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PRACTICE AND RESEARCH

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Research: Psychological variables and HIV infection: a study of their effect on disease progression among long term infected individuals
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suggestions and help with statistical analysis.
1. PERSONAL STUDY PLAN

Summary

1. Academic

a. HIV related cognitive impairment: is there impairment in asymptomatic disease and do early minor deficits predict later major impairment?
b. A critical review of psychological factors in HIV sexual risk behaviour.

2. Clinical

An HIV sexual risk reduction service: from initial proposal to four year evaluation.

3. Research

An investigation of psychological characteristics and their relationship to disease progression among long term HIV infected individuals.
1. **Academic**

a. **HIV related cognitive impairment: is there impairment in asymptomatic disease and do early minor deficits predict later major impairment?**

**Aims**
To acquire an up to date knowledge of recent findings in the area in order to provide relevant information to patients, select appropriate neuropsychological tests and understand the effects of HIV infection in the central nervous system.

**Objectives**
Review literature on neuropathology and neuropsychology in HIV infection particularly as it relates to asymptomatic infection. Compare and evaluate findings from cross sectional and longitudinal studies and relate to changes in the treatment, prognosis and understanding of the nature of HIV infection over time. Consider components of a neuropsychological assessment procedure and how assessment may help in the clinical management of patients.

**Rationale**
Rapid changes have occurred in the understanding of how HIV infection may affect brain function. For example, in the mid 1980s, there were fears that many if not all HIV infected persons would have significant impairment leading to calls for the compulsory testing of armed service personnel as well as for the provision of many beds for long term care. Later, findings of impairment in asymptomatic infection were tempered by demonstrations of the effects of confounding factors such as education and previous history. Although it is probably now generally accepted that there is no significant impairment in asymptomatic infection, some recent studies suggest that minor deficits may be associated with poorer cognitive performance later on. It will be helpful in providing a relevant and sensitive service to have a better understanding of this literature and of whether the reported impairments have a clinical as well as a theoretical significance.
b. A critical review of psychological factors in HIV sexual risk behaviour

Aims
To develop a clearer view about which potentially modifiable individual factors have been reliably associated with risky sexual practices and to identify areas for further investigation.

Objectives
Review recent literature. Evaluate the importance and generalisability of findings taking account of study design and analysis methods. Consider application to clinical population in terms of possible interventions. Identify areas where knowledge is weak and suggest how it could be improved.

Rationale
There have been a large number of studies seeking to identify correlates and predictors of HIV sexual risk behaviour, relatively few of which have gone beyond descriptive data in cross-sectional samples. Psychological processes and characteristics are now attracting more attention in the light of evidence of persisting risk behaviour among individuals with high levels of knowledge about HIV transmission.
2. Clinical

An HIV sexual risk reduction service: from initial proposal to four year evaluation.

Aims
To describe the setting up of a new clinical service and its development over a four year period.

Objectives
Describe the service in terms of (i) Initial proposal and plan (ii) Process: expectations, obstacles, development of interventions, modifications (iii) Evaluation and cost effectiveness (iv) Future plans.

Rationale
There are two main reasons for undertaking this descriptive analysis:

(i) There is continuing evidence that safer sex messages are not universally acted upon. While there is a pressing need for more effective public health campaigns and community level interventions, there is also a case for interventions at an individual level. The service described is directly accessible to individuals attending Genitourinary Medicine clinics who are by definition at risk and it is important to ensure that it is offering relevant and effective interventions. This cannot be achieved without careful monitoring and evaluation, regular appraisal and willingness to change in the light of experience and new research findings.

(ii) The service is funded from HIV prevention monies which were originally ring fenced but are now subject to cost improvements. If it is to continue, a case for its value must be made to the purchasers.
3. Research

An investigation of psychological characteristics and their relationship to disease progression among long term HIV infected individuals.

Aims
To identify potential independent predictors of disease progression in HIV infection.

Objectives
To assess long term infected individuals on measures of personality, psychological morbidity, coping strategies, social support, life events and demographic variables. To identify whether any of these variables are associated with changes in disease state as measured by laboratory variables and clinical status at 6 month follow up.

Rationale
Although the majority of HIV infected individuals eventually develop AIDS, a subgroup of patients have prolonged incubation periods and remain clinically well with stable or even increasing CD4 counts for more than 10 years after infection. The biological and behavioural factors responsible for diminished susceptibility to HIV infection are not well understood. Biological factors thought to be important include initial immune response at seroconversion, viral strain and load, host genetic factors as well as specific immune system activity. Lifestyle and demographic factors have also been suggested as possible determinants of disease progression. There has been a great interest particularly among HIV infected people in the role of psychological factors such as mood, ability to manage stress, psychological attitude to illness and coping strategies. Studies reporting on the effects of psychological factors on markers of HIV disease have produced conflicting results partly attributable to differences in samples, methods and design. Few studies have assessed trait variables and the data to be reported which forms part of a larger study primarily focussed on virological and immunological factors will include measures of trait as well as state psychological variables.
2. ACADEMIC DOSSIER
HIV RELATED COGNITIVE IMPAIRMENT: IS THERE IMPAIRMENT IN ASYMPOTOMATIC DISEASE AND DO EARLY MINOR DEFICITS PREDICT LATER MAJOR IMPAIRMENT?

In this paper, the evidence concerning cognitive functioning among adults with asymptomatic HIV disease will be evaluated. When HIV-associated dementia was first recognised in 1986 (Navia et al, 1986), the prospect of this new manifestation of AIDS gave rise to alarm among people with HIV infection, their carers and those involved in providing and planning care. A year later, a report that cognitive deficits could be detected in otherwise asymptomatic individuals (Grant et al, 1987) added to the general concern and led to calls for the routine testing of aircraft pilots, members of the armed forces and people taking driving tests among others. As a consequence, many patients with HIV infection fear the possibility that they may experience a decline in their intellectual abilities and as the disease progresses, may attribute cognitive failures due to fatigue or illness to a dementing process. The presence of anxiety or depression may also contribute to perceptions that impairment is present.

The question of whether cognitive impairment occurs in the early asymptomatic stages of HIV infection has important implications for patient care and for an understanding of the pathogenesis of HIV-associated dementia in later illness. Evidence from neuropathological and neuroimaging studies will be described followed by a brief outline of the features of HIV-I associated dementia. Finally neuropsychological studies in asymptomatic infection and methodological issues will be reviewed.

1. NEUROPATHOLOGICAL AND NEUROIMAGING STUDIES

Almost all individuals who die with AIDS have cerebral pathology (Budka et al, 1987; Lantos et al, 1989). The most common abnormalities seen are the opportunistic infections toxoplasma gondii, cryptococcus, cytomegalovirus and JC virus which causes progressive multifocal leukoencephathy. Primary central nervous system (CNS) lymphoma is also relatively common (Everall & Lantos, 1991). In addition to these
secondary processes, HIV has a direct effect on the brain. The most important of the several primary HIV pathologies are HIV encephalitis and HIV leukoencephalopathy (Budka et al, 1991). HIV encephalitis consists of inflammatory cell infiltrates including macrophages, microglia and multinucleated giant cells seen mainly in the white matter, basal ganglia, brain stem and cortex. The dominant feature of HIV leukoencephalopathy is diffuse white matter damage with myelin loss and astrocytosis.

Although HIV primarily affects the white matter and subcortical grey matter of the CNS and is not thought to infect neurones, it is now established that neuronal loss and damage are a common consequence of HIV infection (Ketzler et al, 1990). Stereological studies have identified a neuronal reduction of 38% in the frontal cortex among patients who had died from HIV without CNS opportunistic infections or neoplasms and regardless of the presence or absence of HIV encephalitis (Everall et al, 1991). Neuronal loss has also been reported in the occipital, parietal and temporal lobes of the brain of people who have died of AIDS (Wiley et al, 1991; Everall et al, 1993) and in areas outside the cortex including the substantia nigra (Reyes et al, 1991) and possibly the cerebellum (Grauss et al, 1990).

Magnetic Resonance Imaging (MRI) and Computerised Tomography (CT) show clear structural abnormalities in the brains of people with HIV-1 associated dementia most commonly cortical atrophy and white matter lesions in the subcortical and periventricular regions (McArthur, 1987; Dal Pan et al, 1992) and reductions in basal ganglia volumes (Aylward et al, 1993). Results of a more recent study using MRI indicate that grey matter volume reductions occur in the posterior cortex as well as in the basal ganglia in HIV-associated dementia (Aylward et al, 1995).

Viral antigen can be detected in the cerebrospinal fluid within a few weeks of infection (Ho et al, 1985) and it is assumed that HIV enters the brain early on. Proviral DNA has also been identified in the brains of individuals with asymptomatic HIV infection (Sinclair & Scaravalli, 1992) and, although viral load in the brain is the highest of any organ, it usually remains undetectable until symptomatic disease
develops (Donaldson et al, 1994). The question then arises as to when neuropathological and structural abnormalities in the brain first occur.

One of the difficulties in establishing biological markers of HIV related brain impairment is that the pathological changes are not pathognomonic to the clinical dementia. Fewer than 50% of individuals with the condition have autopsy proven HIV encephalitis (Glass et al, 1993). Similarly, the features seen on MRI and CT among those with HIV-I associated dementia are also frequently found among people with other AIDS defining conditions (eg Raininko et al, 1992). Furthermore Manji et al (1994) found white matter abnormalities on MRI among both asymptomatic and seronegative homosexual men. Other studies have also found that CT and MRI show no differences between these two groups (McArthur et al, 1990; McAllister et al, 1992) although Raininko et al (1992) reports greater rates of cerebral atrophy, particularly in the frontal lobes and cerebellum, among asymptomatic than seronegative subjects.

If neuroimaging abnormalities are detected in the asymptomatic phase of infection, are they predictive of future change? Two longitudinal studies suggest not: Post et al (1993) found that while 20% of their cohort with asymptomatic infection had abnormal MRI scans at baseline, only 2 (15%) of these showed further deterioration after up to 42 months follow up. The second study found no evidence of developing cerebral atrophy among asymptomatic individuals over the course of a year (Manji et al, 1994). In addition, apart from a strong association with major cognitive impairment (Grant et al, 1987; Raininko et al, 1992), there is no agreement that MRI abnormalities are correlated with neuropsychological performance, some studies identifying correlations (Cohen et al, 1992; Hestad et al, 1993) while others fail to do so (McArthur et al, 1990; Dooneief et al, 1992; McAllister et al, 1992).

As far as neuropathological changes are concerned, most brains examined in post mortem studies are those of people who have died of AIDS, but there are a few studies of people with asymptomatic HIV infection who have died of other causes.
Non specific changes such as myelin pallor, reactive astrocytosis and mineralisation of vessel walls may be seen and the activity of astrocytes and microglial cells is increased, but neuronal loss has not been identified in asymptomatic individuals (Ciardi et al, 1990; Gray et al, 1992; Everall et al, 1994).

In summary, while CNS abnormalities may be found at all stages of HIV infection, there is little to indicate that those identified among asymptomatic individuals are early signs of later deterioration nor that they are associated with deficits in cognitive function in this group.

2. HIV-I ASSOCIATED DEMENTIA

A progressive decline of cognitive function was first reported by Navia et al in 1986 who found that almost 80% of their series of 121 patients who had died of AIDS had cognitive impairment. While more than half of these had CNS disease secondary to HIV infection, the rest showed unexplained cognitive or behavioural changes. Since then, it has become clear with the use of more precise criteria for HIV-1 associated dementia (World Health Organisation (WHO), 1990), shown in Table 1, that its presentation as a first sign of AIDS is relatively uncommon and that it most often occurs in people with other AIDS defining illnesses (McArthur et al, 1993). Indeed the prevalence of dementia in HIV infection is now known to be far lower than first thought and data from the WHO cross-cultural study gives a current estimate of about 6% (Maj et al, 1994). In a study carried out in central London using the WHO definition, for the years 1991 to 1994, the prevalence was found to be between 3.5 and 6.9 % (Catalan et al, 1995).
<table>
<thead>
<tr>
<th>A</th>
<th>Evidence of a dementia, of a specified level of severity, based on the presence of each of the following:</th>
</tr>
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<tbody>
<tr>
<td>(i)</td>
<td>A decline in memory which may not be severe enough to cause impaired functioning in daily living. The decline should be objectively verifiable and not based on subjective complaint. The level of severity should be assessed as follows:</td>
</tr>
<tr>
<td>-</td>
<td>Mild impairment: A degree of memory loss sufficient to interfere with everyday activities, though not so severe as to be incompatible with independent living. The main function affected is the learning of new material.</td>
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<tr>
<td>-</td>
<td>Moderate impairment: A more severe degree of memory loss. Only highly learned or familiar material is retained. This degree of memory impairment is a serious handicap to independent living.</td>
</tr>
<tr>
<td>-</td>
<td>Severe impairment: Severe memory loss with only fragments of previously learned material remaining. The individual is not able to function in the community without close supervision.</td>
</tr>
<tr>
<td>(ii)</td>
<td>A decline in intellectual abilities characterised by deterioration in thinking and processing of information of a degree leading to impaired functioning in daily living. The level of intellectual impairment should be assessed as follows:</td>
</tr>
<tr>
<td>-</td>
<td>Mild impairment: The decline in intellectual abilities causes impaired performance in daily living, but not to a degree making the individual dependent on others.</td>
</tr>
<tr>
<td>-</td>
<td>Moderate impairment: The decline in intellectual abilities makes the individual unable to function without the assistance of another in activities of daily living.</td>
</tr>
<tr>
<td>-</td>
<td>Severe impairment: The decline precludes not only independence from the assistance of others, but is characterised by an absence, or virtual absence of intelligible ideation.</td>
</tr>
<tr>
<td>(iii)</td>
<td>Decline in motor function may be present.</td>
</tr>
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<td>(iv)</td>
<td>Aphasia, agnosia and apraxia are unusual.</td>
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<td>(v)</td>
<td>Symptoms should be present for at least one month.</td>
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<tr>
<td>B</td>
<td>Absence of clouding of consciousness.</td>
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<tr>
<td>C</td>
<td>A deterioration in emotional control or social behaviour.</td>
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<tr>
<td>D</td>
<td>Laboratory evidence of systemic HIV-1 infection.</td>
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<tr>
<td>E</td>
<td>No evidence of another aetiology.</td>
</tr>
</tbody>
</table>

Table 1: World Health Organisation (1990) definition of HIV-1 associated dementia.
The features of HIV-I associated dementia, as one might expect in a condition which primarily involves the white matter and subcortical structures, involve disturbances of motivation, attention, arousal, timing and rate of information processing (Cummings, 1986). These are reflected in psychomotor slowing, impairment of fine motor control and deficits in executive functions involving strategy and concept formation, problem solving and sequencing. Memory and language impairments are also found but are usually less marked than in cortical dementias (Burgess and Riccio, 1992). A similar but less severe pattern of deficits may be seen in people with advanced HIV disease (Janssen et al, 1989; Perdices & Cooper, 1990) although a recent longitudinal study shows that the development of AIDS is not associated with a general decline in cognitive function except in fine motor skills (Selnes et al, 1995). The prognosis is very poor for those who develop dementia with a median life expectancy from diagnosis of only 6 months (McArthur et al, 1993). Even HIV infected individuals with cognitive impairment which does not meet criteria for dementia have been reported to be four times more likely to die over a three year follow up period than those without impairments (Mayeux et al, 1993).

While it is now generally recognised that dementia is rare, there has been considerable debate about when cognitive impairments can first be detected, whether early deficits predict dementia later on and if so, which as well as whether any deficits can be identified in asymptomatic disease.

3. NEUROPSYCHOLOGY OF ASYMPTOMATIC INFECTION

The first study to report on cognitive function among seropositive individuals with mild or asymptomatic disease appeared in 1987. Forty four gay men were divided into 3 groups according to disease stage and their performance on 8 neuropsychological tests was compared with that of 11 seronegative controls (Grant et al, 1987). Fifteen had AIDS, 13 had AIDS related complex (ARC) and 16 did not meet the criteria for AIDS or ARC. Individuals were classified as impaired if they achieved one definitely abnormal or 2 probably abnormal scores on 9 measures of test.
performance. On this basis, 7 (44%) of the asymptomatic and 1 (9%) of the seronegative groups were impaired. Although it was based on small numbers, this finding was very influential and led to calls for routine testing of drivers and pilots as well as to the introduction of mandatory testing for US armed forces personnel. It is important to note that although the proportions of those with abnormal scores distinguished between the asymptomatic and seronegative groups, when mean scores were considered, there were no statistically significant differences on any of the tests between the two groups. Further, the definition of impairment meant that 13/15 (87%) of those with AIDS were classified as impaired, a much larger proportion than is found in other samples (Tross et al, 1988; Janssen et al, 1989; Perdices & Cooper, 1990).

As well as generating a good deal of concern, Grant et al's study had the positive effect of encouraging research into the neuropsychology of early HIV disease and subsequent studies have either found no impairment in asymptomatic disease or tended to report fewer and less severe deficits. Of 56 studies reporting neuropsychological data in asymptomatic infection, either published or presented at conferences, Burgess and Riccio (1992) identified 30 showing that asymptomatic individuals have some cognitive impairment when compared with seronegative controls while 26 reported no differences. A straightforward count is misleading however, as Perry (1990) who conducted a similar earlier review pointed out, since the studies vary along many dimensions. Although it is outside the scope of this paper to review all the existing studies, a few will be described in detail to illustrate some of the methodological differences which may account for the different conclusions reported.

3.1. Methodological issues

In some transmission groups such as haemophilia and injecting drug use, membership is associated with a risk of cognitive impairment independent of that conferred by HIV infection (Royal et al, 1991; Silberstein et al, 1993). A large proportion of
studies have however reported on cognitive function among asymptomatic gay men and much of what follows will refer to these studies.

**Confounding factors**
The importance of controlling for confounding factors which affect performance on neuropsychological tests such as age, education, premorbid IQ, alcohol and drug use, psychiatric illness, neurological disease or history of head injury has been stressed (Goethe et al, 1989; Wilkins et al, 1990). These factors have not always been considered nor adequately controlled for, particularly in smaller studies (eg. Grant et al, 1987; Perry et al, 1989). In a study of neuropsychological performance among 40 gay men (28 asymptomatic and 12 with ARC), Wilkins et al (1990) tellingly demonstrated the effect of ignoring preexisting confounds. Thirty of their subjects had evidence of these factors: in more than half they were considered severe and included epilepsy, developmental disability and closed head injury. With no screening for confounding factors, 17 (60%) of the asymptomatic group showed a degree of cognitive impairment but when these were taken into account, the number dropped to 5 (22%). A significant association was found between impairment and presence and severity of preexisting confound.

**Homogeneity of sample**
One of the less obvious potential source of differences between studies is the classification of subjects as asymptomatic implying a homogeneous group. In reality, and as clinical experience suggests, some participants may be very well, others may have lymphadenopathy and some while not fulfilling criteria for other disease stages, are suffering from a range of other subclinical physical problems. Asymptomatic study subjects may also vary considerably in the length of time they have been infected. Many of the studies report only the subjects’ clinical stage for which different systems or methods of categorisation may be used. Some do not include details of lymphocyte subsets (CD4 counts or CD4/CD8 ratios) which are widely used indices of disease status. Where they are reported, some studies find correlations between these measures and neuropsychological performance (Goethe et al, 1989;
Mitchell et al, 1989; Stern et al, 1991) while others find little or no association, particularly for counts above 400, a level at which significant symptomatic disease is unlikely (McArthur et al, 1989; Miller et al, 1990). Bornstein et al (1991) however, reporting on a group with a wide range of CD4 counts, found that rate of decline rather than absolute count was associated with cognitive deficits.

Neuropsychological Measures
An important question in evaluating findings is the adequacy of the neuropsychological measures both in terms of range and sensitivity to the diffuse and often subtle effects of HIV on the CNS. Tests which were developed to assess cortical functions primarily may not be appropriate. For example, tests such as those in the Boston Diagnostic Aphasia Examination used by a number of investigators (eg. Janssen et al, 1989; Clifford et al, 1990; Stern et al, 1991) are unlikely to detect differences between asymptomatic and seronegative groups since language deficits are unusual even in HIV-associated dementia (WHO, 1990). Some studies use only a narrow range of tests (eg. McArthur et al, 1989; Janssen et al, 1989). This is sometimes the case in large prospective studies in which participants undergo repeated extensive clinical and other assessments and a balance must be maintained between obtaining useful information and sample attrition. These studies gain greater statistical power to detect differences perhaps at the expense of a more specific description of any deficits which may exist.

In two large studies, the Multicenter AIDS Cohort Study (MACS) and the San Francisco Study (SFS) reporting on baseline measures, investigators attempted to validate their relatively brief assessment procedures by following them with more detailed neuropsychological assessment among a subgroup of participants. The MACS investigators used the following six measures: Grooved Pegboard (Klove, 1963), Rey Auditory-Verbal Learning Test (Rey, 1964), Trail Making Test, Part B (Reitan, 1979), Digit Span (Wechsler, 1989), Controlled Oral Word Association (Benton, 1968) and Symbol Digit Modalities Test (Smith, 1982). The tests used in the SFS were the Thumb Finger Sequential Touch Test (Golden et al, 1980), Wechsler Logical
Memory Test (Wechsler, 1974), Trail Making Test, Parts A and B (Reitan, 1979),
Digit Symbol (Wechsler, 1989) and Boston Naming Test (Goodglass & Kaplan,
1983). Hence in both studies the tests covered visuomotor skills, verbal memory,
ability to maintain and switch attention as well as language, although the test used in
the MACS is generally seen as a test sensitive to frontal lobe function rather than to
language deficits.

Initial screening resulted in similar rates of abnormal test results (12-15%) among
seronegative and asymptomatic subjects in both studies. In the MACS (McArthur et
al, 1989), 270 gay men with asymptomatic HIV infection were compared with 193
seronegative controls and detailed neurological and neuropsychological assessment
was carried out in 119 (53%) of those with one abnormal result. Again, no significant
differences between the two groups were detected and in the 24 subjects found to be
impaired, deficits in performance were in most cases attributable to identifiable
factors such as those referred to above. The SFS sample (Janssen et al, 1989) was
made up of 100 seropositive (26 with ARC, 31 with lymphadenopathy and 43 with
no symptoms) and 157 seronegative gay men 85 of whom (33% of the whole sample),
were given a more extensive neuropsychological assessment. Only subjects with ARC
were likely to score more abnormally than the seronegative group: abnormal results
were noted in 7 of 16 (44%) with ARC, 4 of 28 (16%) asymptomatic and 5 of 41
(12%) seronegative individuals. It is of note however that in this sample, while the
brief assessment was found to be highly accurate in detecting normal results, brief
screening correctly identified only 9 of 16 subjects (56%) with abnormal results
following extensive testing.

Definition of impairment
As well as differences in test selection, differences in methods of defining impairment
hinder comparisons between studies. This is particularly the case where results are
not fully reported, often one suspects because of space constraints imposed by journal
editors when large amounts of data and analysis are involved. Some studies use
standard test norms (eg. Goethe et al, 1989; Stern et al, 1991) while others derive
norms from the seronegative control group (eg. McArthur et al, 1989; Riccio et al, 1993). Impairment may be defined as 1.5 to 2 standard deviations (SD) below the mean (Janssen et al, 1989) or more often, 2 or more standard deviations below the mean of the comparison group (eg. Miller et al, 1990; Riccio et al, 1993). Some studies derive idiosyncratic indices of severity of impairment (eg. Stern et al, 1991; Stern, R. et al, 1992). For example, in Stern et al’s 1991 study, performance on each test was first compared with extant norms then rated as normal, borderline (at least 1 SD below the mean) or defective (at least 2 SDs below the mean). A further global performance rating was then derived to summarise overall neuropsychological performance and to rank it on a five point continuum of severity. Other investigators simply compare mean scores in the different groups (Krikorian & Wrobel, 1991; Collier et al, 1992; McAllister et al, 1992) while some use a combination of approaches (eg. Clifford et al, 1990; Stern et al, 1991). It is much easier for the practising clinician to evaluate the significance of the test findings when means and ranges of scores are reported and although it is informative to compare group performances on individual tests, it is relevant to note that clinical evaluation is typically based on interpretation of the pattern of performance on a full range of tests.

**Effect of mood**

Because depression is common among patients with HIV infection (eg. Williams et al, 1991), it has been thought to account for some of the cognitive abnormalities observed. Bornstein et al (1993a) carried out a carefully controlled study of 121 asymptomatic and 42 seronegative gay men in which depression was assessed by self report (Beck Depression Inventory, Beck et al, 1961), interviewer ratings (Hamilton Rating Scale for Depression, Hamilton, 1960) and the Structured Clinical Interview for DSMIII-R (Spitzer et al, 1987). Cognitive function was indexed by a comprehensive neuropsychological procedure. The data demonstrate no relationship between depression and cognitive measures regardless of the method of defining depression, consistent with findings in symptomatic disease (eg. Hinkin et al, 1992). Depression has been shown to be associated with self report of cognitive deficits among both asymptomatic and seronegative men, although no relationship between
self report and actual test performance was found, even when those obtaining the lowest test scores were examined (van Gorp et al, 1991). Findings from other studies support the association between mood disorder and perceived cognitive failures and the lack of relationship between the latter and neuropsychological performance (Wilkins et al, 1991; Riccio et al, 1993).

Finally, a general problem which applies to many of the studies is the question of how representative of the general HIV positive population the samples are. Many individuals who are seropositive and asymptomatic remain undetected. In addition, the samples are often self selected volunteers or those seen at specialist medical centres.

3. 2. Findings in asymptomatic HIV infection

A number of well designed studies have now reported no differences in cognitive function between asymptomatic and seronegative gay men. The largest of these is the MACS with a total sample of 1500 (Miller et al, 1990) although, as mentioned above, only a limited assessment procedure was used. Other studies from the USA with samples ranging from 80 to 260 (Janssen et al, 1989; Goethe et al, 1989; Clifford et al, 1990) confirm these findings as do two studies from the UK with samples of 125 and 100 respectively (McAllister et al, 1992; Riccio et al, 1993). All these studies take account of possible confounding factors and use detailed neuropsychological assessments.

