it hası	n't even	happened.	
					1		2	3	4	
I pretend that	t it hasn't	really hap	pened.					-		
1	2	3	4	30.	I try to	find co	omfort in	my relig	ijon.	
•	_	-		<b>55.</b>	, ,		2	3	μοι μ. Δ	
I hold off doin	na anvithin	a about it	until		1		-	•	7	
the situation		G	3	31	Ltake	direct 4	action to	get ara	und	
1	2	3	4	01.		oblem		901 010		
					_		•	_		
	,				1		2	3	4	

#### 1 = Not at all 2 = Not often 3 = Often 4 = Most of the time

									4 - 44 - m 1/ m		
32.	I focus on de	aling with	n this prob	lem,	43.			amount o		n pumng	
	and if necessary let other things slide.					into solv	ring ti	ne proble	m,		
	a little.	,	•			1	-	2	3	4	
	1	2	3	4							
	ı	2	5	4	44	1411	-+ -4	emotiono	diatross.		
					44.		-				
33.	I make sure n	ot to mal	ce matter	s worse			•	self expre	ssing thos	е	
	by acting too	soon.				feelings	a lot			•	
	1	2	3	4		ĭ		2 .	3	4	
	'	_	•	~		•		_			
••					AC	I alanıalıa	~~~	hout thin	ar other t	han this	
34.	I seek God's h	neip.	_		45.	i dayare	am c	about thin	Az on en		
	1	2	3	4		ı		2	3	4	
35.	I get sympath	v and un	derstandi	ng ·	46.	I talk to	some	one to fit	nd out mo	re	
•	from someone					about ti	ne situ	uation.			
	1	2	3	4		1		2	3	4	
	ı	2	3	-				-	·	-	
36.	I say to myself		real".		4/.	I put my	must	in God.	_		
	1	2	3	4		1		2	3	4	
37.	I accept that	this has h	appened	1	48.	I put asia	de ot	her activit	ies in ord	er to	
٠,,	and that it ca			-		concen					
	una main ca	111 20 011		4		1	naic	2	3	4	
	ı	2	3	4		ı		4	3	4	
38.	I pray more th	nan usual			49.	l give up	the	attempt t	o get who	at I want,	
	1	2	3	4		1		2	3	4	
30	I let my feeling	as Out			50	I discuss	mv fe	elings wit	h someor	ne .	
٠,,	1	2	3	4	٠.	1	,	•	3	4	
	ı	2	3	4		,		2	3	4	
40.	I sleep more t	han usua	i.		51.	i concer	ntrate	my effor	ts on doin	g	
	1	2	3	4		somethi	ng at	out it.			
						1	•	2	3	4	
41	I try to get ad	vice from	someone	<b>a</b>		•		_	•	•	
	about what to			•	50	1 40 40 0					
	apoul what is	_	•		<b>52</b> .	, .		s a perso			
	ı	2	3	4		result of	the e	experience			
						1		2	3	4	
42.	I try to see it in	a differe	ent light,								
	to make it see				53	Last use	d to t	the idea t	hat it han	pened	
	1	2	3	4	<b>5</b> 0.	1	5	2	3	4	
	'	2	•	-		,		4	J	-	

For each of the following sentences, circle the number that best describes how often it applies to you in general, DURING THE LAST YEAR OR SO. Circle only one number for each item.

- 54. I feel I am doing things I really like.

  Almost never 1 2 3 4 Often
- 55. No one really knows me very well.

  Almost never 1 2 3 4 Offen
- 56. I enjoy myself.

Almost never 1 2 3 4 Offen

- 57. People are around me, but not with me.

  Almost never 1 2 3 4 Often
- 58. I feel in tune with people around me.
  Almost never 1 2 3 4 Often
- 59. I fear I may not manage to attain my goals.

  Almost never 1 2 3 4 Offen
- 60. I can find a relationship when I want it.

  Almost never 1 2 3 4 Often
- 61. I am afraid for the future.

  Almost never 1 2 3 4 Often

PAGE 2

PLEASE CONTINUE ON THE NEXT PAGE ...

APPENDIX 1 (iii)

#### FEELINGS: THE PAST WEEK AND TODAY

Below is a list of words that describe feelings that people have. Please read each one carefully. Then circle ONE number to the right which best describes HOW YOU HAVE BEEN FEELING DURING THE PAST WEEK INCLUDING TODAY.

0 = Not at all

1 = A little

2 = Moderately

3 = Quite a bit

4 = Extremely

1. Friendly	0 1 2 3 4	23. Unworthy 0 1 2 3 4	45. Desperate 0 1 2 3 4
2. Tense	0 1 2 3 4	24. Spiteful 0 1 2 3 4	46. Sluggish 0 1 2 3 4
3. Angry	0 1 2 3 4	25. Sympathetic 0 1 2 3 4	47. Rebellious 0 1 2 3 4
4. Worn out	0 1 2 3 4	26. Uneasy 0 1 2 3 4	48. Helpless 0 1 2 3 4
5. Unhappy	0 1 2 3 4	27. Resteless 0 1 2 3 4	49. Weary 0 1 2 3 4
6. Clear-headed	d0 1234	28. Unable to	50. Bewildered 0 1 2 3 4
7. Lively	0 1 2 3 4	concentrate 0 1 2 3 4	51. Alert 0 1 2 3 4
8. Confused	0 1 2 3 4	29. Fatigued 0 1 2 3 4	52. Deceived 0 1 2 3 4
9. Sorry for		30. Helpful 0 1 2 3 4	53. Furious 0 1 2 3 4
things done	0 1 2 3 4	31. Annoyed 0 1 2 3 4	54. Efficient 0 1 2 3 4
10. Shaky	0 1 2 3 4	32. Discouraged 0 1 2 3 4	55. Trusting 0 1 2 3 4
11. Listless	0 1 2 3 4	33. Resentful 0 1 2 3 4	56. Full of pep 0 1 2 3 4
12. Peeved	0 1 2 3 4	34. Nervous 0 1 2 3 4	57. Bad
13. Considerate	0 1 2 3 4	35. Lonely 0 1 2 3 4	tempered 0 1 2 3 4
14. Sad	0 1 2 3 4	36. Miserable 0 1 2 3 4	58. Worthless 0 1 2 3 4
15. Active	0 1 2 3 4	37. Muddled 0 1 2 3 4	59. Forgetful 0 1 2 3 4
16. On edge	0 1 2 3 4	38. Cheerful 0 1 2 3 4	60. Carefree 0 1 2 3 4
17. Grouchy	0 1 2 3 4	39. Bitter 0 1 2 3 4	61. Terrified 0 1 2 3 4
18. Blue	0 1 2 3 4	40. Exhausted 0 1 2 3 4	62. Guilty 0 1 2 3 4
19. Energetic	0 1 2 3 4	41. Anxious 0 1 2 3 4	63. Vigorous 0 1 2 3 4
20. Panicky	0 1 2 3 4	42. Ready to	64. Uncertain
21. Hopeless	0 1 2 3 4	fight 0 1 2 3 4	about things 0 1 2 3 4
22. Relaxed	0 1 2 3 4	43. Good	65. Bushed 0 1 2 3 4
		natured 0 1 2 3 4	

# THE FOLLOWING INFORMATION IS STRICTLY FOR STATISTICAL PURPOSES. ALL INFORMATION WILL BE TREATED WITH THE UTMOST CONFIDENTIALITY.

TODAY'S DAT	E: DAY MONTH 19
YOUR PRESENT	T AGE: YEARS MONTHS
GENDER:	□ MALE □ FEMALE
SEXUAL PREFE	RENCE:   HETEROSEXUAL   GAY/HOMOSEXUAL   BISEXUAL
HOME LANGU	AGE:   ENGLISH   AFRIKAANS   XHOSA   ZULU OTHER:
HIGHEST EDUC	CATIONAL LEVEL ATTAINED:
EMPLOYMENT	STATUS:   CURRENTLY EMPLOYED  CURRENTLY UNEMPLOYED
INCOME:	Rper month
RESIDENCE:	The following information is to be used to determine how close you live to certain places which offer various HIV-related services:
Provinc	ce: City/Town/Suburb/Township:
Do you have	your own means of transport (e.g., a car)? $\Box$ Yes $\Box$ No
CURRENT HIV-	STATUS:   HIV-SERONEGATIVE
FIRST UN/ TEST	☐ HIV-SEROPOSITIVE
FIRST HIV TEST:	MONTH: RESULT:   NEGATIVE   POSITIVE
	>>>>>
HIV-positive, a likely ways of vaginal or and about the pe	ally occurs through unprotected (eg., no condom) sex with someone who was although blood transfusions and sharing of syringe needles are also two of the less infection. The most common ways of getting the HIV are through unprotected at sex. If you have not yet determined when you became infected, please think ariod of time prior to finding out that you were HIV-positive, and indicate a possible when you think infection was most likely to have occurred.
down. It may you were mos (eg. from Jan	dicate how certain you feel about the dates (or periods of time) that you write be difficult to think of a specific date (such as specific month) in which you think tilkely to have been infected. In this case, indicate the most likely period of time uary to July 1986) instead. However, if you are quite certain about a specific ust indicate the month and year. Please be as accurate as you can be.
MOST LIKELY P	ERIOD OF TIME IN WHICH INFECTION OCCURRED.
IN THE	PERIOD <b>FROM</b> Month: 19 <b>TO</b> Month: 19
Please indicate	e how certain you feel about this date, on a scale from 7 to 1:
Very ce	ertain 7 6 5 4 3 2 1 Very uncertain
PAGE 4	PLEASE CONTINUE ON THE NEXT PAGE APPENDIX 1 (V)

Please indicate whether you have experienced any of the following symptoms, and when they first began (if at all):

\* NOTE: Indicate only those symptoms which lasted for 2 or more weeks in the last 6 months. If you are uncertain, please consult your medical doctor.

IF YES, WHEN (DATE)
IT FIRST STARTED:

PAGE 5	PLEAS	SE CONTINUE ON THE NEXT PAGE	APPENDIX 1 (vi)
		THANK YOU FOR THIS INFORMATION	
Drug:		When used/for how long:	
Drug:		When used/for how long:	
please	specify the drug, a	have you in the past, used antiretroviral and for how long you have been using It. hen, and for how long.	
□ NO	YES:	Any serious fungal infection, such as Myc or Salmonella.	obacterium, Shigella,
□ NO	YES:	Any serious viral infection, such as Cytom virus.	egalovirus or Papova
□ NO	YES:	Any lymphoma or tumours, such as Kapa	si's sarcoma.
□ NO	YES:	Any serious protozoal infection, such as P Toxoplasma, Cryptosporidium, or Isosporo	
□ NO	YES:	Chronic muscle aches	
□ NO	YES:	Chronic fatigue.	
□ NO	YES:	Fever of more than 37.8 degrees Celsius.	
□ NO	YES:	Weight loss of at least 4.5 kg over the las	t six months.
□ NO	YES:	Chronic diarrhoea for more than a mont	h.
□ NO	YES:	Night sweats.	
□ NO	YES:	Persistently swollen lymph glands which of three months, are at least 1 cm in diamoleast two sites away from the groin.	
□ NO	YES:	Decreased in CD4 lymphocytes and low cell counts.	white or red blood .
□ NO	YES:	Sickness such as oral thrush (candidiasis), virus infection on the genitals or lips (feve month.	

#### NEED FOR INFORMATION, ASSISTANCE AND COUNSELLING

This section concerns any emotional and/or practical needs related to HIV-status which you may have. Due to the secrecy because of the general social fear of HIV, and the fact that HIV is usually transmitted sexually, It is difficult to determine the anxieties and needs of people with HIV. Your feedback is therefore highly valued.

The following are a few areas which you may relate to. If there are other areas which you feel deserve attention, please indicate these at item (g) at the bottom of the next page. If you require further space for your comments, please write these on a separate piece of paper, and insert it here.

(a) SERVICES ALREADY UTILISED: If you have already received counselling, or participated in an organized programme for coping with HIV, please indicate this, and specify the organization

•		
	REGARDING FINANCIAL, LEGAL OR MEDICAL PROB ich you have experienced, and for which you wou	
	. INFORMATION: Is there any factual information to V-positive? Please indicate these areas.	that would assist you in dealing
	NE TO SPEAK TO ABOUT YOUR ANXIETIES: Please el you would like to have someone assist you with.	indicate any specific anxietie
(II) He	ow do/did Juch services sult your needs?	
(i) Se	ervices/programmes already utilized, and reason:	

	NCE AND INFORMATION ABOUT METHODS FOR COPING at people use for coping with HIV, such as relaxation tests, etc.		
(i)	Are you aware of such methods of coping?	□ YES	□ NO
(ii)	Would you like more information and/or assistance regarding such methods?	□ YES	□ NO
(iii)	Please indicate any specific methods of coping you would like to find out more about:		
(f) ORGANIZ	ED GROUP PROGRAMMES FOR COPING WITH HIV:		
(i)	Do you have any reservations or objections to partice programmes, which deal specifically with coping wing Eg: Fear of having to reveal your HIV+ status, or fear infections from others? If so, please specify your persuch programmes:	th HIV? of possibly con	tacting
(ii)	If you are willing to participate in such group program of programme do you feel you might benefit from ? for emotional adjustment, or problem-focused meth HIV problems.	Eg: Group sup	port
(g) Any oth	ER AREA OF CONCERN TO YOU WHERE YOU WOULD FIN	D ASSISTANCE U	ISEFUL:
	MANY OF THE ABOVE-MENTIONED SERVICES ARE AVAINCIES AND ORGANIZATIONS.	ILABLE AT	
	ormation, contact <b>LIFELINE</b> in your area, who will then reagency for further assistance.	efer you to the	
	THANK YOU FOR THE INFORMATION THAT YOU HAVE EASE MAIL THIS QUESTIONNAIRE IN THE ADDRESSED ENV Y QUERIES REGARDING THIS SURVEY CAN BE ADDRESSEI AT THE ADDRESS STATED ON THE COVER OF THIS QUE	ELOPE PROVIDED TO NEIL M. OR	

APPENDIX 1 (viii)

PAGE 7

#### **APPENDIX 2**

## **NORMATIVE DATA PERTAINING TO:**

(1) COPING ORIENTATIONS TO PROBLEMS EXPERIENCED SCALE (CARVER *ET AL.*, 1989)

(2) REVISED UCLA LONELINESS SCALE (RUSSELL *ET AL.*, 1980)

(3) HOPELESSNESS AND JOY (LEVENSTEIN *ET AL.*, 1993)

#### **TABLE 2-A**

# COPE SUBSCALES: ITEMS LISTED BY A PRIORI SCALE ASSIGNMENT, WITH LOADINGS ON THE FACTOR TO WHICH EACH ITEM PERTAINS

(Carver, Scheier & Weintraub, 1989)

Scale name and items	Loading
1. SUBSCALE: ACTIVE COPING	
I take additional action to try to get rid of the problem.	.42
I concentrate my efforts on doing something about it.	.37
I do what has to be done, one step at a time.	.33
I take direct action to get around the problem.	.29
2. SUBSCALE: PLANNING	
I try to come up with a strategy about what to do.	.73
I make a plan of action.	.68
I think hard about what steps to take.	.53
think abut how I might best handle the problem.	.49
3. SUBSCALE: SUPPRESSION OF COMPETING ACTIVITIES	
put aside other activities in order to concentrate on this.	.68
focus on dealing with this problem, and if necessary	
et other things slide a little.	.55
keep myself from getting distracted by other thoughts	
or activities.	.51
try hard to prevent other things from interfering	
with my efforts at dealing with this.	.48
S. SUBSCALE: RESTRAINT COPING	
force myself to wait for the right time to do something.	.71
hold off doing anything about it until the	
situation permits.	.67
make sure not to make matters worse by acting too soon.	.62
restrain myself from doing anything too quickly.	.40
5. SUBSCALE: SEEKING SOCIAL SUPPORT FOR INSTRUMENTAL REASONS	
ask people who have had similar experiences what they did.	.66
try to get advice from someone about what to do.	.65
talk to someone to find out more about the situation.	.60
talk to someone who could do something concrete about	
the problem.	.55

(Table 2-A is continued on the next page...)

Scale name and items	Loading
	,
6. SUBSCALE: SEEKING EMOTIONAL SUPPORT FOR EMOTIONAL REASONS	
I talk to someone about how I feel.	.71
I try to get emotional support from friends or relatives.	.71 .69
I discuss my feelings with someone. I get sympathy and understanding from someone.	.69 .58
rger cympanty and analog new control of	
7. SUBSCALE: POSITIVE REINTERPRETATION & GROWTH	
I look for something good in what is happening.	.75
I try to see it in a different light, to make it seem	
more positive.	.59 .23
I learn from the experience. I try to grow as a person as a result of the experience.	.19
, in the grow as a possess as a section of the expensioner.	
8. SUBSCALE: ACCEPTANCE	
I learn to live with it.	.68
I accept that this has happened and that it can't be changed.	.60
I get used to the idea that it happened.	.43 .38
I accept the reality of the fact that it happened.	.30
9. SUBSCALE: TURNING TO RELIGION	
I seek God's help.	.95
I put my trust in God.	.88
I try to find comfort in my religion.	.84
I pray more than usual.	.81
10. SUBSCALE: FOCUS ON & VENTING OF EMOTIONS	
I get upset and let my emotions out.	.79
l let my feelings out.	.76
I feel a lot of distress and I find myself expressing those feelings a lot.	.57
I get upset, and am really aware of it.	.45
11. SUBSCALE: DENIAL	
I refuse to believe that it has happened.	.75
I pretend that it hasn't really happened.	.72
l act as though it hasn't even happened.	.52
I say to myself "this isn't real".	.46

Scale name and items	Loading		
12. SUBSCALE: BEHAVIOURAL DISENGAGEMENT			
I give up the attempt to get what I want. I just give up trying to reach my goal. I admit to myself that I can't deal with it and quit trying. I reduce the amount of effort I'm putting into solving the problem.	.49 .42 .37 .30		
13. SUBSCALE: MENTAL DISENGAGEMENT			
I turn to work or other substitute activities to take mymind off things. I go to movies or watch TV, to think about it less. I daydream about things other than this. I sleep more than usual.	.45 .43 .28 .23		
14. SUBSCALE: ALCOHOL-DRUG DISENGAGEMENT			
drink alcohol or take drugs, in order to think about it less.	n/a		

<sup>\*</sup> From: Carver, Scheier & Weintraub (1989), Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology*, 1989, Vol. *56*, no.2, pp.272. Copyright 1989 by American Psychological Association.

TABLE 2-B

#### CORRELATIONS AMONG DISPOSITIONAL COPE SCALE, COMPUTED AS UNWEIGHTED SUMS OF THE ITEMS COMPOSING EACH SCALE (N = 978)

\*Note: Subscales numbers accord with those in Table 2-A

			(Al	DAPT	IVE)	**********					(MALADAPTI	VE)		
COPE SUBSCA	ALES	1	2	3	4	5	6	7	8	9	10 11	12	13	14
(ADAPTIVE)		<del></del>												
Active C	1	-	.67	.45	.31	.36	.19	.43	.19	.13	.0711	28	06	10
Planning	2		-	.44	.36	.34	.20	.45	.23	.16	.1014	28	04	10
Suppr.Comp.	3			-	.30	.23	.14	.24	.13	.10	.13 .05	.02	.05	.04
Restraint	4				-	.17	.05	.37	.21	.25	0401	.00	.07	07
SocSupinstr	5					-	.69	.28	.17	.14	.39 .03	02	.20	.02
SocSupEmot	6						-	.17	.14	.13	.56 .06	.05	.21	.02
PosReintpr	7							-	.36	.21	.0215	24	.06	14
Acceptance	8								-	.07	.0321	05	.06	02
Religion	9									-	.09 .11	.07	.06	.00
(MALADAPTIV Vent Emot	<b>E)</b>										16	.17	.22	.13
Denial	11										-	.45		.17
BehDiseng.	12											-	.29	.26
MentDiseng.	13												-	.18
Alc-Drug	14													-

Due to sample size of N = 978, all correlations creater than 0.09 are significant at 0.01 level.