Nevertheless, there are a few equally well designed studies which do report impairments among gay men with asymptomatic disease. Stern et al (1991) used various methods to assess impairment (described in section 3. 1. above) derived from performance in 19 neuropsychological tests including a global performance rating and an independent neuropsychologist's impression. Although there were no differences between the HIV positive and control groups on frequency of defective or borderline performance in any cognitive area by either method of assessment, further analysis
showed small but significant differences between the groups on measures of verbal memory, executive function and verbal fluency. The findings have been criticised on the grounds that the asymptomatic group were of lower socioeconomic status, included a large proportion of subjects whose first language was not English, and had unusually low CD4 counts (Miller et al., 1992). Stern et al (1992) maintain that essentially similar results are obtained when these factors are taken into account. The fact remains that this sample may have been unusual in some way since Stern et al also report associations between anxiety, depression and self report of cognitive deficits and neuropsychological performance, as well as between CD4/CD8 ratios and cognitive function none of which have been generally reported (see section 3. 1. above). Three other smaller studies also report deficits in verbal memory and psychomotor speed (Lunn et al, 1990), verbal and visual memory and information processing speed (Wilkie et al, 1991), and motor speed only (Stern R. et al., 1992) among asymptomatic groups.

A few prospective studies have now reported follow up data. These include the MACS which reported on 238 asymptomatic and 170 seronegative subjects tested at six month intervals over a period of up to 2 years (Selnes et al, 1990). The method of data analysis controlled for differences in frequency of follow up and for other possible confounding factors the most important of which have been identified as age and years of education (Satz et al, 1993; van Gorp et al, 1994). There were no differences between the two groups over time and no significant effect of serostatus on any aspect of cognitive function nor on patterns of change. These findings have been confirmed by those from three other well designed prospective studies of gay men (Saykin et al, 1992; Mauri et al, 1993; Burgess et al, 1994).

One study of HIV infected injecting drug users has reported small declines in performance relative to baseline at 4 year follow up on measures of psychomotor speed among subjects remaining asymptomatic. The authors conclude that subtle deficits may develop over time but their progress is slow and generally parallel changes in immune status. Other prospective studies of seropositive injecting drug
users have found no progressive changes in cognitive function in asymptomatic disease (Selnes et al, 1992; Egan et al, 1992).

Two studies among gay men suggest that even when overall performance is within normal limits, subtle abnormalities might be predictive of subsequent decline. Bornstein et al (1993b) found that neuropsychological performance remained stable over 12 month follow up for the majority of their asymptomatic subjects but that about one third showed decline on some measures. Examination of this subgroup's scores at baseline showed poorer performance on measures of information processing, verbal memory and reaction time although the authors do not indicate whether these are the same cognitive areas as those found to have deteriorated later on. Burgess et al (1994) also found that when subjects' performance at follow up was predicted from baseline, controlling for a range of medical and psychosocial factors, a significantly larger proportion of asymptomatic than seronegative control subjects performed at a lower level than expected. The authors acknowledge that the numbers involved are small, and that it is not possible to determine whether the changes are consequent on factors other than HIV infection, nor whether they are permanent or transient much less whether they are meaningful in terms of prognostic value.

In conclusion, there is little evidence to suggest that HIV-1 has a significant impact on cognitive function during the asymptomatic phase of infection and even where small effects are found, they are of little clinical significance and would not be expected to be noticeable in everyday life.

Finally, given the lack of evidence for substantial impairment in early disease, it is relevant to consider the question of when cognitive deficits first arise. Perdices et al (1991) monitored a group of asymptomatic subjects at 6 month intervals and compared those who developed symptomatic disease with those who remained well. They found no evidence of cognitive deterioration associated with disease progression. There is only one study of people for whom detailed neuropsychological data was available over the period before they developed HIV-I associated dementia. Selnes et
al (1991) found that in 11 of 12 individuals, performance remained at or above group means and was essentially stable until a precipitous decline was seen at the onset of dementia.

CONCLUSIONS

Seropositive patients presenting with subjective cognitive complaints may be reassured that the available evidence indicates that dementia is very rare in HIV disease, that there is no evidence of any significant impairment in asymptomatic disease nor that any deficits which may be detected are predictive of cognitive decline later on. It is important to assess mood disorder as well as neuropsychological function given its reported association with perceived impairment and to offer appropriate intervention. As with any other assessment of cognitive function, a full evaluation of developmental and medical factors together with substance and alcohol use, current and past is essential in correctly identifying any changes attributable to the disease process.

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HIV SEXUAL RISK BEHAVIOUR AMONG GAY MEN

Sexual activity is the most important mode of HIV transmission (Piot et al, 1990) and while no vaccine or cure for the infection exists, the main option available for prevention of further spread is the adoption of sustained changes in sexual behaviour. In the UK, most AIDS cases occur among gay men: among the total of 13,720 cases recorded since reporting began in 1982, about three quarters are in the exposure category of sexual intercourse between men. Almost two thirds of all HIV infections in the UK are concentrated in the Thames regions in London (Communicable Disease Report, Feb 1997).

This review will consider the empirical literature about factors associated with HIV sexual risk behaviour among gay men in the developed world since this group has been most extensively studied and the findings are most relevant to the clinical population in London.

1. CURRENT SEXUAL BEHAVIOUR

It is well established that unprotected anal intercourse is the most risky sexual activity for HIV transmission although there is a small risk of infection following orogenital contact (Caceres & van Griensven, 1993). Following discovery of the transmission routes for HIV, recommendations made to gay men to avoid infection were to use condoms consistently for anal intercourse or to refrain from this practice, and to reduce their number of sexual partners. The early years of the epidemic were marked by extraordinary changes in sexual practices and declines in incidence of sexually transmitted diseases among gay men (van de Laar et al, 1990; Winkelstein et al, 1987; Johnson and Gill, 1989). Recent evidence however indicates widespread patterns of inconsistent safer sex over time (Adib et al, 1991; de Wit & Griensven, 1994; Gold, 1994) and an increased prevalence of rectal gonorrhoea in this group (Evans et al, 1993; de Wit et al, 1993). Within the UK, this is particularly the case in London (Communicable Disease Report, Nov 1996). The prevalence of HIV
seropositivity among young gay men in San Francisco is only marginally below that which obtained in 1984, at a time when the threat of AIDS was not yet recognised (Osmond et al, 1994). The incidence of new HIV infections among gay men attending genitourinary medicine clinics in London for repeat HIV testing in the period from 1988 to 1994 is 3.8 per 100 person years (Miller et al, 1995) and is little changed from the incidence in an earlier report covering the years from 1988-1990 (Waigh & Miller, 1991).

2. FACTORS ASSOCIATED WITH UNSAFE SEXUAL BEHAVIOUR

A large number of studies have sought to identify correlates and predictors of HIV sexual risk behaviour. Methodological and sample diversity and the fact that sexual behaviour is subject to individual, social and cultural influences results in findings which are at times inconclusive or contradictory. For example, the operational definition of what constitutes risk behaviour may be in terms of frequency of unprotected anal intercourse, proportion of unprotected episodes, number of partners, number of partners of unknown HIV serostatus or some combination of these. The timing of the study and its geographical location in relation to prevalence and awareness of the infection may also affect the findings. Some broad categorical distinctions characterising unsafe sexual behaviour can nevertheless be made:

2.1. Knowledge about HIV and perception of risk

Risky behaviour cannot in most cases be attributed to ignorance about HIV transmission. Many studies investigating unsafe behaviour show that participants are very well informed about the risk of infection from their engagement in unprotected intercourse (eg. Gold et al, 1991; Church et al, 1993) even if they may underestimate their personal risk (Fitzpatrick et al, 1989; Kelly et al, 1990) indicating that while knowledge is necessary, it is not sufficient to alter behaviour. This is hardly surprising when one considers the marginal impact of public health campaigns on many health related behaviours (Steptoe & Wardle, 1994). A recent study (Kelly et
al, 1995) showed that, contrary to expectations, estimated personal risk was actually higher among men who continued to engage in unprotected intercourse underlining the insufficient effect of knowledge on risk behaviour and illustrating the differences in findings (cf. Kelly et al, 1990, above) that might be attributable to the timing or location of the study.

2. 2. Demographic and cultural factors

Poverty, lower educational levels and belonging to an ethnic minority group have all been associated with unsafe sexual activity (Kelly et al, 1990; Doll et al, 1991; Peterson et al, 1992; Kelly et al, 1995) but other studies have not found relationships between income or education and HIV risk behaviour (Adib et al, 1991). A number of studies indicate that younger gay men are more likely than older men to engage in unprotected intercourse (Ekstrand & Coates, 1990; Hays et al, 1990; Kelly et al, 1990). These findings have been contested by those of other investigators (Connell & Kippax, 1990; Davies et al, 1992, 1993; Ridge et al, 1994) who find no direct association between age and unsafe practices and argue that sexual safety is more appropriately explained in terms of social and cultural cohort effects rather than age specific effects.

In spite of this, recent surveillance data indicates that the incidence of HIV infection is highest among younger gay men (Osmond et al, 1995; Miller et al, 1995). If, as seems likely, younger gay men are at greater risk, no empirical evidence has yet been collected which might help to explain this situation. Ekstrand & Coates (1990) suggest that younger men might have less social support for practising safer sex, poorer skills for negotiating condom use or have perceptions of personal invulnerability.

2. 3. Type of relationship

Many studies have noted the strong association between unprotected sex and having a regular or important partner (eg. McKusker et al, 1990; Adib et al, 1991; Hunt et
al, 1992). Prevention of HIV transmission of course relies critically on the accuracy of any assumption by the partners that the relationship is monogamous and their HIV status is concordant. Hoff et al (1997) show that only about half of those in primary relationships report that they are monogamous regardless of whether their partner is concordant or discordant for HIV serostatus.

2. 4. Knowledge of serostatus

Knowledge of seropositivity is associated with increases in the adoption of safer sex (McKusker et al, 1989; van Griensven et al, 1993). A number of studies show that up to 40% of seropositive men still engage in unprotected intercourse at least occasionally (Coleman & Miller, 1994; Marks et al, 1994; Robins et al, 1994) although it is likely that a substantial proportion of the unsafe encounters occur within regular relationships. Recent evidence from the UK (Catchpole et al, 1996) shows that among seropositive gay men with newly diagnosed sexually transmitted disease, almost half knew themselves to be positive, indicating that substantial numbers of seropositive men are practising unsafe sex. It is also unfortunately the case that some individuals engaging in high risk behaviours are unaware of their positive serostatus. One large study of young gay and bisexual men, one third of whom reported unprotected intercourse within the previous six months, found an HIV seroprevalence of 9.4%. Although almost all the participants (90%) had been tested a median of 7 months previously, only 30% of those infected were aware of their positive serostatus (Lemp et al, 1994).

2. 5. Attitudes to condom use

Condoms are the only widely available means of reducing sexual risk but most people do not like using them (eg. Valdiserri et al, 1988). Enjoyment of unprotected intercourse is associated with both persistent risk behaviour and change from low to high risk (Stall et al, 1990; Hays et al, 1992).
2.6. Social norms

Social and cultural norms about safer sex are clearly important in determining behaviour. The perception that insistence on safer sex is not acceptable within one's peer group and an absence of peer support for safer sex are associated with high risk sexual behaviour (Kelly et al, 1990; Stall et al, 1990; Kelly et al, 1995) and changes to lower risk with the perception that social norms favour safe sex (Centers for Disease Control, 1991; Hays et al, 1992; Lemp et al, 1994).

The research findings reviewed above largely confirm expectations and while they are useful in beginning to formulate public health and community level interventions, they contribute little to an understanding of the psychological processes involved in putting oneself or others at risk of such a major health threat. Nor are factors of the kind identified sufficient to explain sexual risk behaviour. For example, in a recent study of 6000 gay men (Kelly et al, 1995), factors associated with engaging in unprotected intercourse were younger age, lower level of education, belonging to an ethnic minority group, lower perceived peer norms for safer sex, more partners and weaker safe sex intentions. These variables however correctly classified only 32% of the unsafe group indicating that other factors and processes are involved.

Before turning to the available literature concerning psychological factors, it is important to determine how far unprotected intercourse among gay men really does constitute a significant risk for HIV transmission since, in spite of the evidence quoted in section 1 above, controversy exists about the meaning and validity of such statistics. This is exemplified in the continuing discussion about relapse from safer sex and about the concept of negotiated safety.
3. **Relapse from safer sex**

There has been a heated debate between those who believe that there is quantitative evidence that a substantial proportion of men relapse or lapse from safe sex (eg Stall et al, 1990) and those who argue that the concept of relapse is neither useful nor accurate (eg Hickson et al, 1992). The term was first used to describe data from longitudinal studies in the USA in which participants are classified into categories defined by levels of risk at each visit thus allowing identification of those moving from a safer to a more risky category over time. Stall et al (1990) reported that 19% of their cohort relapsed to unsafe practices after reporting only safer sex at a previous measurement point. In a second large longitudinal study, almost half the participants failed to maintain safe sex at least once during two year follow up (Adib et al, 1991) and the proportion rose to 62% over a three year period in another cohort (Ekstrand et al, 1992).

It has been argued that the term relapse not only carries pejorative implications of loss of control over a behaviour which is addictive, but that it fails to take account of the context in which the behaviour takes place (Hickson et al, 1992; Hart et al, 1992). These authors point out that the descriptor was applied to the data post hoc and that the participants were not asked whether they considered themselves to have relapsed. Further, they argue that most high risk sex occurs within a regular relationship: 'relapse' may often reflect a new stable relationship and therefore represent a reduced rather than an increased risk of HIV transmission.

The concept of 'negotiated safety' has been advanced (Kippax et al, 1993) in which an apparent return to risky sex is in fact a negotiated agreement to have unprotected intercourse between partners who identify themselves as having the same serostatus and hence is a low risk activity. Stall & Ekstrand (1994) accept that variables identified in quantitative research are not sufficient to explain interpersonal and contextual issues in sexual risk behaviour and endorse the need for qualitative data to help in constructing theories to explain maintenance or otherwise of safer sex.
They insist however that if qualitative analyses proceed without reference to the one quantitative variable whose validity is not contestable, namely high rates of seroconversion among gay men, they will not be addressing the critical issue. Findings in two qualitative studies have suggested that negotiated safety accounts for little of the occurrence of unprotected intercourse. Ridge (1996) has pointed out that some of the prerequisites for negotiated safety which include open verbal communication and an equal balance of power were not common in his small study of gay relationships. In another qualitative study, Sharp et al (1996) reported that almost half the men in their sample of 35 sometimes deviated from the conditions necessary for negotiated safety and engaged in unsafe activity. In addition, some evidence exists that while those who report frequent engagement in unsafe sex may make a positive choice to do so perhaps in the context of a stable relationship, many men who have occasional high risk episodes report negative emotional consequences including guilt, worry, fear and discouragement suggesting that their behaviour was not the rational choice implied by the idea of negotiated safety (Ekstrand et al, 1992).

4. WHY DO GAY MEN ENGAGE IN UNSAFE SEX?

If then it is accepted that a substantial proportion of gay men engage in unsafe sex but would prefer not to, what hypotheses can be advanced about the reasons? In this section, some of the possibilities and the evidence concerning them will be reviewed.

4.1. Alcohol and substance use

A number of studies suggest that the prevalence and frequency of alcohol and substance use is greater among gay men than in the general population (Lohrenz et al, 1978; Israelstam & Lambert, 1983; Stall & Wiley, 1988). In the context of sexual encounters, alcohol has been implicated as an aphrodisiac (Crowe & George, 1989), as a sexual disinhibitor (Lang et al, 1980) and as an excuse for otherwise unacceptable sexual practices (Lang, 1983). In a study of predominantly HIV positive gay clinic attenders, almost half (41%) of those taking ecstasy, LSD, amphetamines
or cocaine in the last year thought they were more likely to have unsafe sex while using drugs and 64% agreed that they felt less inhibited about sexual activity while using drugs (Mansfield & Owen, 1993).

There have been many studies demonstrating an association between the use of drugs and alcohol before or during unsafe sexual activity (eg. Stall et al, 1990; McKusick et al, 1990; Doll et al, 1991). On the other hand, an approximately equal number of investigations fail to find such an association (Gold et al, 1991; Gold & Skinner, 1992; Myers et al, 1992; Temple et al, 1993). Some studies implicate substance use but not alcohol (Valleroy et al, 1993; de Wit & Grienvsen, 1994) and others alcohol but not other drugs (Sandfort et al, 1994; Perry et al, 1994). Some of the variability in the findings is attributable to sampling and methodological differences and most of the studies above used a cross sectional approach even if some employ multiple regression techniques to extract predictors of the dependent variables. If a significant association is found, it is of course not possible to distinguish between potential explanations for the link. Drugs or alcohol may be taken to obscure a pre-existing intention to have unsafe sex or to reduce social anxiety making it more possible to meet a sexual partner, their use may have a direct effect by reducing inhibitions and increasing arousal, or both unsafe activity and drug use could reflect a third variable such as desire for excitement or risk taking personality. Any link could also be merely coincidental since sexual encounters often begin in situations where drugs and alcohol are available.

A few investigations have used more sophisticated designs to try to clarify these questions. A series of studies by Gold et al (Gold et al, 1991; Gold & Skinner, 1992; Gold et al, 1994) using four different samples of gay men including a seropositive group examined factors which distinguished between safe and unsafe encounters within the same subject. Each encounter was divided into four temporal stages and hence it was possible to conclude that the extent to which respondents had been ‘stoned’ at any stage of the evening and the quantity of substances they had used during sex, did not distinguish between safe and unsafe encounters in any of the
samples. The same was also found in relation to alcohol with the exception of one sample of HIV negative men in whom level of alcohol intoxication was higher for the unsafe encounters (Gold et al, 1994). The authors note that among this group, neither the desire to have a drink nor to get drunk distinguished between safe and unsafe encounters. Further, among those men who at the start of the evening and at the start of sex had desired unprotected intercourse to exactly the same extent in both safe and unsafe encounters, level of intoxication was still higher in the unsafe encounter suggesting that alcohol was not deliberately consumed to facilitate unprotected intercourse nor could it have produced a greater desire for unsafe sex. Gold himself acknowledges (Gold, in press) that these retrospective accounts may not be accurate reflections of the thoughts and behaviours which occurred in the actual situation. He argues however that it is difficult to see how it can be empirically established that they did occur and that the reports are not simply rationalisations invoked after the event. He suggests that the only evidence might come from an intervention study which extinguishes both the reported cognitions associated with unsafe sex and the frequency of unprotected intercourse, a suggestion which has been taken up in our department (see Clinical Dossier, below).

Perry M. et al (1994) asked 1519 gay men to report on the frequency of four behavioural situations in the previous two months: the number of times intercourse occurred with and without condoms both after drinking and when no drinking was involved. They also identified problem drinkers and use of psychoactive substances. Men who had unprotected intercourse after drinking drank more, scored higher on problem drinking and had more unprotected intercourse incidents compared with those having unprotected intercourse but never after drinking. There were no differences in use of psychoactive drugs or number of protected intercourse incidents so that findings were not confounded by overall frequency of intercourse or by the effects of other drug taking. An odds ratio was calculated for unprotected incidents after drinking for the whole sample which suggested that it was less likely after alcohol consumption. Odds ratios were then computed for problem drinkers versus non problem drinkers, for exclusively partnered versus not exclusively partnered and for
three levels of frequency of intercourse and each of these risk estimates also indicated that drinking prior to sexual activity was associated with a lower likelihood of unprotected intercourse. Heavy alcohol use in general and frequent high risk sexual behaviour occurred in the same individuals, echoing the association reported in many other studies, but there was no evidence of a causal link between alcohol use before sexual activity and unprotected intercourse.

It is interesting to note that studies of predominantly heterosexual individuals in alcohol treatment programmes in the USA have found high rates of HIV seroprevalence, much of which is not related to injecting drug use, the main risk factor for heterosexuals in that country (Avins et al, 1994; Mahler et al, 1994). This again suggests that the link between HIV risk behaviour and alcohol is via problem drinking rather than through a direct relationship of alcohol intake and unprotected sex. In summary, the evidence currently available is more in line with the idea of one or more variables influencing both substance and alcohol use and high risk sexual behaviour than with that of a causal relationship.

4. 2. Communication and sexual negotiation skills

A second hypothesis is that men who engage in unsafe sex have difficulty in communicating their wishes about condom use to their partner. Gold's studies (1991, 1992, 1994) show that verbal communication regarding use of a condom occurred significantly more often in safe than in unsafe encounters and multiple logistic regression showed that poorer assertiveness skills in negotiating safer sex predicted relapse to less safe practices in a large longitudinal study (Adib et al, 1991). Other studies have also reported an association between poorer sexual communication skills and unsafe sex (Hays et al, 1990, 1992) and between improvement in the ability to talk to partners about sex and condom use and behaviour change in the direction of safer sex (Centers for Disease Control, 1991). In a recent study, 20% of the respondents reported that they had been verbally coerced into unprotected intercourse (Rompa et al, 1994) implying that some individuals lack the ability to resist such coercion.
One might speculate that poor sexual negotiation skills could be related to fear of rejection, social anxiety and low self esteem. There is some evidence that a strong desire to please one's partner and a belief that condoms imply lack of trust are associated with lapses from safer sex (Stall et al., 1990; Kelly et al., 1991a). Fears that their partner would not like them if they insisted on condom use were among the more common justifications for unsafe encounters reported by respondents in another study (Grant et al., 1991). Although clinical experience suggests that low self esteem plays an important part in facilitating high risk sex for some individuals, the only evidence available is contradictory: low self esteem was associated with unsafe sex in one study (Horn and Chetwynd, 1989) but not in another (Perkins et al., 1993) nor was it associated with lapses from safer sex in a longitudinal study (Adib et al., 1991).

It could be argued that if prospective partners disclosed their HIV status, an appropriate decision about condom use would be more likely. Several studies however indicate that the majority of gay men engaging in unprotected intercourse are unaware of their partner's status (Dawson et al., 1994) even when this occurs with a regular partner (McLean et al., 1994). In a study of voluntary self disclosure of serostatus among 129 HIV positive adults of whom 104 (81%) were gay men, after an average of 2.3 years, one third had not disclosed their status to any present or past sexual partner (Perry et al., 1994). Marks et al (1994) found that while only 9% of a sample of 609 seropositive gay men had engaged in unprotected intercourse in the previous two months, 14 (2.3%) of these had done so with a total of 25 seronegative or unknown serostatus partners who were not informed of the risk.

There may be good reasons for HIV positive men to avoid disclosing their status: in a large study of gay men's partner preferences, seropositive individuals were much less likely to be desired for either romance or friendship by men who were seronegative or who had not been tested (Hoff et al., 1992). HIV positive men are more likely than HIV negative men to believe that disclosure should only occur under certain circumstances, are less likely to volunteer their status and more likely to have been rejected by anonymous partners because of it (Klitzman & Bayer, 1994).
Infected men who fear rejection may rationalise their non disclosure and unsafe sex by the thought that they are not responsible for their partner, as was found by Gold et al (1994). A factor which emerged prominently in all Gold’s samples except in the HIV-infected group, involved inferring from perceptible characteristics, eg. ‘He looks too healthy to be positive’, that the partner was uninfected. The respondents were very well informed about HIV infection and it seems implausible that they were unaware of its long incubation period. One possible explanation for this kind of automatic thinking is that it occurs as part of an avoidance of the more difficult task of discussing condom use or serostatus or to make unsafe sex more acceptable.

4. 4. Mood and personality dispositions

Although mood is a factor which could be expected to affect the occurrence of unsafe sex, the evidence available does little to help clarify whether and how it does so. In the Gold studies, participants were asked to indicate the extent to which they had been in each of eight moods which included ‘depressed’, ‘relaxed’, ‘under stress’ and ‘bored’ at the start of the evening in which the sexual encounter took place. Mood distinguished between the safe and unsafe encounters in all the samples but was more negative in the unsafe encounters only in the sample of young gay men: all three older groups, including the HIV infected men, reported better mood for the unsafe than the safe encounter. Any inferences about these differences would be speculative but interestingly, the first factor to emerge from a factor analysis of the infected group’s cognitions during the unsafe encounter involved reactions to a negative mood state. The mood measure in these studies was evidently intended reflect a state rather than a trait characteristic and one interpretation of the finding among the HIV positive men is that mood changed over the course of the evening.

McKusick et al (1990) have also reported a positive association between low levels of depression and high risk sex. In addition, two studies looking at lapses from safer sex suggest that low mood is not a risk factor for unsafe sex. Kelly et al (1991 b) found that lower levels of depression predicted vulnerability to lapses and de Wit &
Griensven (1994) reported that time from safer to unsafe sex was independent of both depression and psychological wellbeing. On the other hand, feeling depressed was one of three barriers to condom use most frequently reported in a survey of 964 gay men (Sandfort et al, 1994). Three studies of HIV positive groups provide contradictory results. Condom use for anal intercourse was negatively correlated with depression scores on two measures in a small sample of asymptomatic men (Kelly B. et al, 1991) and Kelly et al (1993) found that depression and recreational drug use were significant predictors of high risk sexual behaviour. By contrast, Robins et al (1994) report that seropositive men engaging in risky sexual practices had lower levels of psychological distress than those who were not risky. It is difficult to account for these differences but in the two studies finding associations of risk with depression, the investigators used measures specifically designed to assess depressive symptoms while in the Robins et al study, depression was indexed by scores on one subscale of the Symptom Checklist-90 (Derogatis et al, 1983). In addition, the clinical status of the subjects in this study is not described. These findings do not allow any firm conclusions about the possible effects of low mood on risk behaviour however the presence of mental health problems may be relevant. A study of young gay men over a two year period showed that one of the factors distinguishing the small proportion engaging in very high risk behaviours from other risk takers in the sample was the presence of mental health problems (Meyer & Dean, 1995).

One could suppose that transient mood changes may exert an effect in the sexual situation itself but it also seems likely that individual differences in relatively stable characteristics such as personality dispositions and coping strategies are relevant in determining a person's sexual behaviour. Desire for excitement through sex distinguished between safe and unsafe encounters among HIV infected men only (Gold et al, 1994) and the authors speculate that sexual adventurousness may have led to their infection. In a recent report from a longitudinal study, sexual adventurism assessed on a scale based on Kalichman et al's (1994) measure of sexual sensation seeking was an important predictor of HIV infection. Almost 80% of those seroconverting in the 9 year period under review scored above the median on sexual
adventurism (DiFranceisco et al, 1996). There is some evidence to suggest that gay HIV infected men have a higher prevalence of personality difficulties as assessed by DSM III-R than those who are negative (Perkins et al, 1994) and in another study of hospital in-patients, which included a small number of heterosexuals, borderline personality disorder was diagnosed more frequently among those who were HIV positive than among controls (Ellis et al, 1993). Two recent reports link a history of sexual abuse with current high risk sexual behaviour. Strathdee et al (1996) found that non consensual sex as a youth or young adult was associated with a two-fold increase in sexual risk taking among young seronegative gay men. In a very large sample of almost 2000 gay men, over a quarter reported a childhood sexual abuse experience and significantly more of these men engaged in risky sexual behaviour than those without such a history, particularly if the abuse involved strong coercion (Jinich et al, 1996). There is evidence that sexual abuse in childhood among males may have a wide range of effects including depression, poor self concept, difficulty in establishing and maintaining relationships, sexual dysfunction and sexual compulsion (Cahill et al, 1991).