<sup>\*</sup> Adapted from: Carver, Scheier & Weintraub (1989), Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology*, 1989, Vol. 56, no.2, p.273. Copyright 1989 by American Psychological Association.

TABLE 2-C

CONVERGENT AND DISCRIMINANT VALIDITY OF THE COPE

COPE SUBSCALE	Opti- <sup>1</sup> mism	Con- <sup>2</sup> trol	Self- <sup>3</sup> Esteem	Hard- <sup>4</sup> iness	Type <sup>5</sup> A	Internality <sup>6</sup>
A: ADAPTIVE METHODS						
(Problem-Focused Methods)						
1. Active coping	++	++	++	++	++	+
<ul><li>2. Planning</li><li>3. Suppression of competing</li></ul>	++	++	++	+	++	ns
activities	ns	ns	ns	ns	+	ns
<ul><li>4. Restraint Coping</li><li>5. Seeking social support</li></ul>	++	ns	ns	ns	ns	ns
(Instrumental reasons)	+	ns	ns	ns	ns	ns
(Emotion-Focused Methods)  6. Seeking social support				4		
(Emotional reasons) 7. Positive Reinterpretation	ns	ns	ns	ns	ns	ns
and growth	++	++	+	++	ns	ns
8. Acceptance	++	ns	ns	ns	ns	ns
9. Turning to religion	++	ns	ns	ns	ns	ns
B. MALADAPTIVE METHODS						
(Emotion-Focused Methods) 10. Focus on & venting						
of emotions	-	·	ns	ns	+	-
11. Denial				-	ns	ns
(Disengagement)						•
12. Behavioral Disengagement	-		-	-	_	ns
13. Mental Disengagement			ns	ns	ns	ns
14. Alcohol-Drug Disengagement	-	ns	ns	ns	-	ns

COPE SUBSCALE	Monitoring <sup>7</sup>	Blunting <sup>8</sup>	Anxiety <sup>9</sup>	Social Desirability <sup>10</sup>	
A: ADAPTIVE METHODS		·			
(Problem-Focused Methods)					
1. Active coping	ns	ns		ns	
	ns	ns <sup>-</sup>	ns	ns	
3. Suppression of competing					
activities	ns	ns	ns	ns	
4. Restraint Coping	ns	ns	-	ns	
5. Seeking social support					
(Instrumental reasons)	+	ns	ns	ns	
(Emotion-Focused Methods)					
6. Seeking social support					
(Emotional reasons)	ns	ns	ns	ns	
7. Positive Reinterpretation					
	าร	ns		++	
	าร	ns	ns	ns	
9. Turning to religion	++	ns	ns	ns	
B. MALADAPTIVE METHODS					
(Emotion-Focused Methods)					
10. Focus on & venting					
of emotions -	++	ns	++	-	
11. Denial r	าร	ns	++	ns	
(Disengagement)					
12. Behavioral Disengagem	<b>+</b>	ns	++		
	ns	ns	++	ns	
	าร	ns	ns		

#### Instruments used:

- 1 = Life Orientation Test (Scheier & carver, 1985)
- 2 = (Single item regarding perceived ability to control in stressful situation)
- 3 = Self-Esteem Scale (Rosenberg, 1985)
- 4 = Personal Views Survey (Hardiness Institute, 1985)
- 5 = Student version of Jenkins Activity Survey (Krantz, Glass & Schneider, 1974; Glass, 1977)
- 6 = Rotter's Internal-External Locus of Control Scale (Rotter, 1966)
- 7 = Miller Behavioral Style Scale (Miller, 1987)
- 8 = Miller Behavioral Style Scale (Miller, 1987)
- 9 = Trait portion of State-Trait Anxiety Inventory (Spielberger et al., 1970)
- 10 = Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1964).

<sup>++</sup> = p < 0.01, positive correlation --

<sup>=</sup> p < 0.01, negative correlation

<sup>=</sup> p > 0.05, no significant correlation (All tests were two-tailed) ns

<sup>\*</sup> Adapted from: Carver, Scheier & Weintraub (1989), Assessing coping strategies: A theoretically based approach. Journal of Personality and Social Psychology, 1989, Vol. 56, no.2, p.276. Copyright 1989 by American Psychological Association.

**TABLE 2-D** 

# INTERNAL CONSISTENCY, TEST-RETEST RELIABILITY, AND NORMS OF COPE

NITERNAL   CONSISTENCY   RELST   STUDENTS   STUDENTS   COLLEGE   STUDENTS   STUDENTS   Consistency   Consistency							
Planning .80 .63 12.58 2.66 12.3 2.4 13.4 2.2  Suppression of competing activities .68 .46 9.92 2.42 9.3 2.2 9.2 2.0  Restraint coping .72 .51 10.28 2.53 10.3 2.4 10.8 2.7  Seeking social support - instrumental .75 .64 11.50 2.88 10.9 2.6 12.4 2.1  Seeking social support - emotional .85 .77 11.01 3.46 11.0 3.0 11.6 2.9  Positive reinterpretation and growth .68 .48 12.40 2.42 13.3 1.9 13.2 2.0  Acceptance .65 .63 11.84 2.56 11.8 2.4 10.8 2.7  Turning to religion .92 .86 8.82 4.10 9.3 4.1 7.9 4.7  Focus on & venting of emotions .77 .69 10.17 3.08 10.1 2.4 10.3 2.8  Denial .71 .54 6.07 2.37 5.9 2.6 4.6 0.9  Behavioral Disengage63 .66 6.11 2.07 6.2 1.9 5.8 2.2  Mental Disengagem45 .58 9.66 2.46 9.8 2.4 7.8 2.3	COPE SUBSCALE	CONSISTENCY (Cronbach's Alpha)	RETEST REL. (r)	STUDENTS (n = 1030)	(n = 44)	(n = 13)	
Suppression of competing activities .68 .46 9.92 2.42 9.3 22 9.2 2.0  Restraint coping .72 .51 10.28 2.53 10.3 2.4 10.8 2.7  Seeking social support - instrumental .75 .64 11.50 2.88 10.9 2.6 12.4 2.1  Seeking social support - emotional .85 .77 11.01 3.46 11.0 3.0 11.6 2.9  Positive reinterpretation and growth .68 .48 12.40 2.42 13.3 1.9 13.2 2.0  Acceptance .65 .63 11.84 2.56 11.8 2.4 10.8 2.7  Turning to religion .92 .86 8.82 4.10 9.3 4.1 7.9 4.7  Focus on & venting of emotions .77 .69 10.17 3.08 10.1 2.4 10.3 2.8  Denial .71 .54 .607 2.37 5.9 2.6 4.6 0.9  Behavioral Disengage63 .66 6.11 2.07 6.2 1.9 5.8 2.2  Mental Disengagem45 .58 9.66 2.46 9.8 2.4 7.8 2.3  Alcohol-Drug Disengagement	Active coping	.62	.56	11.89 2.26	11.8 2.3	12.8 1.8	
Competing activities 68 .46 9.92 2.42 9.3 2.2 9.2 2.0  Restraint coping .72 .51 10.28 2.53 10.3 2.4 10.8 2.7  Seeking social support - instrumental .75 .64 11.50 2.88 10.9 2.6 12.4 2.1  Seeking social support - emotional .85 .77 11.01 3.46 11.0 3.0 11.6 2.9  Positive reinterpretation and growth .68 .48 12.40 2.42 13.3 1.9 13.2 2.0  Acceptance .65 .63 11.84 2.56 11.8 2.4 10.8 2.7  Turning to religion .92 .86 8.82 4.10 9.3 4.1 7.9 4.7  Focus on & venting of emotions .77 .69 10.17 3.08 10.1 2.4 10.3 2.8  Denial .71 .54 6.07 2.37 5.9 2.6 4.6 0.9  Behavioral Disengage63 .66 6.11 2.07 6.2 1.9 5.8 2.2  Mental Disengagement	Planning	.80	.63	12.58 2.66	12.3 2.4	13.4 2.2	
Seeking social support       .75       .64       11.50 2.88       10.9 2.6       12.4 2.1         Seeking social support       .85       .77       11.01 3.46       11.0 3.0       11.6 2.9         Positive reinterpretation and growth       .68       .48       12.40 2.42       13.3 1.9       13.2 2.0         Acceptance       .65       .63       11.84 2.56       11.8 2.4       10.8 2.7         Turning to religion       .92       .86       8.82 4.10       9.3 4.1       7.9 4.7         Focus on & venting of emotions       .77       .69       10.17 3.08       10.1 2.4       10.3 2.8         Denial       .71       .54       6.07 2.37       5.9 2.6       4.6 0.9         Behavioral Disengage       .63       .66       6.11 2.07       6.2 1.9       5.8 2.2         Mental Disengagement       .45       .58       9.66 2.46       9.8 2.4       7.8 2.3		.68	.46	9.92 2.42	9.3 2.2	9.2 2.0	
- instrumental .75 .64 11.50 2.88 10.9 2.6 12.4 2.1  Seeking social support - emotional .85 .77 11.01 3.46 11.0 3.0 11.6 2.9  Positive reinterpretation and growth .68 .48 12.40 2.42 13.3 1.9 13.2 2.0  Acceptance .65 .63 11.84 2.56 11.8 2.4 10.8 2.7  Turning to religion .92 .86 8.82 4.10 9.3 4.1 7.9 4.7  Focus on & venting of emotions .77 .69 10.17 3.08 10.1 2.4 10.3 2.8  Denial .71 .54 6.07 2.37 5.9 2.6 4.6 0.9  Behavioral Disengage63 .66 6.11 2.07 6.2 1.9 5.8 2.2  Mental Disengagem45 .58 9.66 2.46 9.8 2.4 7.8 2.3  Alcohol-Drug Disengagement	Restraint coping	.72	.51	10.28 2.53	10.3 2.4	10.8 2.7	
Positive reinterpretation and growth .68 .48 12.40 2.42 13.3 1.9 13.2 2.0  Acceptance .65 .63 11.84 2.56 11.8 2.4 10.8 2.7  Turning to religion .92 .86 8.82 4.10 9.3 4.1 7.9 4.7  Focus on & venting of emotions .77 .69 10.17 3.08 10.1 2.4 10.3 2.8  Denial .71 .54 6.07 2.37 5.9 2.6 4.6 0.9  Behavioral Disengage63 .66 6.11 2.07 6.2 1.9 5.8 2.2  Mental Disengagem45 .58 9.66 2.46 9.8 2.4 7.8 2.3  Alcohol-Drug Disengagement		.75	.64	11.50 2.88	10.9 2.6	12.4 2.1	
Acceptance .65 .63 .11.84 2.56 .11.8 2.4 .10.8 2.7  Turning to religion .92 .86 .8.82 4.10 .9.3 4.1 .7.9 4.7  Focus on & venting of emotions .77 .69 .10.17 3.08 .10.1 2.4 .10.3 2.8  Denial .71 .54 .6.07 2.37 .5.9 2.6 .4.6 0.9  Behavioral Disengage63 .66 .6.11 2.07 .6.2 1.9 .5.8 2.2  Mental Disengagem45 .58 .9.66 2.46 .9.8 2.4 .7.8 2.3  Alcohol-Drug Disengagement		.85	.77	11.01 3.46	11.0 3.0	11.6 2.9	
Turning to religion .92 .86 8.82 4.10 9.3 4.1 7.9 4.7  Focus on & venting of emotions .77 .69 10.17 3.08 10.1 2.4 10.3 2.8  Denial .71 .54 6.07 2.37 5.9 2.6 4.6 0.9  Behavioral Disengage63 .66 6.11 2.07 6.2 1.9 5.8 2.2  Mental Disengagem45 .58 9.66 2.46 9.8 2.4 7.8 2.3  Alcohol-Drug Disengagement		.68	.48	12.40 2.42	13.3 1.9	13.2 2.0	
Focus on & venting of emotions .77 .69 10.17 3.08 10.1 2.4 10.3 2.8  Denial .71 .54 6.07 2.37 5.9 2.6 4.6 0.9  Behavioral Disengage63 .66 6.11 2.07 6.2 1.9 5.8 2.2  Mental Disengagem45 .58 9.66 2.46 9.8 2.4 7.8 2.3  Alcohol-Drug Disengagement	Acceptance	.65	.63	11.84 2.56	11.8 2.4	10.8 2.7	
emotions .77 .69 10.17 3.08 10.1 2.4 10.3 2.8  Denial .71 .54 6.07 2.37 5.9 2.6 4.6 0.9  Behavioral Disengage63 .66 6.11 2.07 6.2 1.9 5.8 2.2  Mental Disengagem45 .58 9.66 2.46 9.8 2.4 7.8 2.3  Alcohol-Drug Disengagement	Turning to religion	.92	.86	8.82 4.10	9.3 4.1	7.9 <b>4</b> .7	
Behavioral Disengage63 .66 6.11 2.07 6.2 1.9 5.8 2.2  Mental Disengagem45 .58 9.66 2.46 9.8 2.4 7.8 2.3  Alcohol-Drug Disengagement		.77	.69	10.17 3.08	10.1 2.4	10.3 2.8	
Mental Disengagem45 .58 9.66 2.46 9.8 2.4 7.8 2.3  Alcohol-Drug  Disengagement	Denial	.71	.54	6.07 2.37	5.9 2.6	4.6 0.9	
Alcohol-Drug Disengagement	Behavioral Disengage.	.63	.66	6.11 2.07	6.2 1.9	5.8 2.2	
Disengagement	Mental Disengagem.	.45	.58	9.66 2.46	9.8 2.4	7.8 2.3	
	Disengagement	n/a	.57	1.38 0.75			

Items are rated on a scale from 1 to 4, with a maximum score of 16 and a minimum of 4 per subscale, except for Alcohol-Drug Disengagement.

#### Sources:

Copyright 1990 by Harwood Academic Publishers GmbH

<sup>1 =</sup> Carver, C.S., Scheier, M.F., & Weintraub, J.K. (1989). Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology*, 56(2), 267-283. Copyright 1989 by American Psychological Association.

<sup>2 =</sup> Blaney, N.T., Millon, C., Morgan, R., Eisdorfer, C., & Szapocznik, J. (1990). Emotional distress, stress-related disruption and coping among healthy HIV-positive gay males. Psychology and Health, 4, 259-273.

# TABLE 2-E ORIGINAL INSTRUCTIONS AND RATING SCALES FOR COPE

(Carver, Scheier & Weintraub, 1989, p.271)

#### **INSTRUCTIONS TO RESPONDENTS:**

"We are interested in how people respond when they confront difficult or stressful events in their lives. There are lots of ways to try to deal with stress. This questionnaire asks you to indicate what you generally do and feel, when you experience stressful events. Obviously, different events bring out somewhat different responses, but think about what you usually do when you are under a lot of stress".

#### **RESPONSE CHOICES:**

- 1 = I usually don't do this at all
- 2 = I usually do this a bit
- 3 = I usually do this a medium amount
- 4 = I usually do this a lot

# TABLE 2-F MODIFIED INSTRUCTIONS AND RATING SCALES FOR COPE

(Carver, Scheier & Weintraub, 1989, p.271)

#### **INSTRUCTIONS TO RESPONDENTS:**

"The following section concerns the many ways in which people respond when they confront difficult or stressful events in their lives. You are asked to indicate what you GENERALLY do and feel, when you experience stressful events".

#### **RESPONSE CHOICES:**

1 = Not at all	l usually don't do this at all
2 = Not often	don't usually do this except every now and again
3 = Often	often do this, but not all the time
4 = Most of the time I	almost always do this

**APPENDIX 2 TABLE 2-F** 

#### TABLE 2-G

#### **TEST SEQUENCE OF COPE ITEMS**

1.	I talk to someone about how I feel.
2.	I make a plan of action.
3.	I talk to someone who could do something concrete about the problem.
4.	I turn to work or other substitute activities to take my mind off things.
5.	I just give up trying to reach my goal.
6.	I go to movies or watch TV, to think about It less.
7.	I drink alcohol or take drugs, in order to think about it less.
8.	I keep myself from getting distracted by other thoughts or activities.
9.	I think about how I might best handle the problem.
10.	i accept the reality of the ract that it happened.
11.	I force myself to wait for the right time to do something.
12.	I admit to myself that I can't deal with it, and quit trying.
13.	I look for something good in what is happening.
14.	I pretend it hasn't really happened.
15.	I hold off doing anything about it until the situation permits.
16.	I try hard to prevent other things from interfering with my efforts at dealing with this.
17.	I think hard about what steps to take.
18.	I refuse to believe that it has happened.
19.	get upset and let my emotions out.
20.	I take additional action to try to get rid of the problem.
21.	I learn to live with it.
22.	I restrain myself from doing anything too quickly.
23.	I get upset, and I am really aware of it.
24.	I try to get emotional support from friends or relatives.
25.	I ask people who have had similar experiences what they did.
26.	I do what has to be done, one step at a time.
27.	I learn something from the experience.
28.	I try to come up with a strategy about what to do.
29.	l act as though it hasn't even happened.
30.	I try to find comfort in my religion.
31.	I take direct action to get around the problem.

(Table 2-G is continued on the next page...)

#### (Table 2-G is continued from the previous page)

- 32. I focus on dealing with this problem, and if necessary let other things slide a little.
- 33. I make sure not to make matters worse by acting too soon.
- 34. I seek God's help.
- 35. I get sympathy and understanding from someone.
- 36. I say to myself "this isn't real".
- 37. I accept that this has happened and that it can't be changed.
- 38. I pray more than usual.
- 39. I let my feelings out.
- 40. I sleep more than usual.
- 41. I try to get advice from someone about what to do.
- 42. I try to see it in a different light, to make it seem more positive.
- 43. I reduce the amount of effort I'm putting into solving the problem.
- 44. I feel a lot of emotional distress and I find myself expressing those feelings a lot.
- 45. I daydream about things other than this.
- 46. I talk to someone to find out more about the situation.
- 47. I put my trust in God.
- 48. I put aside other activities in order to concentrate on this.
- 49. I give up on the attempt to get what I want.
- 50. I discuss my feelings with someone.
- 51. I concentrate my efforts on doing something about it.
- 52. I try to grow as a person as a result of the experience.
- 53. I get used to the idea that it happened.