CONCLUSIONS

Many gay men continue to practice unsafe sex although they are aware of the risk of HIV transmission. In addition to demographic factors, perception of risk, relationship factors and social norms, it is clear that interpersonal factors, cognitive events and processes also play a significant role. Some of these factors are likely to be related to more enduring personality dispositions and to act in interaction with situational variables. These possibilities are only recently beginning to be addressed.

Many factors but perhaps two in particular hamper research efforts in this area. Firstly, individuals are not either 'high risk' or 'safe' but may change their behaviour over time or behave differently according to the prevailing circumstances which may be determined by internal, external or interpersonal factors. Secondly, investigations into variables which may operate in the immediate sexual situation such as mood,
expectations or thoughts have to rely on self report obtained under very different conditions. Indeed, even the data on the main dependent variables of safe and unsafe behaviour are largely self reported and it is difficult to see how these limitations can be overcome satisfactorily. The era of large scale cross sectional studies may have come to the end of its useful life. As Fisher et al (1995) have pointed out, many studies have not used a theoretical framework resulting in the accumulation of relatively unintegrated findings. There is now an increasing use of social psychological theories to structure investigations, in particular the theory of reasoned action (Ajzen & Fishbein, 1980) and theory of planned behaviour (Ajzen, 1991). Although this is welcome, it is also important not to lose sight of individual differences which may influence the dimensions in these models. Careful investigation of psychological processes on an individual basis is likely to prove fruitful in yielding hypotheses which can then be tested on a larger scale and used to inform interventions for those who are unhappy with their HIV risk sexual behaviour.

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3. CLINICAL DOSSIER
In this section, the development of a new service is described from the initial successful bid for funding to outcomes after four years. During this period, a number of service related research projects were carried out by the author and other members of the department some of which have been presented at conferences, submitted to journals or published. Following an introduction to the context in which the service was initiated, the report is divided into two parts. In section 1, the needs of healthcare staff are addressed and in section 2, the clinical service is described. Wherever possible, the basis for what was done is related to research findings and difficulties encountered are discussed.

INTRODUCTION

The HIV/Genitourinary Medicine directorate of Chelsea and Westminster Healthcare has three clinics in central London: the Victoria Clinic in Westminster, the Genitourinary Medicine (GUM) clinic at Charing Cross Hospital in Hammersmith and the St Stephens Centre at Chelsea and Westminster Hospital in Fulham which also houses the two in-patient wards for patients with HIV disease. These parts of London have a high prevalence of HIV infection and it is the leading cause of death among young men resident in the areas covered by the clinics (Aldous et al, 1992). The population attending the clinics is diverse although, among those with HIV infection, most (82%) are gay men. Within St Stephens, the Kobler Clinic is specifically for HIV seropositive individuals while in the Victoria and Charing Cross clinics, HIV infected people are seen in the general GUM clinic. In 1995/6, 80,000 attendances at the clinics were recorded of which 26,000 were by people with HIV infection and currently, the three clinics are treating more than 2000 HIV infected people, the largest group of seropositive individuals in the UK.

The Psychological Medicine Unit, Riverside Mental Health Trust provides mental
health care to patients of the HIV/GUM clinics. This service began in 1989 with the appointment of a consultant psychiatrist who was joined in 1991 by the author as consultant clinical psychologist. Subsequently the team grew following successful development bids and currently has three psychiatrists, two liaison nurses and five clinical psychologists.

In the United Kingdom, the largest proportion of AIDS cases and HIV infections have consistently been reported from the Thames regions. Most of these are among gay men and there is evidence of a continuing high incidence among this group in London (Evans et al, 1993; Miller et al, 1995). As is well known, HIV infection is rising rapidly in the heterosexual population although numbers remain relatively small (Communicable Disease Report, Feb 1997). Prevalence among heterosexual GUM clinic attenders in London is increased by a factor of six for males and twelve for females compared with the rest of the UK (Communicable Disease Review, Jan 1996). There is also evidence that substantial numbers of HIV positive individuals are attending for treatment of new STDs (Catchpole et al, 1996) and that others known to the clinics and with a history of previous negative HIV tests are returning to test positive (Gray et al, 1997): some of the consultant physicians in our local clinics had indicated their concern about evidence of risk behaviours among clinic attenders. Studies from the United States carried out in the late 1980s showed that cognitive behavioural interventions were effective in helping individuals to reduce or eliminate HIV risk sexual behaviour (Kelly et al, 1989; 1990).

With all these factors in mind, a joint bid made by the Psychological Medicine Unit and Chelsea and Westminster Healthcare HIV/GUM directorate for HIV prevention monies in 1992/3 resulted in funding being made available for an HIV sexual risk reduction service. It was agreed that this service would be managed from within the Psychological Medicine Unit by the author and staffed by one clinical psychologist and one project worker. Since the bid was made in collaboration chiefly with a senior health adviser from the HIV/GUM directorate, it was originally envisaged that the project worker would have the background and experience usually found among
health advisers at that time. Many came from a nursing or social work background, others were social sciences or psychology graduates, some had counselling qualifications, and all had a good deal of experience of working in the HIV or GUM fields.

**Aims of the service**

These were two fold:

1. To provide psychological interventions for people having difficulty in adopting or maintaining safer sex.
2. To identify and seek solutions for any difficulties among HIV/GUM staff in dealing with those at risk.

**Organisation**

A small steering group was set up in the early part of 1993 with representation from senior clinicians and managers from both trusts. The functions of this group were:

i. to set priorities and overall strategy for the service
ii. to monitor activity
iii. to facilitate the work of the HIV risk reduction team.

While acknowledging the need for community level interventions such as those provided by the Stop AIDS London initiative, it was agreed that the population attending the clinics the majority of whom are, by definition, at some risk of contracting HIV infection were to be the primary target for psychological intervention. HIV positive gay men were identified as the highest priority. The service would take referrals from any member of staff in the clinics, self referrals were to be encouraged and access should be made as easy as possible. Individual and group meetings with HIV/GUM clinical staff having regular and frequent contact with clinic attenders were to be held initially to gain an understanding of staff needs and
to publicise the new service.

A clinical psychologist and a project worker were appointed and they took up post in April, 1993.

Comment: One of the first problems encountered was recruitment to the clinical psychology post which was seen as a priority. It was necessary for the post holder to have not only good cognitive behavioural skills but also academic, presentational and social skills. In 1993, HIV/GUM services and clinical research within them were both expanding rapidly, attracting clinical staff of high ability and nowhere more so than at the newly opened Chelsea and Westminster Hospital. In this atmosphere of exciting new developments and constant change, with a focus on medical treatments, it was important to make a clinical psychology service for prevention relevant and credible or it could quickly be brushed aside and forgotten. Skills and confidence in communication were required together with a non judgmental approach to a population from many different cultures and lifestyles. At the time, several clinical psychology posts within the Mental Health Trust remained unfilled and there was a real risk that, if a suitable candidate was not found, the post would be deleted and a second project worker recruited instead. In the event, although the response to the advertisement was poor, it was possible to appoint a candidate meeting the criteria outlined above.

Many of the qualities required of the clinical psychologist were also relevant for the project worker but in this case, the expected large response to the advertisement for the post occurred. The job description and person specification were written at an early stage by the senior health adviser before the management arrangements were finalised and included responsibilities for patient contact, which would not conform to those found for example among the tasks of an assistant psychologist. The difficulty for the clinical psychologist manager was in anticipating ways of supervising and structuring the work of the project worker who was likely to come from a different discipline. Once again however, a problem was avoided rather than solved.
in that the interview panel, with equal representation from Psychological Medicine and HIV/GUM, selected a candidate who was a psychology graduate and had just completed a PhD. When this post holder left in the summer of 1994 to do clinical training, the post and job description was changed to that of an assistant psychologist.

A second difficulty was in deciding how best the two post holders could work together. Clearly the clinical psychologist should take the lead in planning and providing the interventions but the project worker should not be perceived as a lesser partner in the team, particularly as he had experience of counselling in a setting for people at high risk of infection and considerable knowledge about HIV infection and prevention. It was decided that he would take the lead in researching and meeting staff needs, including ways of identifying individuals who could benefit from intervention. One was aware that this was potentially a difficult task for a non-clinician in an environment of highly skilled healthcare staff.

Finally, even at this early stage, one was also aware of a probable conflict of interest with the health advisers in the clinics. This professional group was growing rapidly and, as the providers of HIV test counselling, its members were seen as the experts in matters of safer sex. There was potential for an implied criticism of their work, particularly as any effect of HIV test counselling on HIV risk behaviour has not been demonstrated (Zenilman et al, 1992; Otten et al, 1993). Before the development of a mental health team, the health advisers had also been seen as the providers of counselling and it was important to manage this new apparent encroachment on their role so that we could work successfully with them. Health advisers see all those coming for HIV tests, most of whom have been engaging in risk behaviour as well as very many others presenting with STDs who have also been at risk and clearly, they would be a major source of referrals.

It was important in managing these relationships to have and maintain the interest and support of the senior health adviser, the HIV/GUM manager and one of the consultant physicians, all three of whom attended steering group meetings.
1. **STAFF INTERVENTIONS**

While there is a large literature on factors associated with HIV risk behaviours (Thornton and Catalan, 1993), little relevant information was available to guide approaches to the clinic staff and it was necessary to begin with qualitative information gathering before more formal methods of needs assessment could be used.

We hypothesised that there could be a number of barriers to clinic staff raising safer sex issues with patients and to making referrals for risk reduction. In the first instance, some patients do not acknowledge that they engage in risk behaviour or, if they do, they do not regard it as a problem or they do not want any help in changing it. The recognition that a problem exists needs to precede thoughts about action to solve it (Prochaska & Di Clemente, 1982) and clinic staff can become demoralised by the lack of response to health promotion messages of those they know to be at risk. They may be unwilling to risk deterring patients from attending for treatment, if they perceive that suggestions about sexual behaviour change are unwelcome. They might have difficulty in integrating a treatment and a prevention role and they may not have the skills to do so. This is certainly relevant in the case of patients who are already HIV infected. There is a reluctance on the part of clinic staff, who develop close relationships over a long period with HIV positive patients, to discuss safer sex with them. When one considers the fact that most of these patients are young people with a terminal illness who have to withstand many losses, often require unpleasant and lengthy medical treatments and usually belong to groups that have experienced social rejection, this reluctance is understandable particularly since most of the patients have acquired HIV through sexual transmission.

While most seropositive individuals are responsible in their sexual relationships, some are continuing to place themselves and others at risk. The reluctance of HIV clinic staff to raise safer sex issues is apparently shared by the HIV positive patients who
tend to seek treatment for an acute STD at a clinic different to that where they receive care for their HIV infection (Catchpole et al., 1996). This is known to occur at the St Stephen’s Centre, where Kobler Clinic patients will go to the general GUM outpatient clinic two floors above the Kobler which has separate medical notes, without their Kobler HIV physician being necessarily aware that they have contracted an acute STD.

More practical reasons for not discussing safer sex could be lack of time or the perception that it is not part of one’s role. Political and economic reasons might also play a part: purchasing agencies in the area covered by the clinics reduced their spending on health promotion so that cuts in provision had to be made which included a drastic reduction in the budget for free condom distribution in the clinics. This resulted in resistance in principle to absorbing any of this work which was felt to constitute an admission that the funding was not necessary. Another consideration is that in the competitive climate of the NHS today, it is necessary to demonstrate high levels of attendances and treatments in order to justify and attract funding. Prevention activity might be seen as a low priority or even as a deterrent to potential attenders or as a distraction from the medical and nursing activity of the clinics. Finally, clinic staff may not refer patients for risk reduction because they do not believe that psychological factors are relevant or because they do not believe that psychological intervention is effective.

1.1. Publicity and Information about the Service

Initially, efforts to publicise the new service were made. In addition to informal meetings with HIV/GUM staff, formal presentations were made to health advisers, junior doctors, nursing staff, and multi professional groups on all three clinic sites. The purpose of these was to explain ways in which a psychological approach could inform intervention and to discuss mechanisms for referral and access to the service. Publicity materials were produced and distributed in the clinics. The two members of the team were provided with bleeps and it was arranged that referrals could be made
verbally rather than by letter. In addition, as well as providing appointments in the mental health centre on the Chelsea and Westminster site, sessions within the clinics were organised for the clinical psychologist.

1.2. Staff Discussion Groups

Discussion groups were held with nursing staff and junior doctors at St Stephens Clinic during 1993, each group meeting on two occasions. The aims of the meetings were to explore the perceptions of staff about high risk sexual behaviour among their patients, the ways in which these issues were dealt with and ideas about any difficulties experienced and how they could be overcome. It was also hoped that by highlighting prevention during the discussions, referral of patients to the risk reduction service would be stimulated, in particular from the one dedicated HIV clinic, the Kobler Clinic. Some useful suggestions emerged from these meetings, which included the development of a checklist of areas to discuss with patients and the need for an increase in the salience of safer sex materials in the clinics which could serve as a cue for patients and staff to raise the issue.

1.3. Surveys of Staff

Two surveys of staff were undertaken. In the first, carried out in 1993, a questionnaire was sent to all clinical staff at St Stephen's Centre and the HIV wards (n=115). The aims were to investigate the extent of discussion of HIV risk sexual practices with patients and how able members of staff felt to do this; to identify factors thought to be important in patients engaging in unprotected sex and to assess levels of knowledge among staff about risk of transmission of HIV.

Main findings:

There were 45 respondents: doctors 20; nurses 19; health advisers 5; social worker 1. This was a poor response rate (39%) and the survey results could not be regarded
as representative. The majority (92%) saw the discussion of safer sex with clinic attenders as part of the remit of their job and felt comfortable (90%), confident (89%), and skilled (89%) in doing so, although almost half felt that those in other professions were better qualified to do this. About half felt that there was insufficient time to ask all their patients about safer sex and over half the respondents (56%) had had no specific training in this area. More than three quarters (78%) felt they would benefit from further training with approximately equal proportions among doctors and nurses.

The majority of staff accurately identified high risk sexual practices but a minority revealed serious misunderstandings by endorsing practices well known to be associated with HIV transmission as low risk.

In reply to the question: "From your own experience, we would like you to list factors that you believe are important among the patients that you see in their not practising safer sex". A total of 139 responses were grouped into eight categories. Beliefs about why patients had unsafe sex included alcohol or drug use, negative aspects of safer sex, unhelpful or mistaken beliefs about risks, and relationship factors. The largest category however, referred to characteristics of individuals such as low self esteem, social anxiety or stress. Many of these characteristics are potentially amenable to psychological interventions and the findings suggested that the latter are appropriate in this setting and acceptable to HIV/GUM staff (Flynn et al, 1994).

A second survey focusing specifically on knowledge of HIV transmission risks was carried out during the following year (Holder et al, in press). Since clinic staff are regarded by the general public as reliable and specialist authorities on the sexual transmission of HIV infection, it was important to determine their knowledge of the relative risks of different sexual activities on the assumption that this knowledge directly influences the provision of information to patients. In the earlier survey, respondents were required to rate the risks of a range of sexual activities in the
context of combinations of gender, sexual orientation and HIV serostatus. The length and complexity of the questionnaire however, resulted in a relatively high rate of partial completion and obvious errors, and was perhaps responsible for the low rate of return.

For the second survey, the questionnaire was simplified in order to maximise reliable responses. It began with the following statement: "Imagine you are informing an HIV negative patient of the risk of HIV transmission only from their HIV positive partner". Respondents were then asked to rate the risk of each of 21 different activities described to them by the patient on a seven point scale, ranging from 0 for 'no risk' to 6 for 'high risk', with the middle points corresponding to 'low risk' and 'medium risk'. The items covered a range of activities including penetrative anal, vaginal and oral sex, protected and unprotected, with and without ejaculation, during and not during menstruation, with and without bleeding gums, contact involving probable trauma of mucosal surfaces, use of sex toys and contact with urine or faeces. The hypothetical patient was characterized as receptive in some of the activities and active in others. The questionnaires were sent to all clinical members of staff in the HIV/GUM directorate (n=207) for anonymous completion and information was gathered on respondents' gender, occupation and sexual orientation.

Main findings:

(i) demographics

106 questionnaires were returned, a response rate of 51%. Fifty-one (48%) of the respondents were nurses, 27 (26%) doctors, 13 (12%) health advisers, one (1%) an occupational therapist, and 14 (13%) did not indicate their profession. Fifty-eight (55%) were female, 37 (35%) male and 11 (10%) did not indicate their gender; 53 (50%) were heterosexual (42 female, 11 male), 36 (34%) gay (25 gay males, 11 lesbians) and 17 (16%) did not indicate their sexual orientation.
(ii) **distribution pattern of response ratings**

The questionnaire ratings revealed two patterns of scoring. The first can be described as a tendency by some respondents to rate activities as either a high risk or low/not a risk; that is, to rate an activity as falling at either extreme of the rating scale, with no use of the 'medium risk' ratings. In the second pattern, activities were apparently rated according to their relative risk, and thus all points on the scale were used. This in itself indicates an inconsistent approach and suggests that patients will receive different information depending on the member of clinic staff with whom a discussion of sexual risk behaviour takes place.

Response distributions for risk ratings on each question were examined and four patterns could be identified: highly skewed distributions indicating high agreement; flat distributions indicating lack of agreement; normal or skewed normal distributions indicating poor agreement. Univariate analyses were carried out on risk ratings for the questions with low agreement in order to examine if there was any systematic variability according to profession, gender or sexual orientation.

(iii) **summary**

While there was generally good agreement on the well known high risk activities of unprotected anal and vaginal intercourse, there was poor agreement about other activities as reflected in the distributions of responses to 16 of the 21 questionnaire items. This lack of agreement was most pronounced for questions involving excretory and traumatic contact. One could speculate that these kinds of activity may be less commonly discussed among clinic staff and in the literature, resulting in greater uncertainty about the risks involved. If this is the case, it is possible that clinic attenders are similarly hampered in discussing and obtaining information about the risks attendant on more unconventional sexual practices. There was also poor agreement about various sexual activities involving oral contact presumably reflecting the uncertainty about orogenital contact as a significant risk factor in HIV transmission (Department of Health, 1994).
More than one third of our sample identified themselves as gay or lesbian but only one difference in risk ratings according to sexual orientation was found, for an item referring to an activity which is not specific for sexual orientation.

Health advisers were significantly more likely to rate 11 of the 21 activities as riskier than nurses or doctors and this suggests that risk ratings decrease as contact with patients for specific discussion of sexual risk activities increases. It could be that, since health advisers have the responsibility of being the experts on sexual safety, their perceptions of risk are more accurate than those of doctors or nurses. On the other hand, it is conceivable that health advisers, with their greater familiarity with discussing HIV sexual transmission factors over long periods of time, come to downgrade risk for some activities in favour of emphasising more definite high risk behaviours.

Finally, men were more likely to rate some activities as lower risk than women, including unprotected vaginal intercourse. This is interesting in view of the well established finding that women are more at risk than men in sero-discordant couples by a factor of two (European Study Group on the Heterosexual Transmission of HIV, 1992).

These findings indicated a considerable variability in perception of risk among the clinic staff and suggested that the information given to patients is not consistent.

1. 4. Teaching and presentations

Regular presentations were made to clinical staff about the findings in the staff surveys and the progress of the clinical intervention service. Data from other relevant studies carried out in the department and audit findings were also presented. For example, a retrospective audit of attenders was carried out which showed that over a 5 year period, 22% of first time HIV positive gay men diagnosed at St Stephens Centre had at least one previous negative HIV result recorded at the clinic suggesting
that an opportunity for prevention had been missed (Gray et al, 1997). More detailed studies of seroconverters and repeat testers are now under way (see Conclusions, below).

In another audit, the notes of GUM clinic attenders on 4 consecutive days in each of the three clinics were screened for evidence of risk behaviour according to agreed criteria. The notes of those at risk were tagged with a brief precoded questionnaire to identify whether or not they were referred for risk reduction and the reasons for non referral. The audit was constrained by the way in which the clinics operate so that it was not possible to include new attenders nor those attending for HIV tests who may have been at higher risk. The figures show that only 3% of the attenders screened were at high risk, and that among these half were offered the risk reduction service. Among those who were not offered referral, the reason endorsed by the doctor was ‘difficult to bring up’ in almost half the cases again suggesting that further training might be appropriate.

The results of a study of sexual behaviour and perception of HIV transmission among gay men attending the Kohler clinic were presented in an attempt to raise awareness of the relevance of risk reduction among HIV seropositive individuals (Thornton et al, 1996; Shah et al, 1997). Almost two-thirds (64%) of the sample of 349 reported practising anal intercourse and more than one-third (38%) reported inconsistent or absent condom use with their regular partners while for casual partners, the proportion was 24%. A substantial minority (14%) perceived the maintenance of safer sex as a problem for them. There was a positive relationship between condom use and perception of HIV transmission risk but less than one quarter (24%) of the men were aware that certain stages of infection are associated with a greater or lesser risk of transmission. These results suggest that a substantial minority of HIV seropositive gay men continue to have unsafe sex. It seems however that in spite of this evidence, clinical staff caring for those with HIV infection remain unwilling to address the issue of sexual risk behaviour with their patients.
Presentations and workshops are also given on a regular basis to general practitioners who attend a three day course on HIV infection at the Chelsea and Westminster Hospital and to the junior doctors as part of their induction.

1. 5. Summary: Staff interventions

The activities of the HIV risk reduction team have helped to highlight the importance of prevention by clinic staff among HIV/GUM attenders. We sought to address some of the difficulties that were hypothesised to exist for staff in intervening to prevent the spread of HIV infection. In 1996, a Sexual Health Promotion Strategy Group was established in the HIV/GUM directorate with representation from our service. This group has identified a lack of documentation of safer sex discussions and as a consequence of this, the failure at times to follow up on earlier recommendations. There may be a tendency to assume that risk behaviours have been discussed and dealt with where this is not the case, especially if the individual is a repeat attender. The strategy group has begun to clarify and implement a structure for consistent documentation of health promotion and for training in the information given to patients.

2. CLINICAL SERVICE

2. 1. Introduction

When the service began, the available literature suggested that outside of the important work concerning community level interventions, group interventions based on cognitive behavioural principles were effective in reducing HIV sexual risk behaviour among gay men (Kelly et al, 1989). In fact, a recent review (Oakley et al, 1995) confirms that Kelly et al’s study remains one of the few well designed evaluations of an intervention which is demonstrably effective. The intervention they describe consists of a 12-week programme which includes the following elements:
education about HIV risk, identification of triggers to high risk behaviours, teaching of cognitive and behavioural skills to manage high risk situations, assertiveness training to negotiate sexual encounters and deal with risk coercion, and problem solving relating to lifestyle and social supports. Self reported frequency of unprotected anal intercourse declined to near zero levels and condom use showed a significant increase while members of a waiting list control group showed little change. These changes were maintained at sixteen month follow up. Kelly et al (1990) later reported on an 8-week version of the programme with similar positive effects on risk behaviour. Given the large numbers attending the clinics, it was expected that group interventions would be the most useful and cost effective way of delivering the risk reduction service. It was planned that initially all persons referred would be seen on an individual basis and that groups would be run at a later stage.

Comment: Our experience has been that firstly, a relatively small number of individuals are referred, a large proportion of whom fail to attend, or drop out of treatment after one or two sessions. Secondly, among those seen, many have current and past psychological or psychiatric problems and a significant proportion have long standing personality problems. In addition, although HIV positive gay men were identified as a priority for intervention only a small number have been referred. Initially, the presence of the clinical psychologist in the clinics stimulated referrals, but many of these were for individuals with psychological disorders rather than risk behaviour to whom it was nevertheless necessary to offer intervention or appropriate referral.

2.2. Referrals

Table 1 shows the outcome of the total number of referrals made to the service from April 1993 to March 1997 and demonstrates that only 27% of those referred completed intervention. Nevertheless among those who were appropriately referred and assessed, and excluding those who are still being seen or awaiting appointments, 63% completed treatment. Those referred on were mainly individuals with substance
use problems or those requiring psychiatric assessment.

Table 1. Outcome of referrals: April 1993 to March 1997

<table>
<thead>
<tr>
<th>Outcome</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Did not attend</td>
<td>52</td>
<td>20</td>
</tr>
<tr>
<td>Inappropriate referral</td>
<td>51</td>
<td>19</td>
</tr>
<tr>
<td>Referred on</td>
<td>16</td>
<td>6</td>
</tr>
<tr>
<td>Dropped out</td>
<td>56</td>
<td>21</td>
</tr>
<tr>
<td>Completed treatment</td>
<td>71</td>
<td>27</td>
</tr>
<tr>
<td>Ongoing/awaiting assessment</td>
<td>16</td>
<td>6</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>262</td>
<td>100</td>
</tr>
</tbody>
</table>

Referral rate has remained relatively constant at between 52 and 67 referrals per 12 month period. The number of inappropriate referrals has decreased from a peak of 33% in 1994 to its current level of 14% as a result of efforts to make guidelines for referrers clearer. The numbers dropping out of treatment have also been reduced from 31% in the first year to 18% in the last 12 months. On the other hand, those failing to attend their first appointment has increased to one third in both of the last two years: this figure reflects the high non-attendance of HIV/GUM patients for mental health first appointments generally which is currently 25% for the whole department.

The average age of patients was 32 years (range 18-72) and 44 (17%) were female.
Table 2 shows the sexual orientation of the total patients referred and of those referred in 1996/1997 and shows that proportionately more gay men were referred in the last year with a consequent reduction in the proportion of women which was only 8%. This reflects our policy of targeting gay men in view of the recent epidemiological evidence of a continuing high incidence of HIV infection among gay men in London (Miller et al, 1995).

Table 2. Sexual orientation of those referred

<table>
<thead>
<tr>
<th></th>
<th>Total 1996/7</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
</tr>
<tr>
<td>Gay</td>
<td>155</td>
</tr>
<tr>
<td>Bisexual</td>
<td>14</td>
</tr>
<tr>
<td>Heterosexual</td>
<td>66</td>
</tr>
<tr>
<td>Unknown</td>
<td>27</td>
</tr>
</tbody>
</table>

The numbers of HIV positive individuals referred have remained constant at 15% but only 3 referrals in total have been received from the Kohler clinic. The majority of those seen were of white UK ethnic origin (64%) and 26% were white from other countries. Only 10% were from minority ethnic groups, reflecting the population seen in the clinics most of whom are white.