TABLE 2-H
SCORING PROCEDURE FOR COPE SCALES

SCALE:	ITEM	IS TO BE	ADDEC	FOR SCALE
ACTIVE COPING	20	26	31	51
PLANNING	2	9	17	28
SUPPRESSION OF COMPETING ACTIVITIES	8	16	32	48
RESTRAINT COPING	11	15	22	33
SEEKING SOCIAL SUPPORT FOR				
INSTRUMENTAL REASONS	3	25	41	46
SEEKING SOCIAL SUPPORT FOR				
EMOTIONAL REASONS	1	24	35	50
POSITIVE REINTERPRETATION & GROWTH	13	27	42	52
ACCEPTANCE	10	21	37	53
TURNING TO RELIGION	30	34	38	47
FOCUS ON & VENTING OF EMOTIONS	19	23	39	44
DENIAL	14	18	29	36
BEHAVIOURAL DISENGAGEMENT	5	12	43	49
MENTAL DISENGAGEMENT	4	6	40	45
ALCOHOL-DRUG DISENGAGEMENT	7			

<sup>\*</sup> NOTE: ALL ITEMS ARE SCORED IN THE SAME DIRECTION.

Adapted from Carver, Scheier, & Weintraub (1989), Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology*, 1989, Vol. 56 (2), p.272. Copyright 1989 by American Psychological Association.

#### TABLE 2-I

#### THE SURVEY FORM OF THE REVISED UCLA LONELINESS SCALE

(Russell, Peplau & Cutrona, 1980)

#### instructions:

\*Directions: Indicate how often you feel the way described in each of the following statements. Circle one number for each."

Statement	Never	Rarely	Some- times	Offen
I feel in tune with     the people around me.	1	2	3	4
13. No one really knows me well.	1	2	3	4
15. I can find companionship when I want it.	1	2	3	4
<ol><li>People are around me but not with me.</li></ol>	1	2	3	4

Note 1:

Item numbers reflect those of the 20-item scale version.

Note 2:

These items (2 positively worded and 2 negatively worded)

are those items specifically identified by Russell,

Peplau and Cutrona (1980) as being the best Items for a

short survey form.

Note 3:

Items 1 & 15 are reverse scored (1=4, 2=3, 3=2, 4=1)

Source: Russell, D., Peplau, L., & Cutrona, C. (1980). The Revised UCLA Loneliness Scale: Concurrent and Discriminant validity evidence. *Journal of Personality and Social Psychology, 39*(3), 472-480. Copyright 1982 by the American Psychological Association.

# TABLE 2-J UCLA LONELINESS SURVEY FORM NORMATIVE MEANS AND STANDARD DEVIATIONS FOR DIFFERENT AGE GROUPS

Number of respondents	Mean Ioneliness St.	Deviation	<del></del>	
149	8.31	2.02		
94	8.17	1.97		
53	7.51	1.88		
52	7.86	2.32		
34	7.26	2.63		
	149 94 53 52	149 8.31 94 8.17 53 7.51 52 7.86	94     8.17     1.97       53     7.51     1.88       52     7.86     2.32	149     8.31     2.02       94     8.17     1.97       53     7.51     1.88       52     7.86     2.32

4-Items per respondent, rated from 1 to 4

Minimum score = 4; Maximum Score = 16

Significant differences between age groups were found: (F = 2.80; df = 4,377; p < 0.05) Significant correlation between loneliness and age was also found: (r = -0.17, p < 0.001)

Source: Russell, D. (1982). The measurement of Loneliness. In L.A. Peplau and D. Perlman (Eds.), Loneliness: A sourcebook of current theory, research and therapy, Chap.6. New York: John Wiley & Sons. Copyright 1982 by John Wiley & Sons, Ltd.

#### **TABLE 2-K**

# MODIFIED INSTRUCTIONS AND RATING SCALES FOR UCLA LONELINESS SCALE (SURVEY FORM)

"For each of the following sentences, circle the number that best describes how often it applies to you in general, DURING THE LAST YEAR OR SO. Circle only one number for each item".

2

Almost never

1

3

4

Often

#### **TABLE 2-L**

# SINGLE ITEMS FROM LEVENSTEIN *ET AL*.'S (1993) PERCEIVED STRESS QUESTIONNAIRE

7.	You feel you're doing things you really like.	(JOY)
9.	You fear you may not manage to attain your goals.	(WORRIES)
21.	You enjoy yourself.	(YOV)
22.	You are afraid for the future.	(WORRIES)

Item numbers refer to the original PSQ item numbers.

Source: Levenstein *et al.* (1993). Development of the Perceived Stress Questionnaire: A new tool for psychometric rsearch. *Journal of Psychosomatic Research, 37*(1), 19-32. Copyright 1992 Pergamon Press.

#### TABLE 2-M

# FACTOR LOADINGS AND RELATIONS OF PSQ ITEMS TO OTHER CONSTRUCTS

PSQ ITEM	FACTOR 1 Lack of Joy	Sas 2 Worries	<b>RELATIONS</b> 1 Trait Anxiety <sup>ac</sup>	2 Self- reported Stress <sup>b</sup>	3 Perceived Stress <sup>bd</sup>	
	r	r	r	r	r	
You feel you're doing things you really like.	-0.150		-0.41 *	-0.29 <b>*</b>	-0.46 ***	T
You enjoy yourself.	-0.460		-0.25	-0.35 **	-0.40 ***	
You fear you may not manage to attain your goals.		+0.300	+0.40 *	+0.29 *	+0.53 ***	
You are afraid for the future.	•	+0.500	+0.43 *	+0.23	+0.53 ***	

#### SUPERSCRIPTS:

a = Correlation with General PSQ format

(ie., perceived stress over the last year or so)

b = Correlation with Recent PSQ format

(le., perceived stress during the last month).

c = State-Trait Anxiety Inventory

(Spiellberger, Gorsuch & Lushene, in Levenstein et al., 1993)

d = Perceived Stress Scale

(Cohen, Karmarck & Mermelstein, 1983, in Levenstein et al., 1993)

e = Interfactor correlations were r < 0.40;

Factors have eigenvalues greater than 1.

- p < 0.05
- = p < 0.01
- = p < 0.001

Source: Levenstein *et al.* (1993). Development of the Perceived Stress Questionnaire: A new tool for psychometric rsearch. *Journal of Psychosomatic Research, 37*(1), 19-32. Copyright 1992 Pergamon Press.

#### **TABLE 2-N**

#### **MODIFIED PSQ ITEMS**

ПЕМ		CONSTRUCT
1.	I feel I am doing things I really like.	(PRESENT-TIME JOY)
2.	I enjoy myself.	(PRESENT-TIME JOY)
3.	I fear I may not manage to attain my goals.	(FUTURE-RELATED PESSIMISM)
4.	I am afraid for the future.	(FUTURE-RELATED PESSIMISM)

#### **APPENDIX 3**

# NORMATIVE DATA PERTAINING TO McNAIR, LORR & DROPPLEMAN'S (1971) PROFILE OF MOOD STATES (POMS)

#### TABLE 3-A

# PROFILE OF MOODS STATES: INTER-SCALE CORRELATIONS

#### MALE UNDERGRADUATES (N = 113)

:		TA	DD	АН	VA	FI	
T.	A	*					
C	D	+.56	•				
A	ΝΗ	+.50	+.70	*			
V	<b>/</b> A ,	15	32	12	*		
FI	I	+.33	+.61	+.46	43	•	

r (crit) = 0.184, df = 111, 2-tailed, t = 1.960, alpha = 5 %

TA = TENSION-ANXIETY SCALE

DD = DEPRESSION-DEJECTION SCALE

AH = ANGER-HOSTILITY SCALE
VA = VIGOUR-ACTIVITY SCALE
FI = FATIGUE-INERTIA SCALE

CB = CONFUSION-BEWILDERMENT SCALE

Source: McNair, Lorr & Droppleman (1971). EITS Manual for the Profile of Mood States. Educational and Industrial Testing Service.

Ca.: San Diego, p.9.

TABLE 3-B

# PROFILE OF MOOD STATES: INTERNAL CONSISTENCY (K-R 20) OF SCALES

FACTOR	STUDY A	STUDY B
Tension-Anxiety	0.92	0.90
Depression-Dejection	0.95	0.95
Anger-Hostility	0.92	0.93
Vigour-Activity	0.89	0.87
Fatigue-Inertia	0.94	0.93
Confusion-Bewilderment	0.87	0.84

\* STUDY A: 350 MALE PSYCHIATRIC OUTPATIENTS

\* STUDY B: 650 FEMALE PSYCHIATRIC OUTPATIENTS

Source: McNair, Lorr & Droppleman (1971). *EITS Manual for the Profile of Mood States.* Educational and Industrial Testing Service.

Ca.: San Diego, p.10.

TABLE 3-C

# PROFILE OF MOOD STATES: TEST-RETEST RELIABILITY (r<sub>11</sub>)

#### PSYCHIATRIC OUTPATIENTS (N = 100)

	FACTOR	INTAKE TO PRETHERAPY	INTAKE TO SIX WEEKS
	TA	0.70	0.51
	DD	0.74	0.47
	АН	0.71	0.53
,	VA	0.65	0.43
f	FI	0.66	0.45
	СВ	0.68	0.52

TA = TENSION-ANXIETY SCALE

DD = DEPRESSION-DEJECTION SCALE

AH = ANGER-HOSTILITY SCALE
VA = VIGOUR-ACTIVITY SCALE
FI = FATIGUE-INERTIA SCALE

CB = CONFUSION-BEWILDERMENT SCALE

Source: McNair, Lorr & Droppleman (1971). *EITS Manual for the Profile of Mood States.* Educational and Industrial Testing Service. Ca.: San Diego, p. 10.

#### TABLE 3-D

# PROFILE OF MOOD STATES: ASSOCIATIONS WITH SOCIAL DESIRABILITY

CORRELATIONS WITH THE CROWNE AND MARLOWE (1960) MEASURE OF SOCIAL DESIRABILITY (EDWARDS, 1957; IN McNAIR *ET AL.*, 1971):

(N = 150 Male Outpatients)

 	ſ	
TA	-0.21	
DD	-0.36	
АН	-0.52	
VA	+0.33	
FI	-0.18	

TA = TENSION-ANXIETY SCALE

DD = DEPRESSION-DEJECTION SCALE

AH = ANGER-HOSTILITY SCALE VA = VIGOUR-ACTIVITY SCALE FI = FATIGUE-INERTIA SCALE

CB = CONFUSION-BEWILDERMENT SCALE

Source: McNair, Lorr & Droppleman (1971). *EITS Manual for the Profile of Mood States*. Educational and Industrial Testing Service. Ca.: San Diego, p. 15.

#### TABLE 3-E

# PROFILE OF MOOD STATES: SCORING PROCEDURE

'To obtain a score for each mood factor, the sum of the responses is obtained for the adjectives defining the factor. All Items defined In each factor are keyed in the same direction except for two Items, 'Relaxed' in the Tension-Anxiety Scale and 'Efficient' in the Confusion Scale. These Items receive negative weights in calculating the factor scores. A Total Mood Disturbance Score may be obtained by summing the scores (with Vigor weighted negatively) on the six primary mood factors'. (McNair et al., 1971, p.6).

<b>~</b> ^	$\sim$	_	$\overline{}$	
FΑ	$\mathbf{C}$	$\cup$	ĸ	

**ITEMS** 

Tension-Anxiety

Tense; Shaky; On edge; Panicky;

Relaxed; Uneasy; Restless; Nervous; Anxious

**Depression** 

Unhappy; Sorry; Sad; Blue; Hopeless; Unworthy; Discouraged; Lonely; Miserable; Gloomy;

Desperate; Helpless; Worthless; Terrified; Guilty

Anger-Hostility

Angry; Peeved; Grouchy; Spiteful; Annoyed;

Resentful; Bitter; Ready to fight; Rebellious;

Deceived; Furious; Bad-tempered

Vigour-Activity

Lively; Active; Energetic; Cheerful; Alert;

Full of pep; Carefree; Vigorous

Fatigue-Inertia

Wom-out; Listless; Fatigued; Exhausted; Sluggish;

Weary; Bushed

Confusion

Confused; Unable to concentrate; Muddled;

Bewildered; Efficient; Forgetful; Uncertain about

things

Source: McNair, Lorr & Droppleman (1971). EITS Manual for the Profile of Mood

States. Educational and Industrial Testing Service. Ca.: San Diego, pp.6-8.

TABLE 3-F

PROFILE OF MOOD STATES:

COLLEGE MALE NORMS (N = 340)

FACT	OR MEAI	N ST.DEV	
TA	12.9	06.8	
DD	13.1	10.5	·
АН	10.1	07.8	
VA	15.6	06.0	
FI	10.4	06.2	
СВ	10.2	05.2	
		٠	

TA = TENSION-ANXIETY SCALE

DD = DEPRESSION-DEJECTION SCALE

AH = ANGER-HOSTILITY SCALE
VA = VIGOUR-ACTIVITY SCALE
FI = FATIGUE-INERTIA SCALE

CB = CONFUSION-BEWILDERMENT SCALE

Source: McNair, Lorr & Droppleman (1971). EITS Manual for the Profile of Mood States. Educational and Industrial Testing Service.

Ca.: San Diego, p.20.

### **APPENDIX 4**

## CENTERS FOR DISEASE CONTROL (1987 REVISED) HIV INFECTION SYMPTOM CLASSIFICATION

THE FOLLOWING PAGES ARE QUOTED DIRECTLY FROM KIRKWOOD & LEWIS (1989, pp.111-117), AND DESCRIBE THE 1987 (REVISED) CENTERS FOR DISEASE CONTROL HIV INFECTION CLASSIFICATION SYSTEM.

IT SHOULD ALSO BE NOTED THAT SINCE THE PUBLICATION OF THIS CLASSIFICATION SYSTEM, THE SYSTEM HAS BEEN UPDATED. SPECIFICALLY, PEOPLE WITH CD4-CELL COUNTS BELOW 200 CELLS PER MICROLITRE, REGARDLESS OF SYMPTOM-STATUS, ARE NOW CLASSIFIED AS HAVING AIDS.

#### PREVIOUS (1986) CLASSIFICATION OF HIV INFECTIONS

- The seroconversion illness
- Healthy carriers
- Persistent generalized lymphadenopathy
- AIDS-related complex
- AIDS

#### CURRENT (1987, revised) CLASSIFICATION OF HIV INFECTION

\* Group I Acute infection

Group II Asymptomatic infection

\* Group III Persistent generalized lymphadenopathy

Group IV Other disease

Subgroup A Constitutional disease

Subgroup B Neurological disease

Subgroup C Secondary infectious diseases

Category C1 Specified secondary infectious diseases

listed in the CDC surveillance definition

for AIDS

Category C2 Other specified secondary infectious

diseases

Subgroup D Secondary cancers

Subgroup E Other conditions

#### **ACUTE INFECTION (CLASSIFIED AS GROUP I)**

#### Definition of acute HIV infection Group I:

A mononucleosis-like syndrome, with or without aseptic meningitis, associated with seroceonversion for HIV antibody (Antibody seroconversion is required as evidence of initial infection. Current procedures for viral isolation are not adequately sensitive to be relied on for demonstrating infection onset).

#### Symptoms and signs sometimes associated with the seroconversion illness:

- Sudden onset
- Fever and sweats
- Tiredness and malaise
- Nausea and anorexia
- Muscle and joint aches
- Headaches
- Sore throat
- Diarrhoea
- Transient rash
- Generalized lymphadenopathy
- Atypical lymphocytosis
- Seroconversion generally follows in 3 8 weeks
- Syndrome resolves spontaneously
- Lasts 3 14 days

#### ASYMPTOMATIC HIV INFECTION (GROUP II)

#### Definition of asymptomatic HIV infection Group II:

The absence of symptoms or signs of HIV infection in a patient known to be Antibody-positive. If a patient has previous symptoms or signs that would have placed him in group III or IV he cannot be replaced in Group II.

#### PERSISENT GENERALIZED LYMPHADENOPATHY (PGL. GROUP III)

#### Definition of persistent generalized lymphadenopathy (PGL):

Palpable lymphadenopathy (lymph node enlargement of 1cm or greater) at two or more extrainguinal sites persisting for more than 3 months in the absence of a concurrent illness or condition other than HIV infection to explain the findings.

#### CONSTITUTIONAL DISEASE (GROUP IV, SUBGROUP A):

#### Definition of constitutional disease Group IV subgroup A:

One or more of the following:

- Fever persisting more than one month
- Involuntary weight loss of greater than 10% of baseline
- Diarrhoea persisting more than 1 month in the absence of a concurrent illness or condition other than HIV infection to explain the findings.

#### **NEUROLOGICAL DISEASE (GROUP IV, SUBGROUP B):**

#### Definition of neurological disease Group IV subgroup B:

One or more of the following:

- Dementia
- Myelopathy
- Peripheral neuropathy

In the absence of a concurrent illness or condition other than HIV to explain the findings.

## SECONDARY INFECTIOUS DISEASES (GROUP IV, SUBGROUP C):

### Category C1 infections:

- Pneumocystis carinii pneumonia
- Chronic cryptosporidiosis
- Toxoplasmosis
- Extraintestinal strongyloidiasis
- Isosporiasis
- Candidiasis (oesophageal, bronchial or pulmonary)
- Cryptococcosis
- \* Histoplasmosis
- Infection with mycobacterium avium or mycobacterium Kansasii
- Cytomegalovirus infection
- Chronic mucocutaneous or disseminated herpes simplex virus infection
- Progressive multifocal leukoencephalopathy

#### Category C2 infections:

- Oral hairy leukoplakla
- Multidermatomal herpes zoster
- Recurrent salmonella bacteraemia
- Nocardiosis
- \* Tuberculosis
- Oral candidiasis

Category C1 infections only are included in the defition of AIDS

### SECONDARY CANCERS (GROUP IV, SUBGROUP D):

### Cancers associated with HIV infection Group IV subgroup D:

- Kaposi's sarcoma
- Non-Hodgkin's lymphoma (small, non-cleaved lymphoma or immunoblastic sarcoma
- Primary lymphoma of the brain

## OTHER CONDITIONS (GROUP IV SUBGROUP E):

This classification is used for a patient who is infected with HIV and who is suffering from some other condition that is not classified in the groups above.

The coexisting condition may clearly be associated with immune deficiency or may not. It may be a totally unrelated clinical illness and the course and management of this may be complicated by the coexistence of HIV infection.