Although every effort has been made to encourage self referrals by producing different kinds of publicity materials and emphasising the confidentiality of the
service, these have remained disappointingly low: 4% in the first year and 3% in the current year. By contrast, we have been successful in developing good working relationships with the health advisers who referred 64% of the patients in the last 12 months.

2.3. Psychological disorder

Among the first 50 patients assessed for intervention, we found that more than half (57%) had a past history of treatment for psychological or psychiatric disorders. Caseness as assessed on the Hospital Anxiety and Depression Scale (HADS, Zigmund & Snaith, 1983) was 46% for anxiety and 14% for depression with 18% and 27% scoring in the possible caseness range for anxiety and depression respectively (Thornton et al, 1994).

It was felt that standard measures of psychological morbidity did not capture the mixed symptomatology and difficulties seen in many of the patients and we therefore administered the MMPI-2 (Hathaway and McKinley, 1989) to 53 individuals who were consecutive referrals attending for their first appointment. Preliminary data was reported by Scragg (1995). The MMPI-2 is one of the most widely used tests of personality and is considered to be useful clinically for distinguishing between adjustment and abnormality (Helmes & Reddon, 1993). Mean T scores were abnormally high for 5 of the 10 clinical scales and the F scale, which forms part of the validity scales, and is an indicator of overall psychopathology (Meyer, 1993) was also raised above the cut off level. Two thirds of the sample had abnormally high mean T scores. This finding provides convergent validity for the clinical observation of severe and long standing psychopathology as well as personality problems among many of the risk reduction patients.

A study of GUM clinic attenders in London (Ellis et al, 1995) indicates that personality disorder is a significant predictor of sexual risk taking behaviour. In another study by Scragg (1995), 175 attenders at our local HIV/GUM clinics
completed the NEO Five Factor Personality Inventory (Costa & McRae, 1992) and a short questionnaire assessing self reported unsafe sexual behaviour. The main findings were of an association of risk behaviour with low agreeableness among heterosexuals and with low conscientiousness among gay men. These findings also suggest that intervention to promote safer sex behaviours may be complicated by personality factors.

2.4. Intervention

A cognitive behavioural model of risky sexual behaviour has been developed based on our experience (Tallis, 1995) and in particular, informed by the work of Gold (Gold et al, 1991; Gold & Skinner, 1992; Gold et al, 1994; Gold & Rosenthal, 1995) who has drawn attention to the powerful effect of cognitive processes, in the form of self justifications, on unsafe sexual behaviour. The model of risky sexual behaviour which is similar to others based on Beck's (1976) work provides a framework within which to identify the appropriate way of working with a particular individual and is shown in Fig 1. For some, the intervention focusses on the risk escalating or permission giving automatic thoughts which occur in the sexual situation and which tend to become more accessible with sexual arousal. For others, it may be necessary to work at the level of dysfunctional assumptions or core schemas (Young, 1990; Padesky, 1994). Behavioural techniques are an integral part of the approach. Many patients have social anxieties, assertiveness deficits and performance anxiety in sexual situations. Graded exposure, role play, condom practice and behavioural experiments are often useful. Some patients lack the basic condom skills necessary for their effective use and a condom demonstrator is routinely used to assess this.

One group intervention for HIV positive gay men has been conducted with one of the health advisers (Shah, 1996) and is currently being evaluated. Participants were recruited by self referral and the group was primarily focussed on relationship and social issues related to being HIV positive while also covering difficulties with safer sex.
FIG 1 - A COGNITIVE BEHAVIOURAL MODEL OF HIV RISK SEXUAL BEHAVIOUR

It is assumed that repeated unsafe sex is mediated by dysfunctional assumptions. These are often determined by early experiences, are entrenched, rigid and difficult to modify, e.g.:

"I will always be unhappy unless I find someone to love me"
"I cannot tolerate unpleasant feelings"

Automatic thoughts further back in time associated with mood change may determine high risk situations, e.g.:

"I feel low, I need company"
"I feel good and want to enjoy myself"

Risk escalating or permission giving automatic thoughts in sexually arousing situations and are likely to lead to unsafe behaviour, e.g.

Costs of condom use:
"If I use a condom I will lose my erection"

Inferences from partner's appearance:
"He is too young to be infected"

Emotional expression:
"If I don't use a condom, he'll know that I trust him"

Social Inhibition:
"If I use a condom, he'll think I am promiscuous"
2.5. Monitoring and Evaluation

During the first two years, data collection was developed and modified as experience was gained. Some measures used in the initial stages were abandoned as unsuitable (e.g. Impulsiveness Questionnaire, Eysenck and Eysenck, 1991), some were deliberately used for a short period only (e.g. MMPI-2) and others were introduced at a later stage (e.g. EPQ-R Short Scale, Eysenck and Eysenck, 1991). Although these changes mean that pre and post intervention data is inconsistent, it was felt that on balance, it was preferable to use appropriate measures at the expense of completeness of the data set. In addition, we were conscious that we were offering a clinical service to individuals whose motivation for change was sometimes uncertain. We wished to maximise uptake of the clinical intervention and avoid deterring patients with too many measures to complete.

Since cognitive events and processes were often the focus of intervention, it was necessary to develop an instrument to assess risk escalating thoughts and any changes following intervention. The Sexual Risk Cognitions Questionnaire (SCRQ) is a 22-item instrument with additional 8-12 item subsections designed for groups defined by gender, sexual orientation and HIV serostatus. It has good reliability and validity properties (Shah et al, in press). The items are rated on a 5 point scale from ‘never had thought’ to ‘very frequently had thought’. Some examples are given below:

‘I’m sure he’s HIV negative. I can just tell.’
‘I want to show him he is somebody special’
‘Unsafe sex is just one of life’s many risks’
‘My positive state of mind can prevent me from being infected’

The assessment measures routinely collected currently include self reported sexual behaviour in the last month; confidence in the ability to maintain safer sex rated on a 7 point scale from ‘not at all confident’ to ‘completely confident’; SCRQ; EPQ-R Short Scale, and the General Health Questionnaire-28 (GHQ-28: Goldberg & Hillier,
At the end of intervention, patients complete the sexual behaviour questionnaire, confidence rating, SCRQ, and GHQ-28. Follow up measures are the same omitting the GHQ-28.

**Comment:** Although it was planned to follow up all the patients at 3 monthly intervals for at least 6 months, this has proved unsuccessful. Only six have returned the follow up questionnaires and some have been returned indicating that the addressee has moved. All patients are asked for their permission for the questionnaires to be sent but the fact that so few are returned has raised anxieties about confidentiality.

One of the main difficulties in evaluating the effectiveness or otherwise of the intervention is the fact that the important outcome measures of sexual behaviour and confidence in the ability to maintain safer sex are necessarily self-reported. These outcome measures are backed up by inspection of the HIV/GUM database for each patient seen but absence of evidence of new STDs or of HIV seroconversion is not conclusive: many individuals are not consistent attenders at a particular clinic and may present at clinics in other parts of London. As mentioned above, this appears to be particularly the case among HIV-infected gay men. Even ‘hard’ evidence may not be as useful as it seems. Some of the patients are referred precisely because of their anxieties about recent risk behaviour and some are still in the window period when an HIV test will not reliably detect infection. Even if their behaviour changes, they may subsequently test positive because of risk behaviour before intervention.

### 2. 6. Outcome of intervention

Of the 71 individuals who completed treatment, pre- and post-treatment data is available for 45, 7 of whom were women. Table 3 shows changes in indices of risk behaviour reported for the previous month and in self efficacy about maintaining safer sex.
Table 3. Changes in risk behaviour following intervention

<table>
<thead>
<tr>
<th></th>
<th>Pre-treatment mean</th>
<th>Post treatment mean</th>
<th>z*</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of sexual partners</td>
<td>2.6</td>
<td>2.5</td>
<td>-1.2</td>
<td>ns</td>
</tr>
<tr>
<td>Frequency of penetrative sex</td>
<td>6.4</td>
<td>6.7</td>
<td>-2.7</td>
<td>ns</td>
</tr>
<tr>
<td>Frequency of unprotected sex</td>
<td>3.6</td>
<td>2.15</td>
<td>-3.1</td>
<td>.002</td>
</tr>
<tr>
<td>% times no condom use</td>
<td>46</td>
<td>19</td>
<td>-2.8</td>
<td>.005</td>
</tr>
<tr>
<td>Confidence in maintaining safer sex</td>
<td>2.5</td>
<td>4.4</td>
<td>-4.4</td>
<td>.001</td>
</tr>
</tbody>
</table>

* Wilcoxon Signed Ranks tests

The proportion endorsing the ‘completely confident’ pole of the self efficacy scale rose from 3 (6%) before treatment to 18 (40%) afterwards. Several studies indicate that self efficacy about the ability to be safe predicts low risk sexual behaviour (Aspinwall et al, 1991; de Wit et al, 1996) and perceived behavioural control is an independent predictor of condom use (Godin et al, 1996). The numbers reporting no unprotected penetrative sex in the previous month rose from 11 (24%) before intervention to 24 (53%) afterwards. In addition, among those completing the HADS on both occasions (n=24), anxiety and depression levels were reduced in 15 and these differences are statistically significant (anxiety: z=-2.3, p=.02; depression: z=-2.7, p=.007). Data on frequency of clinic visits for acute STDs were available for 19 patients who had pre- and post-treatment clinic visits recorded on the HIV/GUM database. Mean number of STD infections per year were significantly reduced from 0.90 (sd 0.8) before to 0.19 (sd 0.33) after intervention (z=-3.1, p=.002).
Insofar as the evaluation measures are adequate to assess effectiveness, the clinical intervention appears to reduce self reported risk behaviour, increase confidence in the ability to maintain safer sex and reduce the frequency of new STDs. Levels of anxiety and depression are also significantly reduced.

2.7. Cost effectiveness

The difficulties encountered in achieving referrals and the relatively low numbers who complete intervention have led us to question the value and cost effectiveness of the clinical service. A formal cost analysis has not been carried out but a number of points can be made. A generous estimate of the time spent by the clinical psychologist and the assistant psychologist on risk reduction activities amounts to 5 sessions a week each so that total costs are about £30,000 a year including overheads and costs of publicity materials. A recent article reviewing economic evaluation of HIV prevention initiatives states that "an HIV prevention program is cost saving if the cost per HIV infection averted is less than roughly $485,000- $515,000...... Clearly, other intangible factors (eg. pain and suffering) would raise this monetary valuation if they could be quantified. Also, survey methods used to assess a person's willingness-to-pay to avoid certain health hazards have generally yielded much higher valuations of human life" (Holtgrave et 1996, pages 471-472). The monetary value alone translates to over £300,000. Estimates of the medical costs of HIV infection in this country amount to £77,000 assuming 10 asymptomatic years, one symptomatic year and one year with AIDS (Hughes & Morris, 1996). These authors also estimate that 36-41 life years, with the economic productivity and saving of state benefits this implies, are gained for every infection avoided. If the clinical intervention has been successful in preventing only one person per year from being infected, it will have been cost saving in terms of medical costs alone and if only one person has avoided infection in the whole four year period, costs will have been saved when the total economic impact is considered.

The effect of attending for only one or two sessions is unknown but may have
provided some impetus for change. Even if change to consistent condom use in every sexual encounter is not achieved, a decrease in the proportion of unprotected encounters is a legitimate treatment goal. Using model-based estimates, Pinkerton & Abramson (1996) show that occasional condom use can significantly reduce the risk of HIV infection under most HIV prevalence and infectivity conditions. Because the prevalence of HIV infection in London is higher than in the rest of the UK, and particularly high among gay men, the likelihood that those attending the service are at high risk is increased and the cost effectiveness of facilitating even small positive changes in risk behaviour is enhanced.

2. 8. Summary: clinical service

The clinical intervention service has developed pragmatically. Since the funding was initially subject to review by the commissioning agency after a two year period, we were anxious to demonstrate that the service was being taken up. This tended to lead to a very loose criterion for what constitutes risk behaviour, little room to demonstrate positive change and many inappropriate referrals. We were surprised that, in spite of the requests from physicians for the service because of their awareness of continuing high risk behaviour, the referral rate proved to be so low but anecdotal evidence from another central London clinic indicates that uptake of a group intervention which covers other aspects of lifestyle in addition to safer sex is similarly low (Wanigaratne, personal communication).

Group interventions have not proved appropriate for the population referred for risk reduction partly because the numbers referred are low but also because of the nature of the presenting psychological and behavioural difficulties among which the issue of safer sex is often one of many problems. The individuals presenting to the service are clearly different from those who took part in the successful cognitive behavioural interventions described by Kelly et al (1989; 1990). The participants in the latter studies were volunteers and the groups took place at an earlier stage of the epidemic when there was a particularly strong movement among gay men faced with the
possibility of AIDS to mobilise resources to meet the threat. As mentioned in the
review of HIV sexual risk behaviour above, extraordinary changes in sexual
behaviour among gay men were documented at the time that Kelly et al's studies were
carried out. One could speculate that many people with the personal and emotional
resources to effect sexual behaviour change were able to do so when the information
about transmission became widely known so that those experiencing difficulties now
represent a much more vulnerable group.

Data collection needs to be better implemented. A number of patients are known by
the clinical psychologist to have modified or eliminated their risk behaviour but there
is no objective evidence, in the form of self report data to demonstrate this. There are
several reasons for this: many patients are offered therapy for other psychological
problems and when these improve, there is sometimes a tendency to stop attending,
often before outcome measures have been collected. Some patients are given
questionnaires to complete at home but fail to return with them and as mentioned
above, postal follow up has not been successful.

CONCLUSIONS AND FUTURE PLANS

1. Although this cannot be conclusively demonstrated, it seems likely that among
the 127 people seen at least once, and among the 71 who completed
intervention, some have been successful in avoiding HIV infection. It has not
been possible to increase referral rates and the current rate of one or two per
week may be the most that can be achieved in this setting.

2. Activities among HIV/GUM staff have identified inconsistent perceptions of
transmission risks and a need for training in communicating about safer sex
to clinic attenders. A recent survey of GUM clinic attenders in the West
Midlands revealed that 34% received no advice on either HIV or safer sex and
among those who attended for reasons particularly relevant to receiving such
advice, 40% claimed to have received it about neither HIV nor safer sex
(Hope & MacArthur, 1996). These findings reinforce the need to continue raising awareness of the importance of prevention and health promotion in the clinics and to follow through with the new arrangements instituted by the Health Promotion Strategy Group.

3. Good working relationships with the health advisers have been fostered in a number of ways but perhaps principally by collaborating with them on service related research projects (Flynn et al, 1994; Thornton et al, 1994; Gray et al, 1997; Holder et al, in press), in which their greater experience of large numbers of at-risk patients complements our better research skills. They have also collaborated with us in running a group for HIV positive men and the fact that they refer the greatest number of patients for risk reduction is further evidence in support of the successful management of a potentially difficult situation.

4. Current plans, some already partly implemented include:

i. a randomised trial of behavioural and cognitive treatment approaches to risk reduction among gay men.

ii. a qualitative study of seroconverters intended to identify ways in which HIV infection might have been prevented by intervention from the clinic.

iii. a study of 200 gay men attending for HIV tests with a history of 2 or more previous HIV tests in the same clinic to investigate reasons for repeat testing and risk behaviour.

iv. a comparison of psychological and demographic characteristics among those attending for same day result HIV tests and those attending for normal waiting time tests. This study is intended to provide
information to enable improved targeting of resources.

5. The recent advances in treatment for HIV disease indicate that seropositive individuals will live longer and healthier lives and this is likely to have an impact on sexual behaviour and HIV transmission making it even more urgent that ways are found to enable people to protect themselves and others.

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4. RESEARCH DOSSIER

ABSTRACT: This exploratory study examined the relationships of psychological variables and HIV disease progression in a sample of gay men (n=147) infected for at least 8 years and followed up clinically for 5 to 32 months. Psychological variables included personality, coping strategies, psychological morbidity, perceived social support and life events. Biological and medical variables included handedness, viral load, CD4 count and use of antiretroviral medication. Cox’s proportional hazard regressions to the two dependent variables of time to CD4 count below 200 x10^3/L and time to ARC or AIDS diagnosis showed that biomedical variables were predominant in predicting disease progression but acceptance coping was a significant predictor of longer ARC or AIDS-free survival.
PSYCHOLOGICAL VARIABLES AND HIV INFECTION: A STUDY OF THEIR EFFECT ON DISEASE PROGRESSION AMONG LONG TERM INFECTED INDIVIDUALS

INTRODUCTION

Fifteen years have elapsed since the first reports of AIDS in 1981 and today it is estimated that 28 million people worldwide are infected with the human immunodeficiency virus (HIV) which causes AIDS. In the UK, most of those infected with HIV are gay men and infections are concentrated in the London area. Although the majority of HIV infected individuals eventually develop AIDS, a subgroup remain clinically well more than 10 years after infection. The factors responsible for diminished susceptibility to HIV infection are not well understood. Biological factors thought to be important include initial immune response at seroconversion, viral strain and load, host genetic factors as well as specific immune system activity. Lifestyle and demographic factors (eg. smoking, substance use, sexual behaviour, age, socioeconomic status) have also been suggested as possible determinants of disease progression (Easterbrook, 1994).

In this study, the relationship of psychological variables and disease progression will be examined in a sample of gay men who have been infected for at least 8 years. There are good reasons for investigating the possible impact of psychological factors on the outcome of HIV infection. In addition to the evidence that they may influence disease course in other conditions, an important consideration is that many HIV positive people regard factors such as mood, attitude to the illness, ability to manage stress and coping strategies as critical in mediating the effects of the virus. Troop et al (1997) found that among a group of 134 long term infected individuals the most frequently given attribution for maintenance of good health was mental attitude, in particular a positive outlook, and only 3% thought medical intervention had played any role in the outcome of infection. If these patients are correct in their beliefs, the hope is offered that psychological factors with a positive effect on disease progression

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can be fostered and negative factors corrected. On the other hand, misinterpreted psychoimmunological research could constitute a significant health risk in that patients might overlook the importance of medical treatments. In addition, since it is widely believed that poorer health outcomes are associated with negative emotions and high levels of stress, patients may feel responsible for having a mental health disorder or anxious about difficulty in managing unavoidable stresses.

The basic science of HIV infection and the functions and mechanisms of the immune system are both areas of immense complexity. In order to provide definitions of terms used in what follows, a brief and simplified description of immune system measures and of HIV infection is given below:

**Immune system measures.** The function of the immune system is to identify and eliminate foreign matter (antigen) which enters or contacts the body. The system is divided into a humoral system which responds with antibodies (immunoglobulins) produced by B cells and a cell-mediated system which responds by direct killing (cytotoxicity) as well as with production of immune mediators such as interferon. Lymphocytes, designed to attack specific targets, make up 20% of the circulating leucocytes and are predominantly of two types: B cells and T cells. In addition to cytotoxic T cells, there are helper T4 cells which enhance the immune response and suppressor T cells which reduce it. Both the suppressor and cytotoxic T cells have CD8 receptors while the T helper cells have CD4 receptors. Another type of lymphocyte is the natural killer or NK cell which destroys virus-infected and tumour cells. Monocytes/macrophages are leucocytes with CD4 receptors which can recognise and destroy certain bacteria and immunoglobulins and also have a role in assisting the function of lymphocytes by presenting antigen to them. Measures in immune system research are of two types. Enumeration measures quantify the types of cells present in absolute and percentage form to give an assessment of the efficiency of the immune system. Functional measures assess the ability of the system to respond by stimulating it with antigen, usually in the form of mitogens, and measuring lymphocyte proliferation, antibody levels or extent of target cell destruction (O’Leary, 1990).
HIV and the immune system. HIV is a retrovirus which means that its genetic material is encoded in the form of RNA rather than DNA. The virus attaches to cells bearing CD4 receptors where a DNA copy of the viral RNA is made by the reverse transcriptase enzyme and incorporated into the host genome. The primary targets of HIV are the T4 cells which are destroyed once replication has begun thus producing an immunosuppressive effect. The virus also enters monocytes/ macrophages through cell surface CD4 receptors and since these cells are not always destroyed, they can become reservoirs of the virus. The ratio of CD4 to CD8 cells which is 2:1 in healthy individuals is reversed as HIV infection advances and the decline in CD4 or T4 cell count from normal levels of 800-1000x10^6L is the most commonly used marker of disease progression. Viral load which is high at seroconversion and in later disease is now thought to be an important prognostic marker however the high cost of these assays means that they cannot be routinely used in clinical practice. Humoral and NK cell abnormalities are also seen as HIV infection progresses making it a complex and heterogeneous immune system disease (Antoni et al, 1990).

Immediately following infection with HIV, most patients are either perfectly well or may have enlarged lymph glands (lymphadenopathy). After seroconversion, the median time before any AIDS defining illnesses occur is eight to ten years in developed countries (Munoz et al, 1993). Once an initial AIDS diagnosis is made, average survival depends varies between 6 and 28 months with a median of 20 months in the UK (Moorcroft et al, 1997). Significant symptomatic disease (AIDS related complex (ARC) or AIDS) is rare when an individual has a CD4 cell count above 200x10^6L. Disease progression is thought to be more likely when there is co-infection with another virus because of the consequent activation of T cells and possibly of the HIV replication mechanism.

The Centers for Disease Control (CDC) in the USA provides a classification system for HIV infection which is internationally recognised and is shown in the Appendix. In summary, group I refers to acute infection, group II to asymptomatic infection, group III to persistent general lymphadenopathy and group IV to symptomatic
infection including ARC, AIDS, and possible HIV-associated disease (CDC, 1987).

1. **PSYCHOLOGICAL FACTORS AND THE IMMUNE SYSTEM**

It is now well established that the central nervous system can exert a regulatory influence on the immune system and conversely, that the immune system can influence nervous system functions (Dunn, 1990). It is relevant to note that HIV is a neurotropic virus and the nervous and immune systems are the only organs directly affected by HIV. In animals, the conditioned suppression or enhancement of immune system responses have been documented in different species under a variety of conditions and a clinically significant effect of conditioned stimuli on immune mediated conditions has also been demonstrated. Similarly, stressful stimulation can influence susceptibility to disease and immune system response (Ader & Cohen, 1993).

In humans, research can be divided into studies assessing the impact of psychological variables or interventions on measures of immune function in either physically well or unwell populations, on the triggering of latent infection such as herpes simplex or Epstein-Barr virus and on disease onset, progression or severity.

Studies on immune function in well people are reviewed in section 1.1., those on latent infection in section 1.2., followed by studies of the effects of psychological interventions in section 1.3. Section 1.4. describes studies of psychological variables and disease course. Although the investigations of disease vary with respect to design, size of sample, immune system or disease progression measures and range and type of psychological variables assessed and any categorisation of the research is somewhat arbitrary, the studies have been organised as far as possible by findings related to specific psychological factors as follows: 1.4.1. Social support; 1.4.2. Life events; 1.4.3. Mood and coping style and 1.4.4. Personality. Section 1.5. describes findings related to handedness and disease.
In HIV infection, 29 relevant studies of gay or bisexual men have been identified and all but four (Solomon et al, 1988; Nokes & Kendrew, 1990; Temoshok et al, 1990; Reed et al, 1994) have excluded subjects with an AIDS diagnosis. Four of the studies which report on the effects of psychological interventions are included in section 3. The remaining studies are reviewed in each part of section 4 following an overview of findings in other illnesses.

1.1. Relationship of psychological variables and immune system measures:

Among studies in this group, perhaps the most well known are those of Kiecolt-Glaser and Glaser who have demonstrated depressed immune function and increases in illness reports among medical students related to exam stress which are not accounted for by associated changes in sleep or diet (Kiecolt-Glaser et al, 1986). Chronic stress has also been shown to affect immune system parameters in a negative direction (McKinnon et al, 1989; Kiecolt-Glaser et al, 1991), contrary to the suggestion from animal research that adaptation or even enhancement of immune response over time may occur (Ader & Cohen, 1993). Studies assessing immune function following loss, separation and accompanying loneliness also show associations with impaired immunity (Bartrop et al, 1977; Kiecolt-Glaser et al, 1987; Glaser et al, 1985) and social support is associated with better functional immune system measures (Uchino et al, 1996).

Depression and immunity have been the subject of a great many investigations following the observation that depression is associated with an increased risk of physical morbidity and mortality (Elliot & Eisdorfer, 1982) and that changes in affect produce immune system responses (Ader et al, 1991). A meta-analytic review of 35 studies selected to include only those using clear diagnostic criteria for depression, a nondepressed control group and immune outcome shared with at least one other study was carried out by Herbert & Cohen (1993). In addition, separate analyses were carried out for studies which met strict methodological criteria and those which did not. The authors conclude that clinical depression is reliably associated with both
enumerative and functional immune system measures and that in general, effect sizes were larger in the methodologically sounder studies. They point out however that it is not possible to say whether these associations have implications for physical health, although they speculate that depression-related immunosuppression may increase susceptibility to immune-mediated disease.

Finally, there are a few studies examining the relationship of personality traits and coping styles and immune system measures. Power motivation and locus of control in interaction with stress (McLelland et al, 1982; Jemmott et al, 1990) have been associated with lower levels of salivary immunoglobulin A. Repressive coping style was related to reduced monocyte counts and reduced response to mitogens in two studies (Jamner et al, 1988; Brown et al, 1989) and neuroticism, as measured by the Eysenck Personality Questionnaire to leucocyte count (Daruna, 1996).

1.2. Relationship of psychological variables and latent infection:

There has been a good deal of interest in the possibility that psychosocial factors influence susceptibility to infections in healthy people. Of more relevance to the present study, which concerns individuals who are already HIV positive, are investigations looking at activation of latent infection especially that of the herpes viruses which include cytomegalovirus (CMV) Epstein-Barr virus (EBV) and the two types of herpes simplex virus associated with cold sores and genital lesions. HIV itself was until recently considered to enter a latent phase after seroconversion but there is now evidence that viral replication continues even though the patient may remain asymptomatic for many years (Weissman, 1993). Reactivation of herpes viruses however may be particularly important in HIV infection since this would produce a proliferation of T-helper cells and therefore of HIV, and since several of the herpes viruses including CMV and EBV are known to have immunosuppressive effects (Rinaldo, 1990). CMV is one of the most common opportunistic pathogens in those with AIDS and also among the most immunosuppressive of the herpes group. There is also evidence that EBV reactivation may act as a co-factor for progression
to AIDS (Rosenberg & Fauci, 1991). While antibodies to the herpes viruses have been shown to be raised in the presence of stress (Glaser et al, 1985), the evidence from clinical samples of the role of psychological factors in recurrence is suggestive rather than conclusive because of methodological shortcomings in the studies (eg Goldmeier & Johnson, 1982; McLarnon & Kaloupek, 1988). The strongest findings are those from prospective studies relating level of psychiatric morbidity (Goldmeier et al, 1986) and continuing depressed mood (Kemeny et al, 1989) with recurrence.