# APPENDIX 5 RAW DATA

TABLE 5-A

DEMOGRAPHIC DESCRIPTION OF SAMPLE

Subj	Date Psych Meas	Age	Home Lang	Education	Employm. Status	income /month
22	94.1	32.9y	english	tertiary	employed	16250
16	94.0	32.9y	afrik.ns	matric	unempl.	nil
14	94.0	29.3y	english	tertiary	unempl.	9000
01	93.9	32.1y	afrik.ns	matric	employed	1300
13	94.0	29.2y	afrik.ns	matric	employed	2500
23	94.1	36.2y	english	tertiary	unempl.	15000
21	94.0	35.5y	english	matric	unempl.	nil
04	93.9	26.4y	english	<matric< td=""><td>employed</td><td>1250</td></matric<>	employed	1250
07	93.9	49.3y	english	tertiary	employed	32CO
28	94.1	35.3y	english	matric	unempi.	nii
18	94.0	33.2y	eng/af	tertiary	employed	1800
08	93.9	32.2y	afrik.ns	matric	unempl.	380
25	94.0	40.4y	afrik.ns	matric	employed	?
10	94.0	24.5y	english	matric	unempl.	nil
05	93.9	37.8y	english	matric	unempl.	2000
11	94.0	28.3ý	eng/af	tertiary	unempl.	nil
29	94.1	50.8ý	eng/o	?	unempl.	nil
15	94.0	28.9y	english	<matric< td=""><td>employed</td><td>1800</td></matric<>	employed	1800
31	94.2	32.7y	afrik.ns	matric	employed	3500
30	94.1	31.0ý	afrik.ns	matric	unempi.	729
12	94.0	47.2y	eng/af	matric	employed	2100
17	94.0	27.4y	english	?	employed	2000
20	94.0	31.9y	english	tertiary	employed	14000
02	93.9	22.3y	english	tertiary	unempl.	nil
03	93.9	26. lý	english	matric	employed	2800
19	94.0	42.9y	english	tertiary	unempl.	1500
09	94.0	20.3y	afrik.ns	matric	employed	1600

eng = English eng/af = English & Afrikaans afrik.ns = Afrikaans eng/o = English & Other

TABLE 5-B

# SYMPTOMATOLOGY & (UNADJUSTED) IMMUNE MEASURES

Subj	CDC Classif.	Symptom/ Asympt.	Date First Sympt*	CD4 Cell Count	CD8 Cell Count	Date Immune Measure
22	III	Asympt	•	1764	1900	94.0
16	ll .	Asympt	•	1321	1068	93.9
14	11	Asympt	•	821	2035	93.7
01	111	Asympt	•	634	4172	93.8
13	II	Asympt	•	562	1995	93.7
23	lVc1	Sympt	73.4	0	0	94.0
21	11	Asympt	•	823	2237	93.9
04	111	Asympt	•	464	1097	93.7
07	IVc2	Sympt	93.2	190	1350	93.7
28	IVa	Sympt	93.9	269	1109	94.1
18	IVc2	Sympt	93.4	28	320	93.9
08	IVc1	Sympt	93.3	54	1368	93.8
25	IVc1	Sympt	90.3	11	576 ~	93.6
10	11	Asympt	•	545	758	93.7
05	III	Asympt	• ,	319	684	93.7
11	IVa	Sympt	93.9	350	1009	93.7
29	IVa	Sympt	92.7	162	1253	94.1
15	11	Asympt	•	426	1434	93.7
31	lVa	Sympt	94.1	238	724	94.1
30	lVc2	Sympt	92.8	10	•	94.1
12°	IVa	Asympt	•	159	1652	93.9
17	lVc1	Sympt	93.7	94	973	93.9
20	11	Asympt	•	347	1672	93.9
02	ii	Asympt	•	663	835	93.7
03	111	Asympt	•	374	602	93.7
19	11	Asympt	•	91	293	93.9
09	 11	Asympt	•	445	1050	93.7

Sympt = Symptomatic (CDC IVa-e)
Asympt = Asymptomatic (CDC II & III)

SUPERSCRIPT ::

Excluded 'symptoms': PGL, and any previous HIV-related infection which has not been present for at least 2 weeks during the previous six months. In addition, reported symptoms were checked and verifiedagainst medical records. If reported symptoms were medically inaccurate, they have not been included in this table.

Only medically verified symptomatology is indicated in this table.

SUPERSCRIPT a:

Subject 12 is classified as IVa due to CD4-cell counts below 200.

However, he is asymptomatic.

# TABLE 5-C REPORTED TIME OF INFECTION

	FIRST		REPORTED	PERIOD OF INFE	CTION		DATE OF
	HIV+	FROM	to	RANGE	CERT-	MEAN	IMMUNE
SUBJ	TEST	(YR)	(YR)	(YRS)	AINTY	(YR)	TEST
22	83.3	82.7	83.3	0.6	7	83.0	94.0
16	?	90.3	91.1	0.8	3	90.7	93.9
14	89.8	86.8	89.8	3.0	1	88.3	93.7
1	86.0	86.1	86.3	0.2	3	86.2	93.8
13	87.8	87.2	87.2	0.0	7	87.2	93.7
23	87.3	80.7	81.8	1.1	6	81.3	94.0
21°	93.7	89.0	90.0	1.0	1^	89.5	93.9
4**	89.9	86.0	87.0	1.0	1^	86.5	93.7
7	86.3	86.2	86.3	0.1	7	86.3	93.7
28	91.9	87.1	88.0	0.9	4	87.6	94.1
18	90.7	85.6	86.0	0.4	4	85.8	93.9
В	91.4	85.0	87.0	2.0	6	86.0	93.8
25	?	86.7	87.4	0.7	2	87.1	93.6
10	91.7	<del>89</del> .5	91.3	1.8	1	90.4	93.7
5	89. <del>8</del>	89.6	89.8	0.2	7	89.7	93.7
11	90.3	89.6	90.3	0.7	7	90.0	93.7
29	90.5	89.0	90.0	1.0	1	89.5	94.1
15	90.5	90.4	90.5	0.1	1	90.5	93.7
31***	89.9	89.8	89.8	0.0	7	89.8	94.1
30	90.9	89.9	90.0	0.1	4	90.0	94.1
12	91.9	90.8	90.8	0.0	6	90.8	93.9
17	91.6	91.3	91.6	0.3	7	91.5	93.9
20	91.4	<b>92</b> .1	<b>92</b> .7	0.6	4	92.4	93.9
2	93.3	93.1	93.2	0.1	7	93.2	93.7
3	93.3	92.7	92.8	0.1	4	92.8	93.7
19	93.8	<b>92</b> .1	93.8	1.7	2	93.0	93.9
9****	93.3	93.1	93.1	0.0	1^	93.1	93.7
MEAN				0.685	4.1		
ST.DEV				0. <b>725</b>	2.4		

SUPERSCRIPT \*: Subject 21 reported no idea of time of Infection.

However, personal records revealed that this occurred in 1989.

SUPERSCRIPT \*\*: Subject 04 reported time of infection from 1984 to 1989.

However, personal records indicate that infection occurred in 1986.

SUPERSCRIPT \*\*\*: Subject 31 reports that infection occurred in December 1989.

However, he also reports that he tested positive in December 1989, which is inconsistent with the time of infection, as there is an average period of about 2 months from infection until tests indicate seropositivity, (window period). Thus, the date of infection is corrected backward by

two months.

SUPERSCRIPT \*\*\*\*: Subject 09 did not report a time of infection.

However, medical records clearly indicate seroconversion illness

diagnosis in January 1993.

SUPERSCRIPT ^: These are the levels of certainty reported by the subject.

However, due to additional information obtained from medical and personal records, these levels of certainty are not valid for the

actual period of infection as indicated for each subject.

TABLE 5-C
REPORTED TIME OF INFECTION
(Continued from previous page)

SUBJ		TIME/YEARS I	NFECTED	
	FROM	1 10	MEAN	
22	11.3	10.7	11.0	
16	3.6	2.8	3.2	
14	6.9	3.9	5.4	
01	7.7	7.5	7.6	
13	6.5	6.5	6.5	
23	13.3	12.2	12.8	
21	4.9	3.9	4.4	
04	7.7	6.7	7.2	
07	7.5	7.4	7.5	
28	7.0	6.1	6.5	
18	8.3	7.9	8.1	
08	8.8	6.8	7.8	
25	6.9	6.2	6.5	
10	4.2	2.4	3.3	
05	4.1	3.9	4.0	
11	4.1	3.4	3.8	
29	5.1	4.1	4.6	
15	3.3	3.2	3.3	
31	4.3	4.3	4.3	
30	4.2	4.1	4.1	
12	3.1	3.1	3.1	
17	26	2.3	2.5	
20	1.8	1.2	1.5	
02	0.6	0.5	0.6	
03	1.0	0.9	1.0	
19	1.8	0.1	1.0	
09	0.6	0.6	0.6	
Mean	5.230	4.544	4.887	
tDev	3.101	2.976	<b>3</b> .017	

**TABLE 5-D** 

# PERIODS OF USE OF MEDICALLY-PRESCRIBED ANTIRETROVIRALS & EXPERIMENTAL DRUGS

## MONTHS OF USAGE OF MEDICAL DRUGS UNTIL IMMUNE MEASURES

Subj	<b>ANTIRETROVIRALS</b>				EXPERIMENTAL
	AZT	DDC	DDI	D4T	DRUG TRIAL
22	•	•	•	•	•
16	•	•	•	•	5 m
14	• •	•	•	•	1 m
01	7m	•	•	•	4 m
13	•	•	•	•	•
23	48m	•	3m	•	•
21	•	•	•	•	3m
04	•	•	•	•	2m
07	36m	12m	18m	lm	•
28	•	•	•	•	•
18	16m	óm	•	•	3m
08	1m	•	4m	•	3m
25	7m	•	•	•	lm
10	•	•	•	•	2m
05	óm	•	óm	•	•
11	•	•	•	•	2m
29a	12m	•	•	•	5m
15	•	•	•	•	1m
31	•	•	•	•	•
30	•	•	•	•	•
12	lm	•	•	•	3m
17	•	•	•	•	•
20	•	•	•	•	3m
02	•	•	•	•	2m
03	•	•	•	•	2m
19	•	•	•	•	lm
09	•	•	•	•	2m

NOTE: For subjects who had immune measures done at the same time as commencing the experimental drug trial, zero (\*) use of the experimental drug is indicated.

SUPERSCRIPT a: Subject 29 commenced the experimental drug-trial in August 1993. However, despite 5 months of usage, CD4-cell counts remained constant, which is atypical of the effects of the experimental drug, as the remainder of the drug-trial participants displayed a relatively constant percentage increase in CD4-cell counts per month of usage.