1.3. Psychological interventions and immune function or disease:

Early studies used hypnotic suggestion to induce a hypersensitivity reaction in one arm following injection of the same antigen to both arms (Black, 1963; Black & Friedman, 1965). Relaxation training was also shown to produce significant increases in NK cell activity among older adults in a controlled study (Kiecolt-Glaser & Glaser, 1985).

Two reports of group psychological interventions with cancer patients demonstrated greater longevity (Spiegel et al, 1989) and enhanced immune system activity (Fawzy et al, 1990) among group participants compared to controls. Evidence in HIV infection has been mixed. Two studies have found no effect of an 8 week stress management intervention among HIV positive gay men on measures of immune function (Coates et al, 1989; Auerbach et al, 1992). In two related studies, seropositive gay men randomised to an aerobic exercise or stress management programme during a 5 week interval between testing and notification of HIV serostatus showed attenuated declines in NK and CD4 cell counts compared to controls (Antoni et al, 1991a; LaPerriere et al, 1991) but the clinical significance of these differences is unknown. The available evidence that psychological intervention can modulate the effects of disease is therefore not strong.
1. 4. Specific psychological variables and disease:

1. 4. 1. Social support: The association of social support and both morbidity and mortality has been known for many years (eg. Cobb, 1976) consistent with the evidence that the formation and disruption of social relationships have important physiological consequences in animals (Coe, 1993). Many of the studies investigating social support and illness have concerned cardiovascular disease (Eriksen, 1994) although there are a few examining the effects of social support in cancer outcomes, the results of which are mixed (Hislop et al, 1987; Cassileth et al, 1987; Levy & Heiden, 1990). A recent review of 81 studies specifically focusing on physiological dependent variables and using both qualitative and meta analytic procedures is available to clarify the findings (Uchino et al, 1996). The authors include studies of at risk populations as well as of normal subjects, experimental manipulations and clinical interventions. They conclude that the effects of social support appear most reliable on blood pressure, catecholamines and aspects of immune response and they note the heterogeneity of social support measures used, some assessing structural aspects and others functional.

Social support and HIV progression: In a small study of 31 HIV positive men which included those with AIDS, Nokes & Kendrew (1990) found that loneliness was not associated with the number of AIDS related infections in a six month follow up period. Concealing one's sexual orientation could be construed as an indirect measure of lack of social support and this was related to more rapid CD4 decline, and shorter time to mortality in a 9 year follow up of a subgroup of 80 men in the Multicenter AIDS Cohort Study (MACS) although another measure of social support was not (Cole et al,1995). Other studies using conventional measures of social support have also produced negative findings (Goodkin et al, 1992a; Perry et al, 1992; Mulder et al, 1995) but Persson et al (1994), using a composite measure of social network and support, found an association between low support and lower CD4 counts in a cross sectional study of a small sample of 47 men.
1. 4. 2. Life events: Most of the studies in this area have been of cancer patients. Two studies have reported an association between stressful life events and disease progression although both have been criticised for methodological reasons (Funch & Marshall, 1983; Ramirez et al, 1989). A more recent report (Barraclough et al, 1992) failed to find any evidence that any psychosocial measures including life events contribute to relapse in a study of 204 women with breast cancer assessed four, 24 and 42 months after surgery.

Life events and HIV progression: Two studies of HIV positive men have specifically looked at the relationship of life events and disease markers. In Kessler et al’s (1991) study of a large sample of men in the MACS followed for up to 30 months, no relationship of AIDS-related or more general life stresses were found with declines in CD4 counts or HIV related illness. In contrast, a more recent report in which potentially confounding variables including depression and disease state were carefully controlled shows a significant relationship between severe stress and reductions in NK lymphocytes (Evans et al, 1995). Although the activity of these cells is thought to be important in host defences to HIV infection, this cross sectional study cannot assess the significance of the immune response changes on clinical progression. In line with the findings of Kessler et al (1991), a number of other studies, most assessing a range of psychological variables have not reported any relationships of life events and immunological measures or clinical progression (Rabkin et al, 1990; Goodkin et al, 1992a; Perry et al, 1992; Mulder et al, 1995) although the Goodkin et al study reported trends for a negative effect of life stressors and a buffering effect of social support on life stresses on their outcome measure of NK activity. In another small cross sectional study of 11 seropositive men, Goodkin et al (1992b) found a significant association between major life stressor impact and total lymphocyte count.

1. 4. 3. Coping style and mood: Much of the work in this area was stimulated by Greer et al’s 1979 study which showed that women who responded to breast cancer with denial or fighting spirit 3 months post operatively had a significantly improved outcome compared to those responding with stoic acceptance, anxious preoccupation
or helplessness/hopelessness. These predictions still held good at 15 year follow up (Greer, 1991). The study has been criticised on the grounds that psychological response was assessed by clinical interview and that certain disease markers were not adequately controlled. Among other studies, partial support for Greer et al’s findings was provided by DiClemente & Temoshok (1985), Jensen (1987) and Wirsching et al (1988). By contrast, Cassileth et al (1985) and Jamison et al (1987) found no relationship of psychosocial variables including hopelessness on survival. In a study of the effect of stress and coping style on severity of cervical cancer, Goodkin et al (1986) found that hopelessness intensified the predictive effect of life events on disease severity.

A significantly longer survival among women with metastatic breast cancer was found among those with low anger (Hislop et al, 1987) and among those reporting joy, optimism and enthusiasm (Levy et al, 1988). Derogatis et al (1979) however, found that long term survivors of metastatic breast cancer were more uncooperative and complaining and had higher levels of anxiety and dysphoric mood than short term survivors. On the other hand, Barraclough et al (1992) failed to find any relationship of major depression and relapse among breast cancer patients followed up for over 3 years. A few studies have assessed immune function as well as psychological factors in breast cancer: immunoglobulins were increased in association with denial (Pettingale et al, 1981) and Levy et al (1985) found higher levels of NK activity among those with more distress and maladjustment.

A large study of 121 women followed up for 6 to 8 years after mastectomy (Dean & Surtees, 1989) found that a denial coping strategy 3 months post-operatively predicted disease-free survival, as in the Greer et al study. Coping styles were assessed using Greer et al’s interview method, but no differences were found in recurrence-free survival among those adopting the coping styles of fighting spirit, stoic acceptance or helplessness. Psychiatric caseness measured by the GHQ pre-operatively was also associated with a better prognosis and the authors consider that their results are in line with those of Derogatis et al (1979) and Levy et al (1985) described above, in that
expression of negative affect was associated with improved outcome. It is interesting that in Dean and Surtees' study, the outcomes in relation to the psychological variables were different depending on when the latter were measured. Whereas high GHQ scorers preoperatively had a better chance of disease free survival, those with high scores postoperatively did less well. Similarly, while helpless/hopeless and stoic acceptance coping assessed preoperatively contributed to the prediction of recurrence free survival, when coping was assessed postoperatively, only denial coping was a significant contributor to improved outcome. It would appear on the basis of findings in this study that prognosis predicted on the basis of psychological variables which may not be stable over time depends critically on the timing of these measures.

**Coping styles and mood and HIV progression:** In HIV infection, the evidence concerning mood and coping styles is mixed. Kertzner et al (1991) assessed anxiety and depression in 109 HIV positive and 75 HIV negative men at 4 time points over 2 years. No associations were found between the mood measures and CD4 counts in either group. In another longitudinal study, 277 gay and bisexual men with HIV infection completed a self-report measure of depression and were followed up at 6 month intervals for up to 66 months (Burack et al, 1993). Depression was associated with a more rapid decline in CD4 count although not with subsequent AIDS diagnosis or mortality. Reporting on a much larger sample of 1809 HIV infected participants in the MACS followed for up to 8 years, using the same depression measure and including CD4 slope in the dependent variables, Lyketsos et al (1993) found no evidence that depression predicted a worse outcome. The Burack group later reported that in their study, only those with high initial CD4 counts showed the decline and pointed out that the Lyketsos sample had lower initial CD4 counts (Barrett et al, 1994). The implication is that negative affect in the early stages of infection may have an accelerating effect on progression. In response, Lyketsos et al (1994) showed that even in a group with high CD4 counts, there was no relationship of CD4 decline and depression.

In an earlier study, Rabkin et al (1990) found no effect of anxiety, depression,
hopelessness, grief or demoralisation on CD4 and CD8 counts at six month follow up. Similarly, Perry et al (1992) found no effects of any psychosocial variables including depression, anxiety, psychiatric diagnosis, hopelessness and bereavement on CD4 cell count over a one year period. In a cross-sectional study, HIV negative, HIV asymptomatic and HIV symptomatic men were compared on a variety of psychological measures (Sahs et al, 1994). No differences between the groups and were found on the measures which included anxiety, depression, and hopelessness and they were unrelated to NK cell counts. Kemeny et al (1994) looked at psychological distress measured by the Profile of Mood States and both enumerative and functional measures of immune function in 45 bereaved HIV positive men compared with a matched group of non bereaved men. There were no differences between the groups on the immunological measures but in the non bereaved group only, depression was associated with decreased immune function, a finding which is difficult to evaluate.

A number of studies from Antoni and colleagues have examined coping strategies following HIV positive status notification in relation to immune system measures and progression. Their earlier reports indicated that use of denial and behavioural disengagement to cope with the diagnosis was predictive of greater immunological impairment at one year follow up (Antoni et al, 1991b) and greater likelihood of disease progression after 2 years (Ironson et al, 1994). A more recent study (Antoni et al, 1995) controlling for social support, life events and loneliness confirmed the relationship between disengagement (denial, behavioural and mental disengagement) coping and lower immune system indices after diagnosis and at one year follow up, although the reverse was not found in that there was an absence of effect of more active coping strategies on immune function at one year. In two cross sectional studies, Goodkin et al (1992a) found that active coping was positively related to NK cell activity and passive coping (1992b), in association with life events, with lower lymphocyte count among asymptomatic seropositive gay men. In a prospective design controlling for behavioural and biomedical variables, logistic regression showed that decreased clinical progression at one year was predicted by active confrontational coping (Mulder et al, 1995). In one of the few studies of disease progression in
AIDS, Reed et al (1994) assessed coping as well as a range of psychological, behavioural and disease variables. The coping style of realistic acceptance, which the authors feel has some overlap with Greer's stoic acceptance, was a significant predictor of decreased survival time not accounted for by other variables.

1. 4. 4. Personality: There exists a large body of research concerning personality and behaviour patterns and their relationship with heart disease and a great deal of controversy has been generated over the problems associated with self report of symptoms (Stone & Costa, 1990), the measures used and whether the factor in question is a behaviour pattern or a personality trait (Booth-Kewley & Friedman, 1987), whether the construct is unitary (Matthews, 1988) and whether it reflects psychopathology or other personality variables (Stone & Costa, 1990). This literature is too extensive to review here but the consensus view now seems to be that hostility is the critical factor in heart disease (Barefoot, 1992). A recent comprehensive meta analytic review (Miller et al, 1996) concludes that evidence from both cross sectional and prospective studies shows that hostility is an independent risk factor for coronary heart disease. Personality variables have been less studied in relation to other illnesses and, as discussed below, the distinction between coping styles and personality may not always be clear cut. There are probably many reasons for the relative neglect of personality including the question of its stability over time and situations, and the rejection of earlier psychodynamic conceptualisations of specific links between certain diseases and personality types.

An early study involving exposure of individuals to a pathogen pointed to the possible importance of personality in disease severity: using Eysenck's measure, premorbid introversion predicted greater virus shedding as well as symptoms following experimental inoculation with a rhinovirus (Totman et al, 1980). Although there has been some interest in the type C cancer-prone personality (Temoshok, 1985), many of the research findings are contradictory and difficult to interpret (Levy & Heiden, 1990) which is not to say that they lack validity since links are likely to be complex. Some of these concepts have been elaborated by Eysenck & Grossarth-Maticek (eg.
Eysenck & Grossarth-Maticek, 1991) in a series of studies showing relationships of personality types and cancer and coronary heart disease and their amenability to psychological treatment. Unfortunately, their claims have been met with almost universal rejection (eg. Amelang et al, 1996) because of concerns about the psychometric properties of the personality measures used and about the failure of the authors to counter adequately criticisms about details of research design. A few reports have related extraversion and breast cancer outcomes: Hislop et al (1987) found greater survival among women high on extraversion but this was not confirmed by Dean & Surtees (1989). Finally, in a controlled study, neuroticism was shown to be related to recurrent urinary tract infection (Hunt & Waller, 1992).

**Personality and HIV progression:** Personality variables have received little attention in studies of disease progression in HIV infection. As well as the reasons mentioned above, there has perhaps been an additional reluctance to further stigmatise or demoralise HIV positive individuals. Solomon et al (1988) have discussed the concepts of fighting spirit and hardiness, which they consider to be antithetical to the Type C coping style associated by Temoshok et al (1985) with poor outcomes in cancer, in relation to a study of long term survivors with AIDS. Hardiness was proposed by Kobasa (1979) as a personality style made up of the characteristics of commitment, control and challenge, which acts as a source of positive resistance to the debilitating effects of stress on health as well as functioning to enrich an individual’s life. Four studies report on hardiness and disease progression in HIV. In a pilot study, Solomon et al (1988) compared hardiness scores obtained 2-8 weeks after AIDS diagnosis for men who had died by follow up (n=10) with those who were still alive (n=11) and found that the latter had scored significantly higher on the control dimension. The authors point out however that, since the men who had died had a longer follow up and the hardiness measure was correlated with timing of the initial interview, the relationship might be due to the fact that those with a longer time in the study scored lower on control. This might also imply that scores on hardiness depend on when, in relation to a stressful health related event, they are measured. This also seemed to be the case with psychological measures assessed at
two different times in a study of outcomes in breast cancer (Dean & Surtees, 1979) discussed in section 1. 4. 3. above. A second study from the same group of an even smaller sample of 15 men with AIDS but this time controlling for time since diagnosis and CD4 count, found some correlations of hardiness with length of survival (Temoshok et al, 1990). These data were presumably preliminary since they were reported at a conference and have not subsequently been published.

Perry et al (1992), in their study of 221 subjects followed up for 12 months which included a range of psychosocial variables found no relationship of hardiness and CD4 counts either concurrently or prospectively. One longitudinal study (Solano et al, 1993) assessed both hardiness and the coping styles of fighting spirit, denial and hopelessness as well as a number of other variables among 100 seropositive subjects followed up at 6 and 12 months. Denial was associated with clinical progression and fighting spirit with no change in disease status at 12 months. Lower scores on one subscale of hardiness were also associated with progression but only in interaction with initially lower CD4 counts. The concept of hardiness, while having considerable face validity, has been criticised mainly on the grounds that it is not unitary and that one or more of its components reflect factors better measured in other ways such as locus of control or neuroticism (Hull et al, 1987; Funk, 1992). The measurement of denial, fighting spirit and hopelessness in the Solano et al study is also problematic. Subjects read three stories each designed to depict one of the coping styles in response to a diagnosis of a life threatening illness and rated on 10 point scales how similar their reactions would be and how much they would like them to be as depicted (Temoshok, unpublished). Hence the strongest findings in this study are based on a measure of unproven psychometric properties and overall, in contrast to some of the other findings of relationships between psychological variables and progression in HIV infection, the evidence concerning personality styles is not compelling.

The issue of whether a psychological variable influences onset or progression of an illness or both bears upon the distinction between state and trait variables. If 'hostility' predicts disease susceptibility as well as disease recurrence, and is
considered to be a trait, can it be modified? If a person is high in 'hostility', 'denial' or depression after a heart attack or after being diagnosed with HIV, is this a transient response, a coping strategy or an enduring personality trait? There is some evidence that coping strategies are related to personality characteristics (McCrae & Costa, 1986) which themselves might reflect a biological basis which also predisposes to disease. Lazarus (1990) tends to the view that most people use all forms of coping depending on the context but acknowledges that some forms of coping (emotion-focused) are more stable than others (problem-focused). Measures of social support, whether actual or perceived, are also likely to be associated with coping styles as well as with personality and psychological disorder (Monroe & Steiner, 1986) and certain personality traits will tend to make stressful life events more likely (Suls & Rittenhouse, 1990). The relationships of personality with mood and psychological distress are well established (Watson & Clark, 1984).

The models which are most frequently used to link personality and disease are those which associate certain personality traits with more risky health behaviours, stressful life events and social conflict and those which suggest that they cause individuals to react to stressors with exaggerated physiological reactivity (Suls & Rittenhouse, 1990). In practice, these interrelationships are extremely difficult to disentangle and it seems important that studies in this area should at least use a robust personality measure rather than rely on assessment of a limited range of traits.

1.5. Handedness and illness:

In 1982, a biological model was proposed linking handedness, dyslexia and autoimmune disorder (Geschwind & Behan, 1982) in part stimulated by the evidence of an association between male gender, left handedness and developmental disorders. The theory which is an exciting attempt to link brain maturation, cerebral dominance and abnormalities in autoimmune function is made up of two hypotheses both concerning the effects of testosterone on development in utero. The first is that excess foetal testosterone retards development of the left hemisphere which is proposed in
any case to develop more slowly than the right leading to developmental disorders such as dyslexia and stuttering. Secondly, testosterone is hypothesised to delay maturation of the thymus gland leading to impairment of the immune system and predisposition to autoimmune disease. In a later extension of the theory, Geschwind and Galaburda (1985) suggested a link between left handedness and homosexuality in males and speculated that this might confer a greater susceptibility to infection with HIV or to developing AIDS.

The neuroanatomical and hormonal bases of the theory remain controversial. Summarising the evidence, Bishop (1990) notes that maturational differences between the hemispheres are complex, that sex differences in relative rate of maturation of the hemispheres which would be predicted on the basis of testosterone levels in utero have not been found and that there is little consensus over the postulate that foetal testosterone influences maturation of the thymus. There has been some support for associations between handedness and dyslexia and between dyslexia and immune disorder but no study, apart from Geschwind and Behan’s original report, has demonstrated a three way association.

The evidence concerning left handedness and autoimmune disorders is mixed. Geschwind and Behan (1984) found a statistically significant increase in left handedness among patients attending hospital clinics for autoimmune diseases for five of eight conditions including Crohn’s disease and ulcerative colitis. An elevated frequency of left handedness was found among people with inflammatory bowel diseases (Searleman and Fuglazi, 1987) and among patients attending an allergy clinic (Smith, 1987). An increased frequency of mixed dominance was also observed in patients with autoimmune thyroid disease (Wood & Cooper, 1992). With these and a few other exceptions however, most studies have not found associations of handedness and autoimmune disease (Harris, 1993).

Only limited empirical data have been collected on the question of handedness among gay men. Lindesay (1987) demonstrated a significant shift towards non right
handedness in a sample of 94 gay men compared to a control group of 100 heterosexual men. In a postal survey of gay and heterosexual men, although there was a clear excess of left handers among gay men testing for HIV, handedness was not related to likelihood of infection (Marchant-Haycox et al., 1991). A very large study of 1612 gay men enrolled in the MACS examined the relationships of handedness and the following variables: HIV serostatus and CD4 cell counts at study entry; seroconversion rates and progression to AIDS during the follow up period; history of autoimmune and atopic disease (Becker et al., 1992). There was a small but significant elevation in left handedness compared to normative data, but no differences between seropositive and seronegative men nor between those who subsequently seroconverted and those who remained seronegative. In the HIV positive group, there were no differences in CD4 counts at study entry nor in proportions developing AIDS, as a function of handedness. Nor were there any differences in self report of autoimmune disorders although one of three allergic conditions (asthma) was significantly more common among non right handers. If, as Geschwind and Galaburda suggest, there is a link between handedness and susceptibility to the effects of HIV, the direction of the effect could be difficult to predict. It could be assumed that the hypothesised impairment of the immune system among left handers would accelerate the progress of the infection however, if left handers are more susceptible to autoimmune disorder, their hyperactive immune system may be beneficial in responding to HIV. On the other hand, a more efficient early immune response producing an increase in monocyte/macrophage cells could mediate rapid proliferation of the virus which enters these cells through their CD4 receptors.

2. STUDIES OF NON PROGRESSION IN HIV INFECTION

Observations that some HIV infected individuals remain well after more than a decade of follow up have prompted a shift in the focus of natural history studies towards the identification and characterisation of subgroups of non progressors. As would be anticipated, the main focus of these studies is on immunological and virological factors but most have also collected some self reported behavioural data. Four studies
of HIV infected gay men have reported in the last two years. In Buchbinder et al’s (1994) study of 539 men with documented seroconversion dates, 42 (8%) had been infected for at least 10 years, had no AIDS diagnoses and their CD4 counts remained equal to or above 500×10^6/L. This group was compared with individuals with a similar length of infection but with signs of disease progression. The progressors had a more rapid rate of CD4 decline but there were no differences between the groups in prior exposure to recreational drugs or sexually transmitted diseases. Similarly, Keet et al (1994) compared 61 non progressors who had been infected for at least 7 years with 142 men who had progressed to symptomatic infection within 7 years and found no associations of progression with recreational drug use or markers of high risk sex nor were there any associations with self reported coping behaviours except that, counter-intuitively, the non progressors had lower active problem solving scores. A third study (Veugelers et al, 1994) described findings from cohorts in Australia, Canada, The Netherlands and the United States which together produced a sample of 403, all with documented seroconversion dates and infected for up to 9 years. Younger age at seroconversion was related to slower progression but no relationships were demonstrated between high risk sexual behaviour, history of sexually transmitted disease or alcohol, tobacco or recreational drug use and disease progression. Finally, Munoz et al (1995) used data from the MACS, which includes men who were seropositive at entry to the study as well as some who subsequently became HIV positive, to characterise long term non progressors. They found that about 15% of the men who were positive at entry and met their criteria for number of recorded CD4 counts, had positive regression slopes indicating a stable or increasing CD4 count during follow up of up to 9.5 years. A subgroup of these men were compared with groups with moderate or fast CD4 decline matched for age, race and initial CD4 count. Differences between the three groups with respect to onset of AIDS and survival were in the expected direction, but analyses of demographic, sexual behaviour and sexually transmitted disease data revealed no significant differences among the three groups. These studies indicate that risky sexual behaviour, alcohol, tobacco and recreational drug use which may be assumed, at least in part, to reflect other psychological variables, are not relevant to disease progression in HIV
infection.

There are two further points to make about studies of non progressors. Firstly, only a few studies have the advantages of the availability of seroconversion dates, prospective designs, large samples, long periods of follow up and detailed psychosocial assessments. Such designs are prohibitively expensive and the more detailed and comprehensive the data collection, the more likely subject attrition becomes and the less generalisable the results. Secondly, among the existing studies, most define non progression and compare individuals who meet the criteria with one or more control groups. A recent article (Strathdee et al, 1996) demonstrates a marked lack of consistency between different definitions in proportions of non progressors identified. Five different definitions were used with a large sample and the results show that no two definitions classified all of the same subjects as non progressors. The authors observe that criteria based on CD4 slopes are more consistent than other definitions and recommend that a minimum of three CD4 counts, including a recent count should be used.

In this study, long term HIV infected gay men with documented clinical histories are assessed on self report measures of psychiatric morbidity, coping styles, life events and social support, variables suggested by the literature as of potential importance in disease progression. In addition, a well established measure of personality and an assessment of handedness have been included, measures not previously used in this context.

The review of the literature indicates that among physically well people, clinical depression, lack of social support, stress, neuroticism and repressive coping style may be associated with depressed immune system measures and physical morbidity. Psychiatric caseness and depressed mood may be relevant to activation of latent viruses and personality factors, coping styles, negative mood and expression of feelings may be important in disease onset and prognosis in some conditions. The evidence concerning life events and social support in illness progression is mixed. In
HIV infection, the strongest evidence is in relation to coping styles: several studies implicate disengagement or denial in poorer immune functioning and active coping with improved immune measures. One study suggests that realistic acceptance coping is associated with greater mortality risk. Although mood has been investigated in a number of studies, no significant relationships with clinical progression have been reported. Only one study reports a significant effect of life events on immune system measures and the balance of evidence is against any effect of social support on disease progression. Finally, one study reports that higher socioeconomic status is associated with slower progression of HIV infection independently of access to health care (Schecter et al, 1994).

Hypotheses for this exploratory study are therefore provisional: disengagement, denial and accepting coping styles, depressed mood, psychiatric morbidity, neuroticism, introversion, and lower socioeconomic status may be related to more rapid disease progression. The roles of handedness, life events and social support are uncertain. In addition, it was hypothesised that relationships between personality, psychological morbidity and coping style would be found, in particular, positive associations of neuroticism and psychological morbidity scores and of neuroticism and coping styles with an avoidant component (McCrae & Costa, 1986); and positive associations between extraversion and socially oriented coping (Gallagher, 1996).
METHODS

Sample

In August 1993, a study was initiated of the biological, behavioural and psychological determinants of non progression in long term HIV infected individuals, funded by the Medical Research Council. The study population was drawn from patients of the Kobler Clinic, Chelsea and Westminster Hospital, London and from two other linked clinics, the Victoria Sexual Health Clinic and the Genitourinary Medicine Department at Charing Cross Hospital. Subjects known to have been infected before January 1, 1988 (n=809), were identified from the database of whom 169 had a current CD4 count of >500x10⁶L and 640 a count of <500x10⁶L. Criteria for entry to the study were that subjects had a fully documented clinical history including at least four serial CD4 counts, had attended the clinic within the previous year, were in the homosexual risk group and were still living at the time of recruitment. Since the focus of the study was on the determinants of non progression, potential non progressors meeting these criteria, with a current CD4 count of >500x10⁶L and no AIDS or ARC diagnoses (CDC IVA, B, C1, C2 or D) were first identified (n=93): among these, 57 were recruited. From the group of 640 with faster progressing disease, three individuals for each non progressor matched for years of infection (+/- 1 year) and age (+/- 5 years) and meeting the study criteria were identified. The expectation was that approximately two thirds of these would be recruited and it was intended that the final sample would include a range of disease progression rates within a group homogeneous for age, years of infection and transmission route. There were 90 individuals with faster progressing disease in the final sample of 147 which included both seroprevalent individuals and seroconverters. All subjects gave written informed consent and the study was approved by the ethics committee of Chelsea and Westminster Hospital.
Procedure

Subjects completed standardised self report questionnaires at the time of enrollment in the study when blood samples were also taken for CD4 counts. Viral load assays were also carried out for 129 (90%) of the participants. Since the assessment procedure also included a structured interviewer administered questionnaire to obtain information on clinical and lifestyle history (not reported here), the psychological measures were deliberately kept relatively brief.

Measures

HIV Infection
Where subjects had a documented negative HIV test before testing positive (n=22), date of seroconversion was calculated as the midpoint between the two tests. For all other subjects, duration of infection was calculated from the date of the positive test. Clinical information, at infection or HIV positive test, and during the subsequent clinical course, including CD4 counts, was available from the medical records. The development of all HIV related symptoms, AIDS diagnoses and use of anti retroviral therapy at any stage of infection was documented. The study ended in February 1996.

Demographic data
Age, ethnic origin, education and occupation were recorded. Occupations were coded according to the IPA definitions (Market Research Society, 1990). Information was also gathered on the presence of past psychiatric history requiring in-patient treatment or medication.

Psychological measures

Personality: The Eysenck Personality Questionnaire-EPQ (Eysenck & Eysenck, 1975) has 90 items in the form of simple statements to be endorsed yes or no. It measures the personality dimensions of extraversion (E), neuroticism (N) and psychoticism (P)
as well as including a lie scale to identify those giving socially desirable responses. The EPQ has highly satisfactory reliability and the validity of its scales is the best supported of any personality measure (Kline, 1993). A recent study comparing the EPQ-R, which differs from the EPQ only in the development of the P-scale, and the five factor NEO-PI (Costa & McRae, 1985) found that the instruments had a good deal of variance in common most of which was accounted for by E and N and that a five factor model was not supported (Draycott & Kline, 1995). It was selected for these reasons and because of the hypothesised genetic and neurophysiological bases of the dimensions it measures.

**Psychological morbidity:** The General Health Questionnaire-28 (GHQ-28: Goldberg & Hillier, 1979) is a 28 item instrument assessing current (in the past few weeks) psychological symptoms on the four subscales of somatic symptoms, anxiety and insomnia, social dysfunction and severe depression. Each item is endorsed on a 4-point scale and Likert scoring (0-1-2-3) was used, rather than the 0,0,1,1 format which screens for psychiatric cases, in order to express a greater range of scores. The GHQ-28 has been shown to have good reliability and validity in a large number of studies (Kline, 1993).

**Coping strategies:** The Coping Orientations to Problems Experienced (COPE: Carver et al, 1989) is a 57 item measure tapping 13 theoretically derived, behaviourally specific coping strategies. Respondents indicate on a 4 point rating scale how often they cope as stated. The instruction was modified to ask respondents to indicate how they feel and behave in relation to HIV infection rather than to stressful events in general. The scale has been shown to have acceptable reliability and both discriminant and convergent validity (Carver et al, 1989). A principal components analysis was performed and, on the basis of a scree test, six factors were extracted, accounting for 50% of the variance, and rotated using oblimin. Standardised scores were calculated on the basis of these six factors.

**Social Support:** The Interpersonal Support Evaluation List (ISEL: Cohen et al, 1985)
assesses individuals’ perceptions of available support with 40 items each rated on a 4-point scale. It provides four subscales: appraisal, belonging, tangible and self esteem. The concept of functional or perceived support has become accepted as a more psychologically valid way of assessing social support on the grounds that while structural measures describe the extent of an individual’s network, they do not assess the function or the supportiveness of the relationships and the proposed buffering effect of social support on stress appears only if its perceived availability is assessed (Cohen & Wills, 1985). The ISEL has been shown to have good reliability and validity properties (Cohen et al, 1985; Sarason et al, 1987).

*Life events:* The Life Experiences Survey (LES: Sarason et al, 1978) assesses the number and impact of life stressors in the previous six months and consists of 47 items to be rated on a Likert scale from extremely negative to extremely positive. It allows for separate assessment of positive and negative experiences as well as individual ratings of their impact. The LES has adequate reliability and reasonable validity in terms of the significant relationship of negative events and stress related measures (Sarason et al, 1978). Minor adaptations were made for homosexual men and for HIV-related concerns resulting in a 48 item measure.

*Handedness:* The Edinburgh Inventory (Oldfield, 1971) assesses hand preference and strength of preference for 10 activities. It is one of the most commonly used in research studies (Bishop, 1990) and scores were transformed to produce an index from 100 to -100 indicating degree of handedness from completely right handed to completely left handed.

**Study Design and Data Analysis**

Subjects were enrolled over a period of 2 years from June 1993 to August, 1995 and analysis was carried out to include clinical information available up to February 1996. Hence the period between assessment and data analysis varied between 32 and 5 months and retrospective clinical information was available also for variable times.
depending on the dates of infection. In addition, the subjects while sharing the common characteristic of surviving up to 11 years of infection, differed in disease status at assessment and at follow up.

The important clinical outcomes in HIV infection are the point at which an individual's CD4 count drops to less than $200 \times 10^6 \text{L}$, when an ARC or AIDS diagnosis becomes more probable and an ARC or AIDS diagnosis itself, after which survival is limited. Where serial CD4 counts are available, the slope, calculated by least squares regression, is also often regarded as an efficient marker of disease progression (Dawson & Lagatos, 1991). A preliminary inspection of the data showed that, for the group as a whole, CD4 slope/6 months was correlated with time since infection suggesting that the relationship is not linear and that, in this sample, the slope changed over time. Although the analysis in this study is exploratory, there are a relatively large number of independent variables, and in order to minimise the chances of Type 1 error, analyses were restricted to the most clinically relevant dependent variables of time to CD4 count $< 200 \times 10^6 \text{L}$ and time to ARC or AIDS diagnosis. Since these events had not occurred for all the subjects by the end of the study, a regression model which takes account of censored cases is appropriate and the conventional method is survival analysis. One possibility is the Kaplan-Meier procedure however this method requires that the cases are divided into groups based on the values of the predictor variables. In this study there are both categorical and continuous independent variables and an analysis which can accommodate this is the Cox Proportional Hazards model.

Statistical analyses were carried out using SPSS. Two sets of main analyses were carried out using Cox regressions with the dependent variables of time to CD4 count $< 200 \times 10^6 \text{L}$ and time to AIDS or ARC diagnosis (CDC, 1987 definition), respectively. In each case, because of the numbers involved, independent variables were first entered separately and all variables found univariately significant at $p < .10$ were entered into the multivariate Cox regression analysis.
RESULTS

Sample Characteristics

At data analysis, it was found that 4 subjects had not completed any of the questionnaires and these subjects (2 AIDS, 1 CD4 > 500x10^6/L, 1 CD4 < 350x10^6/L) were excluded leaving a final sample of 143. All the subjects were gay men with an average age of 40.4 years (sd 7.8). The majority (132; 92%) described themselves as White, 4 (3%) as AfroCaribbean, and 7 (5%) were of mixed or other ethnic origin. At study entry, they had an average duration of infection of 8.6 years (sd 1.1) and a mean current CD4 count of 455.3 (sd 284.5). There was no difference between the 22 seroconverters and 121 seroprevalent subjects in duration of infection, current CD4 count or CD4 slope (Mann-Whitney z = - .59; -.91; -1.8, ns). Viral load was assessed for 129 (90%) of the subjects and because the distribution ranged from viral load below detectable level to 1,400,000 RNA/ml, subjects were grouped as follows: below detectable levels (n = 24), 19%; 1-100,000 RNA/ml (n = 79), 61%; > 100,000 RNA/ml (n = 26), 20%. Antiretroviral medication use currently or in the past was recorded for 99 (69%).

Clinical status at entry and at the end of the study (Table I) shows that a greater proportion of the sample at follow up had AIDS and 4 had died: 15 of the 17 subjects who were asymptomatic at entry remained so at the end of the study. There was a strict criterion for defining the asymptomatic category: subjects with any symptoms at all even if these were very minor were excluded. Subjects in the symptomatic category had CDC4E disorders either currently or in the past and these were mainly skin conditions. Subjects were also classified on more specific criteria of disease status partly based on CD4 slope and this is shown in the second half of Table 1.
Table 1 Clinical status at entry and at end of study (Feb '96)

<table>
<thead>
<tr>
<th>Clinical status</th>
<th>Entry</th>
<th>Feb '96</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>17</td>
<td>12</td>
</tr>
<tr>
<td>Symptomatic</td>
<td>81</td>
<td>57</td>
</tr>
<tr>
<td>ARC</td>
<td>26</td>
<td>18</td>
</tr>
<tr>
<td>AIDS</td>
<td>19</td>
<td>13</td>
</tr>
<tr>
<td>Died</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*1. CD4 ≥500x10^6/L, +ve slope
*2. CD4 ≥500x10^6/L, -ve slope
*3. 500x10^6/L > CD4 >350x10^6/L
*4. CD4 <350x10^6/L
*5. AIDS/ARC ≥ 7.5 years of infection
*6. 7.5 years < AIDS/ARC > 5 years of infection
*7. AIDS/ARC < 5 years of infection

*Groups 1-4: no AIDS/ARC

The variability and complexity of disease progression is illustrated by the differences between the two different ways of classifying the subjects: for example, it can be seen that although there were 33 individuals (23%) at entry with both a high CD4 count and a positive CD4 slope, who would be described as non progressors according to a strict criterion (Strathdee et al, 1996), only 17 were symptom free. The proportion
of these non progressors was reduced to 10% by the end of the study. There were 26 (18%) individuals with rapidly progressing disease who developed AIDS or ARC within 5 years of infection.

**Education and socioeconomic status:** The subjects were a highly educated group with only 16 (12%) claiming no educational achievements. 23 (16%) had reached GCE/GCSE O level standard, 20 (14%) A level/HNC, 22 (15%) had vocational qualifications, 32 (22%) had degrees and 30 (21%) postgraduate education. Socioeconomic status was consistent with the high level of education: 29 (20%) were classified AB compared with 16% of the general population, 79 (55%) C1 (general population, 27%), 26 (18%) C2 (general population, 26%) and only 8 (6%) were classified DE (general population, 31%).

**Psychiatric history:** 48 (34%) reported past use of psychotropic medication and 12 (8%) in-patient psychiatric treatment.

**Principal components analysis of the COPE**

Six factors were extracted accounting for 50% of the variance and items loading .40 or more on each factor are shown in Table 2. Full details of item loadings are shown in the Appendix. The factors were labelled as follows: Factor 1, Social Support; Factor 2, Disengagement; Factor 3, Religion; Factor 4, Acceptance; Factor 5, Problem Solving; Factor 6, Denial.
Table 2. COPE item loadings

<table>
<thead>
<tr>
<th>Factor 1 Social Support</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>I talk to someone about how I feel</td>
<td>.91 ESS1</td>
</tr>
<tr>
<td>I get sympathy and understanding from someone</td>
<td>.90 ESS4</td>
</tr>
<tr>
<td>I try to get emotional support from friends or relatives</td>
<td>.85 ESS2</td>
</tr>
<tr>
<td>I discuss my feelings with someone</td>
<td>.79 ESS3</td>
</tr>
<tr>
<td>I talk to someone to find out more about the situation</td>
<td>.76 ISS3</td>
</tr>
<tr>
<td>I try to get advice from someone about what to do</td>
<td>.73 ISS2</td>
</tr>
<tr>
<td>I feel a lot of emotional distress and find myself expressing those feelings a lot</td>
<td>.71 VENT3</td>
</tr>
<tr>
<td>I get upset and let my feelings out</td>
<td>.58 VENT1</td>
</tr>
<tr>
<td>I ask people who had similar experiences what they did</td>
<td>.56 ISS1</td>
</tr>
<tr>
<td>I get upset and am really aware of it</td>
<td>.55 VENT4</td>
</tr>
<tr>
<td>I keep myself from getting distracted by other thoughts or activities</td>
<td>.55 SOCA3</td>
</tr>
<tr>
<td>I talk to someone who could do something concrete about the problem</td>
<td>.53 ISS4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Factor 2 Disengagement</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>I just give up trying to reach my goal</td>
<td>.70 BDIS2</td>
</tr>
<tr>
<td>I admit to myself that I can’t deal with it and quit trying</td>
<td>.64 BDIS3</td>
</tr>
<tr>
<td>I give up the attempt to get what I want</td>
<td>.56 BDIS1</td>
</tr>
<tr>
<td>I reduce the amount of effort I’m putting into solving the problem</td>
<td>.52 BDIS4</td>
</tr>
<tr>
<td>I sleep more than usual</td>
<td>.47 MDIS4</td>
</tr>
<tr>
<td>I take additional action to get rid of the problem</td>
<td>-.45 ACT1</td>
</tr>
<tr>
<td>I learn something from the experience</td>
<td>-.41 PRAG3</td>
</tr>
<tr>
<td>I look for something good in the experience</td>
<td>-.40 PRAG1</td>
</tr>
<tr>
<td>I try to grow as a person as a result of the experience</td>
<td>-.40 PRAG4</td>
</tr>
</tbody>
</table>
### Factor 3 Religion

<table>
<thead>
<tr>
<th>Loading</th>
<th>.97 REL2</th>
<th>I put my trust in God</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>.96 REL1</td>
<td>I seek God’s help</td>
</tr>
<tr>
<td></td>
<td>.91 REL3</td>
<td>I try to find comfort in my religion</td>
</tr>
<tr>
<td></td>
<td>.79 REL4</td>
<td>I pray more than usual</td>
</tr>
</tbody>
</table>

### Factor 4 Acceptance

| .70 ACC1 | I learn to live with it |
| .70 ACC3 | I get used to the idea that it happened |
| .68 ACC4 | I accept the reality of the fact that it happened |
| .67 ACC2 | I accept that this has happened and that it can’t be changed |

### Factor 5 Problem Solving

| -.78 REST2 | I hold off doing anything about it until the situation permits |
| -.78 REST3 | I make sure not to make matters worse by acting too soon |
| -.57 REST4 | I restrain myself from doing anything too quickly |
| -.54 PLAN4 | I think about how I might best handle the problem |
| -.52 REST1 | I force myself to wait to wait for the right time to do something |
| -.50 PLAN3 | I think hard about what steps to take |
| -.48 PLAN2 | I make a plan of action |
| -.48 SOCA4 | I try hard to prevent other things from interfering with my efforts at dealing with this |
In the factor analysis, the factors were rotated using oblimin since there was no a priori reason to assume independence. Correlations between the factors are shown in Table 3. Religion was positively correlated with Social Support (Pearson’s r = .28, p < .01) and negatively with Problem Solving (r = -.27, p < .01). Social Support was negatively correlated with Problem Solving (r = -.33, p < .01). All other correlations were smaller than r = .13.

<table>
<thead>
<tr>
<th></th>
<th>Diseng</th>
<th>Relig</th>
<th>Accept</th>
<th>Prob</th>
<th>Denial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social support</td>
<td>-.01</td>
<td>.28**</td>
<td>-.01</td>
<td>-.33**</td>
<td>.10</td>
</tr>
<tr>
<td>Disengagement</td>
<td>.02</td>
<td>-.08</td>
<td>.01</td>
<td>.12</td>
<td></td>
</tr>
<tr>
<td>Religion</td>
<td></td>
<td>.08</td>
<td>-.27**</td>
<td>.12</td>
<td></td>
</tr>
<tr>
<td>Acceptance</td>
<td></td>
<td>-.01</td>
<td>-.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Problem Solving</td>
<td></td>
<td></td>
<td></td>
<td>-.03</td>
<td></td>
</tr>
</tbody>
</table>

**p < .01 (2 tailed)
Psychological characteristics at baseline

Table 4 (i-iv) shows the scores on the psychological variables at study entry. Where distributions were skewed, median scores are given.

4(i) Personality dimension scores (EPQ)

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>sd</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPQ Extraversion</td>
<td>13.3</td>
<td>4.9</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>13.6</td>
<td>5.4</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>4.6</td>
<td>2.7</td>
</tr>
<tr>
<td>Lie</td>
<td>7.45</td>
<td>3.9</td>
</tr>
</tbody>
</table>

Mean scores in the EPQ were close to those listed in the manual for males in the 40-49 year age group except for neuroticism where the mean score was at the upper end of the normal range.

4(ii) Psychiatric morbidity (GHQ-28)

<table>
<thead>
<tr>
<th></th>
<th>Median</th>
<th>Q1,Q3</th>
</tr>
</thead>
<tbody>
<tr>
<td>GHQ Somatic symptoms</td>
<td>6</td>
<td>3,11</td>
</tr>
<tr>
<td>Anxiety</td>
<td>6</td>
<td>4,9</td>
</tr>
<tr>
<td>Social dysfunction</td>
<td>7</td>
<td>6,9</td>
</tr>
<tr>
<td>Depression</td>
<td>1</td>
<td>0,4</td>
</tr>
<tr>
<td>Total</td>
<td>22</td>
<td>14,33</td>
</tr>
</tbody>
</table>
The median scores for the somatic, anxiety and social dysfunction scales fall just above the cut off point for caseness recommended by Goldberg & Hillier (1979) while that for the severe depression scale is well below caseness.

Further analysis showed that for somatic symptoms, 84 (59%) met the criterion for caseness and for anxiety and social function, the numbers were 102 (71%) and 127 (89%) respectively. For the depression scale, only 33 (23%) met the criterion for psychiatric caseness.

4(iii) Perceived Social Support (ISEL)

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>sd</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tangible</td>
<td>22.3</td>
<td>5.2</td>
</tr>
<tr>
<td>Belonging</td>
<td>22.8</td>
<td>5.6</td>
</tr>
<tr>
<td>Appraisal</td>
<td>19.5</td>
<td>3.9</td>
</tr>
<tr>
<td>Self esteem</td>
<td>20.9</td>
<td>4.4</td>
</tr>
<tr>
<td>Total</td>
<td>85.5</td>
<td>15.7</td>
</tr>
</tbody>
</table>

The scores obtained are similar to those reported by Lamping et al (1991) in a sample of 73 HIV infected individuals with a range of disease states (25% asymptomatic; 49% symptomatic; 26% AIDS) comparable to the present sample. The average total score in the Lamping et al group was 90.6, slightly higher than the 85.5 found here. Both samples with HIV obtain lower scores than those of other chronically ill groups.
4(iv) Life Events in last 12 months (LES)

<table>
<thead>
<tr>
<th></th>
<th>Median</th>
<th>Q1,Q3</th>
</tr>
</thead>
<tbody>
<tr>
<td>LES Negative</td>
<td>2</td>
<td>1,5</td>
</tr>
<tr>
<td>Positive</td>
<td>2</td>
<td>0,4</td>
</tr>
<tr>
<td>Total</td>
<td>6</td>
<td>5,12</td>
</tr>
</tbody>
</table>

Total number of life events is relatively low and there is no excess of negatively over positively rated events.

Table 5. shows the distribution of handedness. Since most of the subjects were completely or predominantly right handed, the distribution of positive scores was categorised into three groups and all completely or predominantly left handed individuals (negative scores) were grouped together. The proportions of completely left handed (8%) and completely right handed (61%) individuals were almost identical to those reported by Becker et al (1992) in their survey of a large sample of HIV positive gay men which indicated an increase in left handedness compared to normative data.

Table 5. Handedness distribution (Edinburgh Inventory)

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Completely right handed</td>
<td>87</td>
<td>61</td>
</tr>
<tr>
<td>(score = 100)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strongly right handed</td>
<td>29</td>
<td>21</td>
</tr>
<tr>
<td>(score = 50 to 99)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weakly right handed</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>(score = 1 to 49)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left handed</td>
<td>17</td>
<td>12</td>
</tr>
<tr>
<td>(score = -100 to - 1)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
The mean time from infection to CD4 count below 200 x 10^6/L for those not censored was 8.9 years (sd 2.5). Cox’s univariate regression analyses were carried out using the following continuous variables: EPQ, ISEL, LES, GHQ and COPE factor scores, age, CD4 slope and the following categorical variables: past psychotropic medication, psychiatric in patient treatment, Edinburgh Handedness, socioeconomic status, anti retroviral use and viral load. Values obtained for all the independent variables in the univariate analyses are given in the Appendix. Variables significant at p < .10 and which were entered in the final regression analysis are shown in Table 5. GHQ total was omitted to reduce the number of variables in the model since three of its subscales were represented.
Table 5. Variables identified at p<.10 in Cox Regression analyses of time to CD4<200x10^6L.

<table>
<thead>
<tr>
<th>Variable</th>
<th>R</th>
<th>Exp(B)</th>
<th>lower</th>
<th>upper</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISEL Tangible</td>
<td>-.07</td>
<td>.95</td>
<td>.90</td>
<td>1.00</td>
<td>.05</td>
</tr>
<tr>
<td>GHQ Somatic symptoms</td>
<td>.12</td>
<td>1.08</td>
<td>1.03</td>
<td>1.14</td>
<td>.004</td>
</tr>
<tr>
<td>GHQ Anxiety</td>
<td>.06</td>
<td>1.08</td>
<td>1.00</td>
<td>1.18</td>
<td>.06</td>
</tr>
<tr>
<td>GHQ Social dysfunction</td>
<td>.05</td>
<td>1.09</td>
<td>.99</td>
<td>1.20</td>
<td>.07</td>
</tr>
<tr>
<td>GHQ Total</td>
<td>.07</td>
<td>1.02</td>
<td>1.00</td>
<td>1.04</td>
<td>.03</td>
</tr>
<tr>
<td>COPE Problem solving</td>
<td>-.07</td>
<td>.73</td>
<td>.55</td>
<td>.98</td>
<td>.04</td>
</tr>
<tr>
<td>COPE Social support</td>
<td>.10</td>
<td>1.43</td>
<td>1.07</td>
<td>1.90</td>
<td>.01</td>
</tr>
<tr>
<td>CD4 Regression</td>
<td>-.12</td>
<td>.94</td>
<td>.97</td>
<td>.99</td>
<td>.004</td>
</tr>
<tr>
<td>Viral load:</td>
<td>.19</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>* Low vs below detectable level</td>
<td></td>
<td>1.88</td>
<td>.65</td>
<td>5.48</td>
<td>.24</td>
</tr>
<tr>
<td>High vs below detectable level</td>
<td></td>
<td>6.36</td>
<td>2.13</td>
<td>19.00</td>
<td>.001</td>
</tr>
<tr>
<td>Antiretroviral drugs</td>
<td>.10</td>
<td>.61</td>
<td>.61</td>
<td>.90</td>
<td>.01</td>
</tr>
</tbody>
</table>

* Low=1-100,000 RNA/ml ; High=100,001-1,400,000 RNA/ml
Table 6 shows the values obtained in the Multivariate Cox Regression analysis. The overall model was significant (Chi-Square = 35.95, df 9, p = .001).

Table 6. Multivariate Cox Regression: time to CD4 < 200 $\times 10^6$L

<table>
<thead>
<tr>
<th>Variable</th>
<th>R</th>
<th>Exp(B)</th>
<th>95% confidence intervals</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISEL Tangible</td>
<td>-.07</td>
<td>.94</td>
<td>.88 1.00</td>
<td>.06</td>
</tr>
<tr>
<td>GHQ Somatic symptoms</td>
<td>.02</td>
<td>1.06</td>
<td>.98 1.16</td>
<td>.15</td>
</tr>
<tr>
<td>GHQ Anxiety</td>
<td>.001</td>
<td>.97</td>
<td>.83 1.13</td>
<td>.68</td>
</tr>
<tr>
<td>COPE Social support</td>
<td>.001</td>
<td>1.22</td>
<td>.90 1.67</td>
<td>.20</td>
</tr>
<tr>
<td>COPE Problem solving</td>
<td>.001</td>
<td>.81</td>
<td>.59 1.18</td>
<td>.28</td>
</tr>
<tr>
<td>Viral load:</td>
<td></td>
<td>.12</td>
<td></td>
<td>.01</td>
</tr>
<tr>
<td>* Low vs below detectable level</td>
<td></td>
<td>1.50</td>
<td>.49 4.60</td>
<td>.48</td>
</tr>
<tr>
<td>High vs below detectable level</td>
<td></td>
<td>4.02</td>
<td>1.22 13.20</td>
<td>.02</td>
</tr>
<tr>
<td>Antiretroviral drugs</td>
<td>.09</td>
<td>3.38</td>
<td>1.16 9.87</td>
<td>.03</td>
</tr>
</tbody>
</table>

* Low = 1-100,000 RNA/ml; High = 100,001-1,400,000 RNA/ml

Viral load was the most significant predictor of time to CD4 count below 200 $\times 10^6$L (p = .01) and examination of the viral load groups demonstrates that this was largely due to the difference between the group with no detectable viral load and that with a high viral load of between 100,001 and 1,400,000 RNA/ml. The CD4 counts of those with a high viral load were 4 times as likely to decline to below 200 by the end of the study compared to those with no detectable viral load (p = .02). The use of antiretroviral drugs
was also a significant predictor of shorter time to CD4 count below 200 (p = .03): current or past users were more than three times as likely to have a CD4 count below 200 compared with non-users. None of the other variables except ISEL Tangible support which approached significance at p = .06, made a significant contribution to survival time to CD4 count below 200 x 10^9 L. The value obtained for tangible support suggests that higher perceived tangible support is associated with a decreased risk of decline in CD4 count to below 200 x 10^9 L. The effects of viral load and of antiretroviral use are shown in Figs 1 and 2.
Figure 1. Cox proportional hazards survival curves of time to CD4<200×10^6 L: Viral load group

Figure 2. Cox proportional hazards survival curves of time to CD4<200×10^6 L: Antiretroviral use
Effects on time to ARC/AIDS diagnosis

The analysis was repeated with the same independent variables using time to ARC/AIDS diagnosis as the dependent variable.