TABLE 5-E

ADJUSTMENTS TO CD4-CELL COUNTS FOR ANTIRETROVIRAL & EXPERIMENTAL DRUG USAGE

SUBJ	RANKED UNADJ	BEFORE	. USE > 3 MONTHS AFTER	EXPERI- MENTAL	TOTAL CD4-CELL	FINAL ADJUSTED
	CD4 COUNT	SYMPTOMS $ADJ = -20$	SYMPTOMS ADJ = +10	DRUG ADJUST	adjust- Ment	CD4-CELL COUNTS
(A) CELL	-COUNTS > 475	5 N = 8				
(A) CELL	-COUNTS > 4/3	14-0				
22	1764	0	0	0	0	1764
16	1321	0	0	-264	-264	1057
21 14	823 821	0	0 0	-107 -39	-107 -39	716 782
2	663	ő	ŏ	- <del>6</del> 0	-60	603
ī	534	-20	ŏ	-102	-122	512
13	562	0	, O	0	0	562
10	545	0	0	-50	-50	495
MEAN	891.6				-80.3	811.4
ST.DEV	431.4				86.3	426.5
(B) CELL	-COUNTS <475	TO >200 N :	= 9			
4	464	0	0	-42	-42	422
9	445	0	0	-40	<b>-40</b>	405
15	426	0	0	-20	-20	406 340
3 11	374 350	0	0 0	-34 -32	-34 -32	318
20	347	0	Ö	-45	-45	302
5	319	-20	Ö	Õ	-20	299
28	269	0	Ō	0	0	269
31	238	. 0	0	0	0	238
MEAN	359.1			······································	-25.9	333.2
ST.DEV	77.2				17.1	65.1
(C) CELL	-COUNTS < 200	N = 10				
7	190	-20	0	0	-20	170
29	162	0	10	0	10	172
12 17	159 94	0 0	0 0	-21 0	-21 0	138
17 19	94 91	0	0	-4	- <b>4</b>	87
3	54	Ö	10	- <del>8</del>	2	56
18	28	-20	Ö	-1	-21	7
25	11	0	10 .	-1	9	20
30	10	0	0	0	0	10
23	0	-20	0	0	-20	0
MEAN	79.9				-6.5	75.4
ST.DEV	70.6				11.9	67.3
OTAL: N						
MEAN	413.5	-3.7	1.1	-32.2	-34.8	379.4
ST.DEV	409.7	7.8	3.1	54.3	56.2	381.5

APPENDIX 5 TABLE 5-E

TABLE 5-F

CALCULATING THE RATE OF CD4-CELL DECLINE FROM INFECTION UNTIL IMMUNE MEASUREMENT

SUBJ	CD4-CELL 1119-CD4adj ADJUSTED		MEAN CD4-RATE YEARS OF DECLINE INFECTED		
22	1764	-645	11.0	-58.6	
16	1057	62	3.2	19.4	
14	782	337	5.4	62.4	
1	512	607	7.6	79.9	
13	562	557	6.5	<b>85</b> .7	
23	0	1119	12.8	87.4	
21	716	403	4.4	91.6	
4	422	697	7.2	96.8	
7	170	949	7.5	126.5	
28	269	850	6.5	130.8	
18	7	1112	8.1	137.3	
8	56	1063	7.8	136.3	
25	20	1099	6.5	169.1	
10	495	624	3.3	189.1	
5	299	820	4.0	205.0	
11	318	801	3.8	210.8	
29	172	947	4.6	205.9	
15	406	713	3.3	216.1	
31	238	881	4.3	204.9	
30	10	1109	4.1	270.5	
12	138	981	3.1	316.5	
17	94	1025	2.5	410.0	
20	302	817	1.5	544.7 <b>*</b>	
2 .	603	516	0,6	860.0 *	
3	340	779	1.0	779.0 •	
19	87	1032	1.0	1,032.0 *	
9	405	714	0.6	1,190.0 *	
MEAN	379.4	739.6	4.896	288.9	
ST.DEV	381.5	381.5	3.069	317.0	

Superscript \*: These 5 subjects have very high rates of CD4-cell decline. All 5 of these subjects have also been infected for between 0.6 and 1.5 years (approx. between 6 and 18 months).

TABLE 5-G PROFILE OF MOOD STATES (POMS) SCORES (N = 27)

POMS SCALES										
Subj	TA	DD	АН	VA	FI	СВ	FR			
22	00	00	00	32	00	00	24			
16	26	42	35	19	23	18	10			
14	18	11	23	28	15	08	20			
01	24	25	10	14	13	16	15			
13	14	17	30	18	18	06	09			
23	07	06	13	25	07	03	22			
21	34	56	43	03	28	27	05			
04	14	19	25	19	10	10	14			
07	06	02	06	20	21	03	18			
28	27	14	06	27	06	07	21			
18 08	21	26	21	09	15	19	11			
08 25	11	14	21	12	15	13	21			
25 10	28	34	27	09	24	20	12			
05	17	33	07	16	08	08	18			
05 11	15	41	27	08	23	15	19			
29	08	08	03	13	18	10	23			
2 <del>7</del> 15	00 06	05	02	25	01	08	25			
31	36	14	02	22	08	02	20			
30	36 17	57	47	10	23	26	07			
12	18	26 13	19 17	18	13	14	14			
17	05	05	02	14	14	18	18			
20	08	04	02	16 15	00	03	19			
02	01	04	02	28	14 01	11	13			
)3	01	06	08	20 21	08	03 07	22			
9	33	55	27	05	23	07 25	20 15			
19	06	15	13	24	05	08	23			
/IEAN:	14.9	20.4	16.4	17.4	13.1	11.4	17.0			
TDev:	10.9	17.2	13.2	07.5	08.2	07.7	05.4			

TA = Tension-Anxiety DD = Depression-Dejection AH = Anger-Hostlitty

VA = Vigour-Activity FI = Fatigue-Inertia

**CB = Confusion-Bewilderment** 

FR = Friendliness

TABLE 5-H COPING ORIENTATIONS TO PROBLEMS EXPERIENCED (COPE): PROBLEM-FOCUSED SCALES (N = 27)

Subj	Activ	Plan	Suppr	Restr	SSSupi	PROBLEM-FOCUSED COPING TOTAL
22	16	16	07	10	13	62
16	08	12	09	10	08	47
14	13	15	12	12	13	65
01	06	06	08	10	07	37
13	16	16	11	13	07	<b>63</b>
23	15	16	13	11	15	70
21	08	09	06	09	10	42
04	14	14	12	12	12	64
07	12	14	09	11	13	59
28	06	08	06	13	09	44
18	11	11	13	13	11	59
08	13	14	11	13	10	61
25	07	08	10	08	08	41
10	10	12	11	11	13	57
05	08	10	08	08	08	42
11	09	09	07	14	04	43
29	11	12	07	07	09	46
15	15	16	13	04	12	60
31	07	05	05	14	07	38
30	11	12	13	13	08	57
12	13	13	12	13	12	63
17	11	10	13	12	14	60
20	12	12	09	07	14	54
02	14	13	12	10	10	59
03	15	13	11	09	11	59
19	12	14	11	08	10	55
09	12	14	11	10	14	61
MEAN:	11.30	12.00	10.00	10.56	10.44	54.37
STDev:	03.06	03.04	02.50	02.49	02.80	09.46

Suppr = Suppression of competing activities

SSSup = Seeking social support for Instrumental reasons

Restr = Restraint

TABLE 5-I

COPING ORIENTATIONS TO PROBLEMS EXPERIENCED

(COPE): OTHER SCALES (N = 27)

Subj	SSSpE	Pos	Accp	Relig	Vent	Deny	Bdis	Mdis	AlcDis
22	16	16	16	09	13	04	04	04	01
16	04	08	09	04	10	13	08	08	03
14	04	16	16	08	12	07	09	13	01
01	09	07	12	08	<b>07</b> ·	09	08	15	01
13	07	12	16	04	14	04	04	11	02
23	15	13	16	13	14	04	04	12	01
21	11	16	13	10	10	16	14	13	04
04	12	14	15	13	14	06	11	11	02
07	11	12	15	09	10	04	06	08	01
28	07	07	12	04	09	14	15	10	04
18	09	10	12	08	09	04	06	09	01
80	12	14	15	07	12	06	12	11	02
25	12	06	16	04	13	05	09	16	01
10	08	12	13	08	07	07	11	12 .	01
05	06	10	15	07	08	06	10	11	03
11	06	15	13	16	12	11	08	12	01
29	09	15	16	16	08	10	10	13	01
15	08	16	16	08	07	04	06	07	01
31	10	11	10	16	07	12	12	10	02
30	09	11	14	16	05	11	07	12	01
12	07	16	15	09	08	04	06	10	01
17	15	13	13	06	11	.09	08	13	01
20	10	13	12	04	11	04	05	12	01
02	09	16	16	04	09	05	07	08	03
03	10	16	15	04	08	08	04	10	04
19	08	12	11	06	08	04	06	09	02
09	15	14	16	06	15	04	04	12	02
MEAN:	9.59	12.63	14.00	8.41	10.04	7.22	7.93	10.82	1.78
STDev:		03.08	02.08	4.09	02.70	3.62	3.14	02.53	1.05

SSSupE = Seeking Social Support for Emotional Reasons

Deny = Denial Bdis = Behavioural Disengagement
Mdis = Mental Disengagement AlcDis = Alcohol & Drug Disengagement

TABLE 5-J

UCLA LONELINESS SCALE,
HOPELESSNESS, & JOY
(N = 27)

Subj	LONELINESS	JOY	HOPELESSNESS
22	06	08	02
16	09	06	07
14	11	08	06
01	09	06	06
13	07	07	. 06
23	05	08	04
21	16	03	08
04	12	08	04
07	11	06	05
28	16	04	08
18	11	05	03
08	12	06	08
25	11	03	08
10	08	04	07
05	09	05	08
11	11	06	05
29	07	08	03
15	07	07	03
31	15	02	07
30	08	05	03
12	08	07	04
17 20	11	08	08
20 20	09	07	05
02 03	06 08	08	04
J3 19	08 16	06 04	04 06
09	04	08	02
MEAN:	9.74	6.04	5.33
STDev:	3.31	1.81	2.02