Table 7. Variables found significant at p < .10 in univariate Cox regressions of time to AIDS/ARC diagnosis

<table>
<thead>
<tr>
<th>Variable</th>
<th>R</th>
<th>Exp (B)</th>
<th>95% confidence intervals</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPQ Lie</td>
<td>.08</td>
<td>1.08</td>
<td>1.01 1.16</td>
<td>.03</td>
</tr>
<tr>
<td>GHQ Somatic</td>
<td>.13</td>
<td>1.09</td>
<td>1.03 1.15</td>
<td>.002</td>
</tr>
<tr>
<td>GHQ Anxiety</td>
<td>.11</td>
<td>1.12</td>
<td>1.04 1.22</td>
<td>.005</td>
</tr>
<tr>
<td>GHQ Total</td>
<td>.11</td>
<td>1.03</td>
<td>1.01 1.05</td>
<td>.004</td>
</tr>
<tr>
<td>COPE Acceptance</td>
<td>-.07</td>
<td>.77</td>
<td>.60 .99</td>
<td>.04</td>
</tr>
<tr>
<td>Viral load:</td>
<td>.12</td>
<td></td>
<td></td>
<td>.007</td>
</tr>
<tr>
<td>*Low vs below detectable level</td>
<td>.001</td>
<td>1.29</td>
<td>.53 3.13</td>
<td>.58</td>
</tr>
<tr>
<td>High vs below detectable level</td>
<td>.10</td>
<td>3.30</td>
<td>1.27 8.52</td>
<td>.01</td>
</tr>
<tr>
<td>Antiretroviral drugs</td>
<td>.10</td>
<td>2.80</td>
<td>1.31 5.97</td>
<td>.008</td>
</tr>
</tbody>
</table>

*Low=1-100,000 RNA/ml ; High=100,001-1,400,000 RNA/ml

Variables found significant at p < .10 in the univariate analyses were entered into a multivariate Cox regression. As before, GHQ total was omitted.
Table 8 shows the values obtained in this analysis. The overall model was significant (Chi-Square = 34.30, df 7, p = .001).

### Table 8. Multivariate Cox regression: time to AIDS/ARC diagnosis.

<table>
<thead>
<tr>
<th>Variable</th>
<th>R</th>
<th>Exp(B)</th>
<th>lower</th>
<th>upper</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPQ Lie</td>
<td>.05</td>
<td>1.08</td>
<td>.99</td>
<td>1.17</td>
<td>.09</td>
</tr>
<tr>
<td>GHQ Somatic</td>
<td>.05</td>
<td>1.07</td>
<td>.99</td>
<td>1.16</td>
<td>.09</td>
</tr>
<tr>
<td>GHQ Anxiety</td>
<td>.001</td>
<td>1.04</td>
<td>.92</td>
<td>1.18</td>
<td>.51</td>
</tr>
<tr>
<td>COPE Acceptance</td>
<td>-.11</td>
<td>.69</td>
<td>.52</td>
<td>.93</td>
<td>.01</td>
</tr>
<tr>
<td>Viral load:</td>
<td>.13</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Low vs below detectable level</td>
<td>.001</td>
<td>.73</td>
<td>.28</td>
<td>1.93</td>
<td>.53</td>
</tr>
<tr>
<td>High vs below detectable level</td>
<td>.04</td>
<td>2.38</td>
<td>.85</td>
<td>6.64</td>
<td>.09</td>
</tr>
<tr>
<td>Antiretroviral drugs</td>
<td>.08</td>
<td>2.64</td>
<td>1.06</td>
<td>6.56</td>
<td>.04</td>
</tr>
</tbody>
</table>

*Low = 1-100,000 RNA/ml; High = 100,001-1,400,000 RNA/ml

Significant predictors of ARC or AIDS diagnosis were viral load (p = .006), anti retroviral use (p = .04) and COPE Acceptance (p = .01). Viral load was significant overall but although the comparison between high viral load and no detectable viral load approached significance (p = .09), the 95% confidence intervals suggest that this is not a reliable finding. Antiretroviral use was again a significant predictor in the model. Higher COPE acceptance was associated with a decreased risk of an ARC or AIDS diagnosis. The effects of viral load and antiretroviral use are shown in Figs. 3 and 4 and that of COPE acceptance in Fig. 5.
Figure 3. Cox proportional hazards survival curves of time to ARC/AIDS diagnosis: Viral load group

Figure 4. Cox proportional hazards survival curves of time to ARC/AIDS diagnosis: Antiretroviral use
Figure 5. Cox proportional hazards survival curves of time to ARC/AIDS diagnosis: COPE acceptance.
Relationship of personality with coping, psychiatric morbidity and perceived social support

Correlations were carried out using Pearson’s r to determine the relationships between EPQ dimensions and scores on the COPE factors, and using Spearman’s rho for relationships between EPQ and GHQ and ISEL scores.

Table 9. Correlations between EPO and COPE, GHQ and ISEL

<table>
<thead>
<tr>
<th></th>
<th>Extraversion</th>
<th>Neuroticism</th>
<th>Psychoticism</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>COPE</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social support</td>
<td>.22***</td>
<td>.10</td>
<td>-.01</td>
</tr>
<tr>
<td>Disengagement</td>
<td>-.14</td>
<td>.41**</td>
<td>.14</td>
</tr>
<tr>
<td>Religion</td>
<td>.10</td>
<td>.08</td>
<td>.01</td>
</tr>
<tr>
<td>Acceptance</td>
<td>.04</td>
<td>-.08</td>
<td>-.05</td>
</tr>
<tr>
<td>Problem solving</td>
<td>.02</td>
<td>.05</td>
<td>-.01</td>
</tr>
<tr>
<td>Denial</td>
<td>-.03</td>
<td>.12</td>
<td>.33***</td>
</tr>
<tr>
<td><strong>GHQ</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Somatic symptoms</td>
<td>-.17*</td>
<td>.30**</td>
<td>.02</td>
</tr>
<tr>
<td>Anxiety</td>
<td>-.11</td>
<td>.47**</td>
<td>.01</td>
</tr>
<tr>
<td>Social dysfunction</td>
<td>-.13</td>
<td>.28**</td>
<td>-.06</td>
</tr>
<tr>
<td>Depression</td>
<td>-.11</td>
<td>.55**</td>
<td>.15</td>
</tr>
<tr>
<td><strong>ISEL</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tangible</td>
<td>.15</td>
<td>-.33**</td>
<td>.52</td>
</tr>
<tr>
<td>Belonging</td>
<td>.36**</td>
<td>-.34**</td>
<td>-.10</td>
</tr>
<tr>
<td>Appraisal</td>
<td>.20*</td>
<td>-.37**</td>
<td>-.09</td>
</tr>
<tr>
<td>Self-esteem</td>
<td>.28**</td>
<td>-.35**</td>
<td>-.03</td>
</tr>
</tbody>
</table>

*p < .05  **p < .01 (2-tailed)
Among the COPE factors, there were only three significant correlations, each with one of the EPQ dimensions: extraversion was associated with social support coping \((r = .22, p < .05)\), neuroticism with disengagement \((r = .41, p < .01)\) and psychoticism with denial \((r = .33, p < .01)\). All the subscales of the GHQ showed significant associations with EPQ neuroticism \((all \ p < .01)\) and there was a modest negative correlation between extraversion and somatic symptoms \((r = .17, p < .05)\). For the ISEL, there were positive associations between extraversion and belonging support \((r = .36, p < .01)\), self esteem support \((r = .28, p < .01)\) and appraisal support \((r = .20, p < .05)\). EPQ neuroticism was significantly negatively correlated with all four ISEL subscales \((all \ p < .01)\) and there were no significant relationships between EPQ psychoticism and any of the social support measures.

**DISCUSSION**

This study examined the role of psychological variables in disease progression among a group of gay men infected with HIV for at least 8 years representing a range of clinical states and who were followed up clinically for up to 32 months. Only one psychological factor was found to be significantly related to disease progression while biomedical variables were predominant in predicting survival time to both the dependent variables of CD4 count below \(200 \times 10^6\) and an ARC or AIDS diagnosis.

Decreased survival to \(CD4 < 200 \times 10^6\) was predicted by higher levels of viral load and by antiretroviral use and none of the psychological variables made a significant contribution to the model. Viral load measures have only recently become accepted as important prognostic and clinical markers and this finding supports the significance of viral load in predicting a CD4 count level at which symptomatic illness becomes more likely. The finding that antiretroviral use is associated with decreased time to a low CD4 count is at first sight counterintuitive and two factors are likely to be responsible. Until the findings of the Concorde trial were published in 1994 (Concorde Coordinating Committee), zidovudine, the only licensed antiretroviral drug available at the time, was widely prescribed at relatively early stages of infection and
before the development of significant symptoms. Since the sample represents a population infected before 1988 and recruited to the study between 1993 and 1995, it is not surprising to find that almost 70% had a history of anti retroviral drug use. In other words, many individuals with HIV infection took zidovudine when their CD4 counts were still relatively high and when their future disease progression was unknown. A proportion may have been those with eventually more rapidly declining CD4 counts or, more importantly, those who developed resistance to the drug so that it was ineffective in preventing decline later on in disease. More recently, the practice has been to prescribe zidovudine when individuals have significant symptoms and since more than one third of the sample had developed ARC or AIDS by the end of the study, the relationship of antiretroviral use and CD4 decline may be mediated by more symptomatic disease. An association between zidovudine use and faster progression from seroconversion to death was reported in a longitudinal study of 403 gay men (Veugelers et al, 1994) and attributed by the authors to the fact that those with more rapid disease progression are more likely to take antiretroviral medication.

In the analysis of time to ARC or AIDS diagnosis, which are clinically more important markers of disease progression, viral load and antiretroviral drug use were again significant predictors of increased risk while acceptance coping was significantly associated with decreased risk of progression. This latter finding is at odds with the report from Reed et al (1994) in which realistic acceptance coping derived from a factor analysis of the Ways of Coping Checklist (Folkman & Lazarus, 1980) was a significant predictor of decreased survival time among gay men with AIDS. The statistical analysis conducted by Reed et al was similar to that carried out in the present study although the authors acknowledge that their sample of 74 was relatively small. Realistic acceptance was included in each of a series of five Cox proportional hazards regression analyses to control for established biological and demographic risk factors, subjective state including psychological distress and dispositional optimism, and potential behavioural mediators: acceptance predicted significant variance in survival time in all the analyses.
What then are the possible sources for these contrasting findings? Reed et al felt that there was a considerable overlap between realistic acceptance and the stoic acceptance response pattern associated by Greer et al (1991) with reduced survival time among women with breast cancer. Although the coping response labels in all three studies appear to indicate similar concepts, their components are somewhat different. The items making up the acceptance factor in the present sample eg. ‘I accept the reality of the fact that it has happened’, ‘I learn to live with it’ do not have the elements of resignation and fatalism apparent in Greer et al’s description of stoic acceptance eg. ‘It’s cancer, I just leave it all to the doctors’ nor the future orientation of some of the realistic acceptance items in the Reed et al study eg ‘Try to accept what might happen’, ‘Prepare myself for the worst’. These differences of emphasis may only reflect the differences in the methods of assessing coping or those in the samples which, in the case of the two other studies, involved subjects already coping with the knowledge that they had a serious illness. Reed et al’s realistic acceptance was positively correlated with three of the five other coping factors identified namely active cognitive coping, seeking social support and seeking information although these factors were not significant predictors in the regression analysis in which they were included. In the present study, acceptance coping was not significantly associated with any of the other factors derived from the COPE nor with personality, psychiatric morbidity or perceived social support suggesting that it made a relatively independent contribution to the prediction of ARC and AIDS free survival.

Kubler-Ross (1987) has extended her ideas about adjustment to terminal illness to those with AIDS and suggests that acceptance, characterised by a tired and peaceful state and by resignation to the prospect of death, is psychologically adaptive. In contrast, Taylor et al (1992) found that unrealistically optimistic views among seropositive gay men about the future consequences of HIV were associated with better psychological adjustment although of course neither these findings nor the proposals of Kubler-Ross imply any effect on disease progression. How could acceptance act to influence disease outcome? Accepting individuals may be more likely than individuals low in acceptance to monitor their health more closely, seek
medical attention promptly and comply with treatment recommendations and they may also be more likely to adopt healthier lifestyles. It is also conceivable that acceptance is a response to a less aggressive disease state or that it has an impact on immune functioning although as discussed in the introduction above, a clinically significant effect has proved difficult to demonstrate for any psychological factor.

In the factor analysis of the COPE, although coping styles describing disengagement and denial were identified, no coping factor which could accurately be described as active was found and there were no associations between disengagement or denial coping and disease progression in contrast to the findings of other studies (Goodkin et al, 1991; Antoni et al, 1991b, 1995). One of the difficulties in comparing studies is the fact that different measures of coping are used. Even in studies using the COPE, the data is treated in different ways: in the Antoni et al studies, the original 13 scales were used while Goodkin et al (1992) conducted a principal components analysis to reduce the number of variables and produced a four component solution. The first factor, labelled ‘active coping’, was composed of eight of the original COPE scales including acceptance and restraint as well as scales with a more clearly active component and this was the factor found to have a significant relationship to NK cell count. Mulder et al (1995) describe an active confrontational coping factor which was developed by first carrying out a factor analysis of responses to eight of the original COPE scales in a separate study, producing three subscales to which were added two extra subscales. A second factor analysis produced two factors with active confrontational coping representing seeking social support, problem solving and the inverse of denial and proving to be significant in the prediction of decreased clinical progression. Hence in the present study, coping factors were identified which showed some overlap with those reported in the other studies, but there was no significant effect of denial or disengagement nor of a clearly active coping component.

The negative findings in relation to psychological distress, past psychiatric history, life events and perceived social support are in line with those of other studies nor were there any effects of personality, handedness or socioeconomic status on disease.
Part of the interest in seeking to identify psychological factors in progression is the potential to develop interventions to modify them in a helpful direction and to this extent, it is reassuring that handedness and personality traits which by definition are not amenable to modification, did not confer any change in risk. Finally, age which is an established risk factor for more rapid progression (Kaslow et al, 1987) was not related to either time to CD4 count below 200 or time to an ARC or AIDS diagnosis in this sample. This is unsurprising since the sample was selected to be reasonably homogeneous for age, the range of which was therefore limited.

As expected, EPQ neuroticism was positively correlated with psychiatric morbidity as indexed by scores on the four subscales of the GHQ confirming the well established relationship of neuroticism and psychological distress. Neuroticism was also negatively correlated with all aspects of perceived social support while extraversion showed positive associations with perceived social support subscales referring to the availability of people to share time and pleasant activities with and to the availability of positive comparisons between self and others. This is consistent with the findings of Henderson et al (1981) who reported significant relationships between extraversion and the perceived availability and adequacy of supportive ties in a community sample. The problem of measurement redundancy has been highlighted by Monroe and Depue (1986) who point out that items in the EPQ used to define and measure the personality dimensions are almost synonymous with the items often used to assess social support rendering associations between them inevitable.

The hypotheses with respect to personality and coping were partially supported: neuroticism was significantly associated with disengagement coping, although not with the only other avoidant coping factor of denial, and extraversion was positively correlated with social support coping. Psychoticism was significantly associated with denial which is perhaps not a predictable relationship: psychoticism would more
readily be expected to have a negative relationship with socially oriented coping. As in the case of the ISEL, relationships between social support coping and extraversion are partly predictable on the basis of item content overlap between the two measures.

The sample in this study is of course not representative of HIV infected individuals since it was specifically selected to include only those with a long period of infection and to represent a range of disease progression rates. Other sources of bias include the specific inclusion of a subgroup of long term infected participants with high CD4 counts and few or no symptoms, the high level of education and socioeconomic status and the unusually low levels of depression. The fact that the subjects agreed to take part when patients of HIV clinics are frequently and repeatedly asked for participation in research is a common difficulty in interpreting the results in studies in this area and those who did not may have been more or less ill, or different on any or all of the psychological measures.

The study design has considerable shortcomings. The main difficulties are the timing of the psychological measures, the variable period of follow up and the fact that, apart from the 22 seroconverters, the duration of infection cannot be definitely established. With respect to the latter, although the first AIDS case in the UK was reported in late 1981, HIV testing was not widely available until 1984 so that at the beginning of recruitment to the study in August 1993, no subject could have been identified as HIV positive for longer than 9 years unless they had been tested in the USA or in the course of a research study. The mean duration of infection at study entry was 8.6 years, close to the maximum possible.

The proportion of non progressors at the end of the study when average duration of infection was close to 10 years, was 10% and similar to proportions of between 5 and 15% reported from other studies of long term infected individuals (Easterbrook, 1994). Nevertheless, this relatively small proportion of non progressors means that if psychological factors are relevant, underestimating length of infection in this group as well as among the small number of subjects with rapidly progressing disease could

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attenuate any effects on the dependent variables. Another potential reason for null findings is the disease-based spectrum bias (Miller, 1994) which has been described for studies of psychological factors in coronary heart disease. The proposal is that since both the levels of disease and the presumed risk factor (hostility) are increased, the ranges of the severity of illness measures and of the psychological variables of interest are reduced, resulting in less likelihood of finding significant effects.

Because the psychological variables were assessed after a long period of infection, it is impossible to know at least for those variables which are not necessarily stable over time and situations, whether scores represent adjustment to infection, transient change or more enduring characteristics. As discussed above, there is also some evidence from studies of breast cancer that the time at which coping, mood or psychiatric morbidity are assessed influences predictions about disease outcome. These issues can only be adequately addressed in the context of a prospective study with psychological assessment at the inception of infection.

In the present study, although the period between assessment and the end of the study was variable, longer follow up would probably not have proved useful because of radical changes in treatment introduced during 1996. Combination antiretroviral therapy and protease inhibitors mean that for many HIV infected individuals, including the surviving participants in this study, viral load can be reduced, CD4 counts augmented and the course of the disease altered. The pace of change in HIV research and treatment has always made it difficult to control all the variables in any investigation but it now does seem that HIV infected people can feel more optimistic about their future than ever before.

In summary, the results of this study indicate that biomedical variables are the most important factors in HIV disease progression. No effects of personality, psychological morbidity, perceived social support, life events or handedness were demonstrated but acceptance coping was a significant predictor of longer ARC and AIDS-free survival.
REFERENCES


Veugelers, P. J., Page, K. A., Tindall, B., Schechter, M. T., Moss, A.R.,


APPENDICES

1. CDC Classification
2. Factor Analysis: COPE
3. Time to CD4 $< 200 \times 10^6$: Univariate Cox Regressions
4. Time to AIDS/ARC Diagnosis: Univariate Cox Regressions
CDC CLASSIFICATION

CDC 1. Recently observed illness, with seroconversion: (1)

CDC 2. Well, no generalised lymphadenopathy: (2)

CDC 3. Well with generalised lymphadenopathy: (3)

CDC 4A. Significant constitutional disease: (ARC OR AIDS)
- Fever or nights sweats for over one month (ARC) (4)
- Weight loss >10% (ARC) (5)
- Diarrhoea for over one month, no cause (ARC) (6)
- # Wasting Syndrome (AIDS) weight loss and diarrhoea, or fever and weakness for over one month (7)

CDC 4B. Neurological:
- # Dementia (disabling encephalopathy) (AIDS) (10)
- Myelopathy (11)
- Peripheral Neuropathy (12)

CDC 4C1. Infections in AIDS:
Diagnosed by definitive method:
- # Pneumocystis Carinii Pneumonia (15)
- # Cerebral Toxoplasmosis (16)
- # Cryptosporidiosis over one month (17)
- # Oesophageal Candida (18)
- # Bronchial Candida (19)
- # Cryptococcosis (20)
- # Mycobacterium Avium Complex (MAC) (21)
- # CMV Retinitis (22)
- # CMV Oesophagitis (23)
- # CMV Colitis (24)
- # CMV Pneumonitis (25)
- # Other proven CMV (26)
- # Mucocutaneous HSV over one month (27)
- # Other HSV (28)
- # Progressive Multifocal Leukoencephalopathy (PML) (29)
- # Isosporiasis (30)
- # Histoplasmosis (31)
- # Coccioidioidomycosis (32)
- # Extrapulmonary TB (33)
- # Pulmonary TB (46)
- # Recurrent Salmonella Septicaemia (34)
- # Recurrent Bacterial chest infections (73)

Diagnosed presumptively:
- # Pneumocystis Carinii Pneumonia (37)*
- # Cerebral Toxoplasmosis (38)
- # Mycobacterium Avium Complex (MAC), only AFB+ (39)
- # Oesophageal Candida (40)

*Presumptive PCP = recent symptoms, abnormal CXR, PaO2 < 9.3 and no bacterial pathogen

Diagnosed by presumptive methods:
- Oral Hairy Leukoplakia (43)
- Shingles, multidermatomal (44)
- Nocardia (45)
- Oral Candida (47)

CDC 4C2. Infections in ARC:
- Oral Hairy Leukoplakia (43)
- Shingles, multidermatomal (44)
- Nocardia (45)
- Oral Candida (47)

CDC 4D. Neoplasms in AIDS:
Diagnosed by definitive method:
- # Cerebral Lymphoma (50)
- # Kaposi's Sarcoma (51)
- # Non-Hodgkins Lymphoma (52)
- # Invasive Cervical Carcinoma (78)

Diagnosed presumptively:
- # Kaposi's Sarcoma (55)
- # Cerebral Lymphoma (56)

CDC 4E. Possible HIV associated disease (not AIDS):

Constitutional (not ARC or AIDS):
- Fatigue (59)
- Intermittent Diarrhoea (60)
- Intermittent fever/sweats (61)
- Weight loss < 10% (62)

Infections:
- Strongyloidesis (64)
- Leishmaniasis (65)
- Presumptive CMV disease (66)
- Aspergillosis (67)
- Microsporidiosis (68)
- Salmonella, septicaemia-one episode (69)
- Shigellosis (70)
- Campylobacter (71)
- Giardiasis (72)
- Amebic Dysentery (74)
- Other Infections (75)

Neoplasms:
- Hodgkin's Lymphoma (76)
- Invasive Anal Carcinoma (77)
- Other Neoplasms (79)

Skin:
- Seborrhoeic Dermatitis (82)
- Folliculitis (83)
- Dry skin (84)
- Frequent HSV (85)
- Porpura (86)
- HSV, Shingles, one dermatome (87)
- Facial warts (88)
- Néo-genital Molluscum Contagiosum (89)
- Other skin (90)
- Fungal Toenail Dystrophy (91)
- ZDV Nail Dyschromia (113)

ENT:
- Gingivitis (93)
- Aphthous Ulcer (94)
- Angular Stomatitis/Chelitis (95)
- Sinusitis, chronic (96)
- Otitis Externa, chronic/recurrent (97)

Haematological:
- Thrombocytopenia, idiopathic = < 20 (100)
- Thrombocytopenia 21-150 (101)
- Anaemia, no cause except HIV (104)
- Haematological Other (105)

Neurological:
- Aseptic Meningitis (107)
- Guillain-Barre syndrome (108)
- Neurological other (109)

Cardiovascular:
- Cardiomyopathy (114)
- Cardiovascular Other (115)

Gastrointestinal:
- Sclerosing Cholangitis (116)
- Other GI (117)

General other:
- Arthralgia (102)
- HIV Myopathy (103)
- Splenomegaly (111)
- ZDV myopathy (112)
- Primary Gonadal failure (118)
- Endocrine Other (119)
- General Other (120)

[4] CD4 Count <200 without an AIDS defining illness is considered as an AIDS diagnosis in the USA (but not in Europe).

# denotes AIDS Diagnosis
( ) Diagnosis Code
# Factor Analysis

## Pattern Matrix:

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5. DIP. CLIN PSYCH DISSERTATION

THE EFFECTS OF AUTOHYPNOSIS AND PROGRESSIVE MUSCLE RELAXATION ON HABITUATION OF ELECTRODEMAL RESPONSES TO AUDITORY STIMULI AMONG ANXIOUS PATIENTS.
THE EFFECTS OF AUTOHYPNOSIS AND PROGRESSIVE MUSCLE
RELAXATION ON HABITUATION OF ELECTRODERMAL RESPONSES
TO AUDITORY STIMULI AMONG ANXIOUS PATIENTS

SUSAN THORNTON, 1983

Submitted in fulfilment of Part III
of the British Psychological Society
Diploma in Clinical Psychology
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ABSTRACT

The function of relaxation training in behavioural treatments of anxiety reduction remains a contested issue. Relaxation techniques are generally assumed to produce reductions in tonic levels of autonomic arousal and similar decrements have been found to accompany progressive muscle relaxation and auto-hypnosis in comparisons among non-clinical subjects. It is argued that habituation of the electrodermal orienting response is an appropriate analogue of anxiety reduction and that optimal effects will result from procedures which act on both the sensitisation and habituation components of response decrements. It is not clear that progressive muscle relaxation facilitates habituation of electrodermal orienting responses but such facilitation has been found among subjects listening to an auto-hypnosis induction.

In this study anxious patients were given five sessions of auto-hypnosis or progressive muscle relaxation. Pre-treatment measures of habituation of electrodermal activity to auditory tones were compared with measures obtained in the fifth treatment session.

Although facilitation of habituation occurred in both groups in the latter session, there were no differences among the groups which were attributable to treatment.
INTRODUCTION

Since the introduction of relaxation training by Jacobson (1938), its use in the treatment of anxiety and stress disorders has become widespread and it is frequently used in behavioural methods of fear reduction. As well as progressive muscle relaxation, a wide range of other techniques, including meditation and hypnosis is reported in the literature. A recent review of procedural variables in relaxation training (Hillenberg and Collins, 1982) concludes that such training is apparently an effective therapeutic intervention across a wide range of problems. However, the procedural variability, lack of specification of content of the training, and differences in choice of dependent variables make it difficult to draw conclusions, either about the relative efficacy of relaxation procedures, or about the mechanisms underlying their effects. There is consequently little guidance for the clinician in making an appropriate choice of technique for a particular client.

Investigations of progressive muscle relaxation have often used measures of autonomic activity as the dependent variables: the central assumption is that relaxation acts to lower muscle tension or autonomic arousal, or both. A review of procedural variables in twenty five studies of the physiological effects of muscle relaxation (Borkovec and Sides, 1979a) showed that patient populations are more likely than analogue populations to evidence physiological changes. This observation was also made by Mathews (1978). A review of controlled studies among patient populations, in which muscle relaxation training was used as sole or adjunctive treatment for fear reduction, indicates that the training has its principal effect on physiological measures (Glaister, 1982).
These effects include reductions in heart rate, blood pressure, muscle tension and both skin conductance levels and nonspecific responses. A lowered state of arousal induced by relaxation is thought to enhance fear reduction in a number of ways. The traditional reciprocal inhibition approach assumes that the response to fear stimuli is inhibited and a response antagonistic to anxiety produced (Wolpe, 1958). An alternative explanation is that the lowered state of arousal increases vividness of imagery, and the autonomic response to it, and maximises response decrements to repeated exposures of fear stimuli (Mathews, 1971).

Hypnosis as a means of producing relaxation has a long history (reviewed by Edmonston, 1981). It would be appropriate to define what is meant by hypnosis, however such a definition has proved difficult. The main division in theoretical positions concerns the state, non-state issue. State theorists (e.g. Bowers, Hilgard, Orne) tend to focus on the psychological and physiological differences between the hypnotic and normal waking state. Non-state theorists (e.g. Barber, Coe, Sarbin) emphasise the interactional effects of person and setting and deny the need to invoke a particular state as an explanatory concept. Sheehan and Perry (1980) have pointed out that the research strategies used to investigate hypnosis have inevitably been influenced by the theoretical assumptions of the investigator concerning the nature of hypnosis. No attempt at defining hypnosis in the wider sense will be made here. Instead, an empirical approach will be adopted in which hypnosis is defined as the physiological state produced in individuals by the application of a standard hypnotic induction, Gill and Brenman (1959) summarised the characteristics shared by hypnotic induction techniques as: (a) the limitation of sensory intake, bodily activity, and attention; (b) the provision of restricted and monotonous stimulation; and (c) the alteration of the quality of bodily awareness.
Standard instructions produce neutral hypnosis (Edmonston, 1981) and do not confound induction with the addition of therapeutic or other direct suggestions regarding physiological or emotional states. Dengrove (1973) among others, has advocated the use of hypnosis in behavioural approaches to treatment, because of the facilitating effects of relaxation and visual imagery, and Wolpe himself noted the effectiveness of hypnotic techniques in reciprocal inhibition (Wolpe, 1958).

Authors of general surveys of the physiology of hypnosis conclude that there is no stable concomitant of the hypnotic state, e.g. Barber (1970). However, there are few well controlled studies which measure physiological changes following a neutral hypnosis induction. Many of the studies have faults such as the omission of procedural details, the use of inadequate or inappropriate control groups, and the failure to consider hypnotic susceptibility in the selection of subjects. Two recent controlled studies report on physiological measures among non-patient subjects. Walrath and Hamilton (1975) compared baseline and treatment periods for meditation, autohypnosis and relaxation. Subjects were screened for susceptibility and measures of heart rate, respiration rate, skin conductance levels and non-specific responses were recorded. While there was a significant decrease in all measures from the baseline to the treatment period, there were no differences among the groups. Similar results were obtained by Morse et al. (1977), who compared six treatment conditions, including autohypnosis and meditation, among four groups of subjects, defined by training or no training in these two techniques. Each subject received all treatments in a counterbalanced order. None of the physiological measures, which included skin conductance, EEG, pulse rate, and EMG, differentiated among the various forms of relaxation. There appears to be only one investigation of the
physiological concomitants of hypnotic treatment among anxious patients, in which a direct comparison was made with another form of relaxation. Benson et al. (1978) divided their subjects into two treatment groups approximately equated for hypnotic susceptibility. Baseline physiological measures of blood pressure, oxygen consumption and heart rate were taken, as well as measures of anxiety, and the subjects were taught either meditation, relaxation or autohypnosis. They practiced for eight weeks with no further therapeutic intervention. Upon reassessment, there was no difference among the groups in effectiveness on anxiety reduction, nor any difference, except for diastolic blood pressure, in the physiological measures. However, because over half the subjects did not complete the study, the data must be considered unreliable.

There are apparently no studies comparing the physiological effects of hypnotic induction and progressive muscle relaxation in a clinical population, but studies among non patients have been reported. Paul (1969) compared progressive muscle relaxation with hypnotic relaxation and self-relaxation among 20 female undergraduate students. Physiological measures included heart and respiration rates, muscle tension, and skin conductance. Both hypnosis and muscle relaxation produced significant decreases in all the measures with the exception of skin conductance. The failure of this measure to differentiate among the three groups was attributed to the increased vasodilation produced by relaxation which would tend to maintain the conductance levels originally measured in an aroused state. Coleman (1976) compared hypnotic induction with progressive muscle relaxation in a standard form, and without the use of descriptive language (i.e. instructions to tense and release only). Measures of EMG and EEG did not differentiate the groups. These findings have led to the view that induction of neutral hypnosis and other forms of relaxation, including progressive muscle relaxation, lead to essentially the same physiological state characterised
by decreased activity of the sympathetic nervous system (Benson et al., 1981; Edmonston, 1981).

All the studies described above have monitored tonic levels of physiological arousal. The use of phasic measures enables the investigator to specify more closely which central processes may be involved and to relate the investigation to specific theoretical concepts about clinical conditions and their treatments. The orienting response and its habituation have been the subjects of extensive study and the electrodermal orienting response (EDOR) has been used in studies of anxiety and fear reduction. There are several theoretical arguments underpinning the adoption of habituation of this response as an analogue of fear reduction. The work of Pribram and his associates (Pribram and McGuinness, 1975) has shown that elicitation and habituation of the EDOR is a function of fronto-limbic regions of the brain. Current models of habituation focus upon its relationship to attentional processes (Waters et al., 1977; Ohman, 1979) and distinguish sensitisation as well as inhibitory processes underlying response decrements (Groves and Thompson, 1970). Gray (1975, 1982) has proposed a three system model of emotion consisting of an arousal system, which increases behavioural intensity, and two mutually antagonistic systems which determine whether behaviour is activated or inhibited. This latter behavioural inhibition system is postulated to be responsive to conditioned stimuli of punishment or non-reward, innate fear stimuli, and novel stimuli. Its outputs are the inhibition of ongoing behaviour, increments in arousal, and increased attention. Gray proposes that excessive activity in this system constitutes anxiety and its postulated structure includes the septo-hippocampal system and neocortical inputs from the prefrontal cortex. The basic function of the behavioural inhibition system is the comparison of actual with expected stimuli, and Gray presents extensive evidence relating attentional and habituation
processes to its neuropsychological structure. Fowles (1980), in a review of much of the relevant psychophysiological human literature, concludes that electrodermal activity increases with activation of the behavioural inhibition system. He also suggests that the directional fractionation of heart rate and electrodermal activity, described by Lacey (1959), can be understood in terms of Gray's three system analysis. While heart rate is strongly associated with activity of the behavioural activating system and active coping in the face of threat, electrodermal activity is responsive to stimuli which activate the behavioural inhibition system, and do not produce increases in heart rate.

Lader and his colleagues proposed a model of anxiety reduction during systematic desensitisation based on the concept of habituation, as an alternative to the current explanations of reciprocal inhibition and extinction. The maximal habituation hypothesis, outlined by Lader and Mathews (1968), proposed that the rate of decrement of electrodermal responses to phobic stimuli is a habituation process, maximised by the low levels of central arousal induced by relaxation. Evidence in support of their formulation came from studies in which the rate of habituation of EDOR to auditory stimuli was found to correlate with clinical response to treatment (Lader, et al., 1967), and in which progressive muscle relaxation reduced the number of nonspecific electrodermal responses (Mathews and Gelder, 1969). However, direct evidence of the facilitation of habituation of electrodermal responses by relaxation is lacking. Teasdale (1972), in a series of four experiments among non-clinical subjects, found no effect of relaxation on response decrements to a series of tones. While the evidence from more recent clinical studies leaves little doubt that exposure treatments produce reliable decrements in physiological response to fear evoking stimuli (e.g. Watson et al., 1972), the conditions which maximise these decrements are
still unclear, and the role of relaxation remains a contested issue. In an experiment designed to clarify the roles of relaxation and expectation in a systematic desensitisation paradigm, Borkovec and Sides (1979b) found no support for the Lader and Mathews proposition that relaxation produces maximal (low tonic arousal) conditions for habituation. Relaxation appeared to facilitate heart rate response decrements to hierarchy scenes, however this may have been due to the increased response to the first stimulus among those receiving relaxation. This explanation has also been offered for the similar results of Wolpe and Flood (1970). Further, if the Gray model is adopted, together with Fowles' evidence of a dissociation of heart rate and electrodermal activity with respect to systems underlying fear reduction, heart rate data become difficult to interpret. Another difficulty of interpretation in studies where phobic stimuli are used arises from the fact that habituation is critically determined by the signal value attributed to the stimuli (e.g. Graham, 1979). Procedural variability in the presentation of stimuli and in instructions to the subjects introduces a considerable source of variance. Given Iacono and Lykken's (1979) suggestions regarding the importance of instructions, one hypothesis could be that relaxation may in some circumstances be a means of giving non signal value to the orienting stimuli, by engaging the subject's attention.

Watts (1979), in a reformulation of the habituation hypothesis of fear reduction, suggests that a consideration of the dual process theory of habituation (Groves and Thompson, 1970) may serve to integrate disparate findings and to generate testable predictions about factors which will enhance anxiety reduction. He proposes that short term (within - session) decrements are facilitated by relaxation, because of its action on sensitisation and implies that long-term (between - session)
anxiety reduction will be dependent on procedures which facilitate the habituation component. There is a body of work suggesting that within and between-session habituation are partly independent processes (Davis, 1970) with the former being associated with processes in the reticular formation (Groves and Lynch, 1972) and the latter involving elaboration by forebrain structures. However, recent research on the return of fear between sessions in which substantial reductions have been achieved (Grey et al., 1979), has indicated that between-session habituation is dependent upon within session habituation (e.g. Sartory et al., 1982; Grayson et al., 1982). Thus the sine qua non of fear reduction appears to be the facilitation of within-session habituation by action on both components of the habituation process. So far, progressive muscle relaxation has not been clearly shown to have such an effect.

There has been very little controlled research into the effects of hypnotic induction on habituation of the EDOR. In a study of hypnotically induced amnesia, Stern et al. (1963) made the incidental observation that habituation of the EDOR appeared to be somewhat accelerated during hypnosis. Pessin et al. (1968), in an experiment manipulating hypnotic instructions and attentional sets, found a reduction in numbers of EDOR's in the hypnosis groups. Brow and Gruzelier (1982) investigated bilateral EDORS to incidental tones heard during a standardised hypnotic induction. Subjects were cardiac outpatients and medical students and groups were equated for individual differences in habituation rate to the tones in a pretest. The hypnosis condition was counterbalanced with two control conditions designed to counter the effects of relaxation or divided attention, and susceptibility to hypnosis was measured during the induction. Hypnosis was found to alter habituation rate. Susceptible subjects
showed faster habituation and a reversal of bilateral differences in amplitude of responses, compared to the pretest and control conditions. These changes were not found among unsusceptible subjects. The results implied facilitation of frontal-limbic inhibitory influences on habituation under hypnosis coupled with a shift in hemispheric balance from left to right. Hypnotic procedures have been hypothesised to involve a shift in lateral balance such that the left hemisphere's influence is reduced, liberating the normally inhibited functions of the right hemisphere (Graham, 1977). However, empirical evidence for this view has been equivocal (e.g. Frumkin, 1978; Graham and Pernicana, 1979).

The mediation of hemispheric influences on electrodermal activity is imperfectly understood. Evidence suggests that influences can be both excitatory and inhibitory and that ipsilateral and contralateral pathways exist. (List and Peet, 1939; Wang, 1964; Wilcott et al., 1970). In an investigation of hemispheric influences on habituation of EDOR, Gruzelier et al. (1981) found that slow habituation was associated with larger right hand responses and fast habituation with larger left hand responses, among non-psychiatric subjects. Current views suggest a predominance of contralateral inhibitory influences on response amplitudes (Lacroix and Comper, 1979) and hemisphere specialisation for attentional processes (Dimond, 1979). The Gruzelier et al. results implied that fast habituation is associated with left hemisphere activity dominance and better selective attention, while slow habituation is associated with broadened sustained attention and a predominance of right hemisphere activity. Hypnosis would consequently appear to have complex effects on hemispheric influences in that right hemisphere activity seems to increase, producing larger right hand responses, but some concurrent left frontal activity is assumed to
inhibit responding. Whatever the precise nature of these lateral influences, it seems plausible that hypnotic induction affects attentional processes, perhaps by maximising broadened attention.

The relationship of anxiety to hemispheric influences is not clear, however there is evidence of anomalous laterality in non clinical anxiety (Tucker, 1980). In the Gruzelier et al. (1981) investigation, a minority of the slow habituators showed larger left than right hand responses and these subjects also had higher scores on questionnaires of anxiety. Phelan and Gruzelier (1980) found evidence of right hemisphere processing of verbal material in students only when anxious and the same anomaly characterised anxious patients (Bennett and Gruzelier, 1983). In a test of lateral shifts under hypnosis among normal and clinically anxious subjects, Ronder and Gruzelier (1983) replicated the shift from left to right among susceptible normal subjects in a sorting task. Despite the anxious patients' susceptibility to hypnosis, as evidenced by marked changes in processing time, they did not show the same lateral shift. If anything, the opposite was found, consistent with a model of left hemisphere over arousal following the Yerkes-Dodson law. While normal subjects were apparently under aroused in the left hemisphere during hypnosis, anxious patients appeared to change from over arousal to moderate arousal.

The effects of hypnotic induction and progressive muscle relaxation on habituation to auditory stimuli among clinically anxious patients have not previously been compared, and the question of whether relaxation facilitates habituation has not yet been resolved. This study was therefore designed to test the hypotheses that (a) autohypnosis training would produce faster habituation to tones of no
attentional significance among anxious subjects susceptible to hypnosis, than among unsusceptible subjects or those receiving relaxation training. (b) Any facilitation of habituation by relaxation would be due to a reduction in sensitisation. A dishabituating stimulus was included to test for sensitisation in both condition. For exploratory purposes only, the following tentative hypotheses relating to anxiety and laterality in the two experimental conditions were formulated: if relaxation facilitated habituation it would do so in a lawful manner increasing left over right asymmetries: a possible reversal of asymmetry might be found in subjects remaining slow habituators, that is, they would change to the normal slow habituating pattern of right over left asymmetry, because of a concurrent reduction in anxiety. Hypnosis would be expected to produce the pattern previously found, where larger right hand responses would be accompanied by facilitation of habituation.
Methodological considerations

It was necessary to approximate as closely as possible to the best available format for each of the treatments while keeping similarities between them to a minimum. There is no evidence about the parameters necessary for effective autohypnosis training, but muscle relaxation has been investigated in more detail (e.g. Borkovec and Sides, 1979a). It seems to be more effective if more than one session of live training is given and if some passive focus on relaxation as well as active tensing and releasing is included. The length of the sessions in reported studies varies between 15 and 60 minutes. It was decided to give four sessions with therapist contact, two of which would include taped instruction, and a fifth taped session in the laboratory. The first two sessions would be 30-40 minutes long and the remainder, 20 minutes long. The procedure for muscle relaxation was based on Bernstein and Borkovec's manual (1973). Since there is no well known standardised procedure for autohypnosis, the procedure was based on traditional methods as described in Hartland (1971). Studies of hypnosis often omit details of the procedure used: a full transcript of the training instructions is therefore included in the Appendix (p. 41). It was decided to refer to the two treatments as muscle relaxation and mental relaxation to avoid arousing differential expectations (Reyher and Wilson, 1973). The rationales for the treatments were standardised and designed to be equally plausible (see Appendix (pp. 50,51). Both treatments were modified for the final session because of the need to avoid movements during psychophysiological recording. For the muscle relaxation group, a greater emphasis on passive muscle relaxation was introduced with the taped instruction in
the third treatment session (See Appendix, p. 45). For the auto­hypnosis group, reassessment of hypnotic susceptibility during the final laboratory session was incorporated into the taped instruction. A scale which allows subjects to remain still was used. This was derived from the Stanford Susceptibility Scale (Weitzenhoffer and Hilgard, 1959) and has previously been used in this laboratory. A transcript of the modified autohypnosis instruction and the scale of hypnotic susceptibility are given in the Appendix (p.48 and p. 52 ).

Since individuals vary widely in electrodermal responsivity and habituation rate, a repeated measures design was chosen and, in order to equate the groups for habituation rate and susceptibility to hypnosis, these variables were assessed before allocation to treatments. Groups were also balanced for age and sex.

A control for the effects of repeated measures without treatment was not included for ethical and practical reasons. A control for the effects of listening to a voice in the second session could have been included. However, previous research among non-anxious subjects (Brow and Gruzelier, 1982), had shown that changes in habituation under hypnosis were independent of this effect. Electrodermal responsivity varies with the attentional significance of the stimuli, so instructions to the subjects emphasised the irrelevance of the tones.

The questionnaire measures of anxiety were selected for ease of comparison with other research (State-Trait Inventory, Spielberger et al., 1968) and for assessment of cognitive and somatic dimensions of anxiety which may be related to response to treatments with a cognitive or somatic emphasis (Anxiety Symptom Questionnaire, Turner, 1978).

The experimental design was a repeated measures design with two
between subject factors of Treatment and Susceptibility.

Subjects

Details of the subject sample are given in Table 1. Subjects were recruited from referrals to the Psychology Department at Charing Cross Hospital. Referrals were mainly from other departments within the hospital (Psychiatry, Neurology and Cardiology) and all subjects were out-patients. They were aged between 18 and 65 years, with general anxiety or tension as the primary presenting problem. Eight of the subjects were taking psychotropic medication and were asked to keep its level constant throughout the experimental period. Individuals who were psychotic, brain damaged, alcoholic, or depressed were excluded and subjects had to reach a minimum criterion of electrodermal responsivity for inclusion in the study (see Procedure, p. 17).

Twenty nine subjects were assessed for the study. Five were excluded because they did not meet the electrodermal responsivity criterion. One appeared to be mentally handicapped and was thought unable to follow instructions adequately. One was excluded because her problem (pain) was found to have a physical basis and two subjects did not complete the study. Details of these subjects are given in the Appendix (p. 53).

Subjects were allocated to either Hypnosis or Relaxation treatment groups. Details of allocation and of hypnotic susceptibility and habituation classification are given in the Procedure (p. 17). The Hypnosis group contained four women and six men and the Relaxation group six women and four men. Six members of each group were susceptible and four unsusceptible to hypnosis. Seven members of each group were slow or non-habituators, and three were fast habituators. There were two
left handed individuals in the Relaxation group and one in the Hypnosis group. Five members of the Hypnosis group and three members of the Relaxation group were taking tranquilliser medication.

The mean scores on Trait anxiety were at the 67th percentile (Hypnosis group) and 79th percentile (Relaxation group) for neuropsychiatric patients, the most appropriate comparison group reported by Spielberger et al. (1968).

The mean scores on State anxiety were at the 41st percentile (Hypnosis group) and 48th percentile (Relaxation group) for neuropsychiatric patients.

There were no differences among sources of referral for treatment groups. (Fisher's exact probability tests, all p > 0.2).

Table 1: Summary of Characteristics of Sample by Treatment Group

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Therapists

Therapist A (the author) was a female clinical psychologist aged 40. Therapist B was a male doctor aged 60. Both were experienced in relaxation training with both methods.
Apparatus and Stimuli

Skin conductance was measured bilaterally from placements on the medial phalanges of the first and second fingers of each hand. Silver-silver chloride cup electrodes, 1 cm in diameter, used with Beckman KCL electrolyte, were connected to a Grass model 7D polygraph via a constant voltage skin conductance system, after the design of Lykken and Venables (1971). Subjects sat in a sound attenuated room in which temperature and humidity were kept constant and could be observed through a screen. Bilateral hand temperatures were taken before and after recording. Stimuli were presented through headphones (Koss type 6LC) and produced by a tone generator designed by the Charing Cross Department of Medical Physics. In Session II, taped autohypnosis or muscle relaxation instructions were played on a Sony TKL tape recorder. The tones were mixed with the tapes through a device also built by the Charing Cross Department of Medical Physics, so that tones and voice were heard through the headphones at a fixed volume.

A sequence of 16 tones was presented with a quasi-randomised inter stimulus interval between 20 and 40 secs. The first 14 tones were of 1000 Hz, 72.5 dB and 1 sec duration with a 25 msec rise and fall time. The fifteenth stimulus employed a change of frequency to 690 Hz, while the other stimulus characteristics remained unchanged. The change stimulus was followed by a single presentation of the original stimulus to test for the presence of dishabituation. The orienting response was defined as occurring between 0.8 and 3 secs after tone onset (Martin and Venables, 1979). Non specific fluctuations were defined as occurring outside these intervals. The minimum gain was set at 0.02 μmhos/cm with a minimum amplitude criterion of 1 mm. Habituation was said to occur when there was an absence of responding on three successive trials up to and including tone 14. Dishabituation was said to occur when the
response on tone 16 was larger than that on the previous presentation of the original stimulus.

Procedure

Assessment: Potential subjects were interviewed by Therapist A to assess their suitability for inclusion in the study and to obtain their informed consent. Following this they were assessed for hypnotic susceptibility (by the supervisor of this research), using the Barber Suggestibility Scale (Barber, 1965), presented as a test of mental relaxation. They then completed a modified Oldfield-Humphrey handedness questionnaire, the State-Trait Anxiety Inventory and the Anxiety Symptom Questionnaire. The results of the latter are not reported here. Subjects were taken to the laboratory for recording of electrodermal activity (Laboratory Session I). They were told that we aimed to assess their state of physical relaxation. Headphones would help to screen out sounds in the environment. Occasional tones would be heard but these were of no significance and the subject was to ignore them. Recording took place as soon as the door was closed. Ten minutes of self relaxation preceded the tone sequence to make this laboratory Session I comparable to Session II. Subjects who did not produce at least one elicited skin conductance response or 10 non specific fluctuations were excluded at this stage.

Allocation: Subjects were sequentially allocated to the two treatment groups and therapists on the basis of susceptibility to hypnosis, habituation status, age and sex so that the groups were approximately equated for these variables. Susceptible subjects were defined by a score of 50 per cent or above on the Barber Suggestibility Scale (1965). Habituation status was determined by the number of trials to habituation (TTH) in the non preferred hand, and subjects were classed as slow
(TTH < 7), or fast (TTH ≥ 7) habituators. Allocation was made by the supervisor of this research, and the therapists were blind to the susceptibility and habituation status of the subjects.

**Treatment:** All subjects received four sessions of individual treatment and a fifth session of taped treatment in the laboratory. The sessions were at approximately weekly intervals. In Session 1, a standardised version of the aims of relaxation treatment was given and subjects received a copy to read at home. They were then given standardised instruction. Subjects were asked to accept the instructions passively and avoid thinking about whether they were being successful or not since this would enhance the achievement of a relaxed state. An example of a situation in which each subject felt anxious, and ways in which the relaxation could be helpful in it, was discussed. It was emphasised that early attempts might not be successful and that practice was essential. Subjects were asked to practice every day for 20 minutes and to keep a record. Each subject was given a relaxation diary for this purpose. Sessions 2-4 began with a brief discussion of how the patient had used the relaxation and any problems were dealt with. In Sessions 3 and 4 subjects listened to a taped version of the relaxation made by his therapist. The procedure for the second laboratory session was as in the first except that subjects heard taped versions of treatment mixed with the tones. Subjects in the autohypnosis group were told that the tape was slightly different from their usual session. All subjects were reminded to accept the instructions passively and to relax in the usual way. The anxiety questionnaires were again completed, before the session where possible. Subjects were interviewed by Therapist A, following the laboratory session and either discharged, given a follow-up appointment or offered further treatment.
Statistical Analysis

Trials to habituation, numbers of elicited responses and numbers of non-specific fluctuations are measures which do not meet the assumptions of parametric statistics. Non parametric methods were therefore used, but since there is no test for repeated measures with more than between subject factor, separate Kruskal Wallis analyses of variance were carried out for the data in Session I and II and for changes from Session I to Session II. Multiple comparisons of means were calculated, based on Kruskal Wallis Rank Sums (Hollander and Wolfe, 1973). The distributions of skin conductance levels and response amplitudes were positively skewed, and logarithmic transformations were applied before analyses of variance. A constant was added (log +1), to allow zero amplitude responses to be included in the analyses. Anxiety scores were converted to T-scores to allow parametric analysis. Parametric data were analysed using split-plot ANOVAS and multiple comparisons of means were calculated using the Newman-Keuls test for a posteriori comparisons. Probability level was set at p < 0.05. Although comparisons of means are not strictly appropriate where effects obtained in analyses of variance are of p > 0.05, comparisons were carried out for effects of 0.05 < p > 0.1 to further elucidate the data.

Data Reduction

Therapists: In ANOVAS on State and Trait anxiety with two between subject factors of Treatment and Therapist and one within factor of Session, there was no effect attributable to Therapist (both F ratios < 0.69). In analyses of electrodermal variables by Therapist and Session there were no significant differences for Session I (all H < 1.27) nor for Session II (all H < 2.2).
Electrodermal Activity

In an ANOVA on the mean log +1 response amplitudes with two between factors of Treatment and Susceptibility, and two within factors of Hand and Session, there was no main effect of Hand \((F_{1,16} = 0.97)\), nor any significant interactions of this with the other factors (all F ratios < 1.45). All subsequent analyses on skin conductance responses were performed on data extracted from the non-preferred hand of each subject.

Compliance with treatment: Reported compliance with the instruction to practice daily was similar in both groups. Those in the hypnosis group practiced on 88% and those in the relaxation group on 90% of days. Amount of practice was therefore disregarded in the analysis.

Missing data: One subject failed to complete the questionnaires in Session II. The mean score of the other members of the group (Hypnosis) was entered for this subject.
RESULTS

Electrodermal Activity

Table 2 summarises the data for trials to habituation, numbers of elicited responses and numbers of non specific fluctuations in Sessions I and II. In Session I, there were no differences among the four groups defined by treatment and susceptibility, in trials to habituation, (H = 2.32), number of elicited responses (H = 3.14), or number of non-specific fluctuations (H = 5.62). However, comparisons between Session I and Session II, combining all groups, indicated significant reductions in these three measures (All Wilcoxon T < 32.5).

Analysis of the changes from Session I to Session II showed a significant difference among the groups for trials to habituation (H = 8.01). Multiple comparisons indicated that subjects in the Susceptible Relaxation group showed a significantly larger reduction in trials to habituation in Session II than subjects in the other groups. Differences among the groups for changes in numbers elicited responses from Session I to Session II approached significance (H = 7.24, p < 0.07). Once again, multiple comparisons indicated a significantly larger reduction in numbers of elicited responses in the Susceptible Relaxation group. Since there were no differences among the groups for changes in numbers of non specific fluctuations (H = 1.55), these differences apparently indicated a treatment effect, independent of general responsivity, among subjects in the Susceptible Relaxation group. However, such an interpretation is qualified by inspection of the data in Session I in which it is clear that members of this group produced lower numbers of non specific fluctuations. This was not revealed by statistical analysis, presumably because of the large variability on this measure and the small size of the groups. Analysis of non specific fluctuations in Session II
Table 2: Trials to Habituation (TTH), Numbers of elicited responses (EDOR), and numbers of nonspecific fluctuations (NSF) in Laboratory Sessions I and II by treatment group and hypnotic susceptibility

<table>
<thead>
<tr>
<th>TREATMENT</th>
<th>SESSION I</th>
<th></th>
<th>SESSION II</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TTH</td>
<td>No. EDOR</td>
<td>No. NSF</td>
<td>TTH</td>
</tr>
<tr>
<td></td>
<td>Median SIQR</td>
<td>Median SIQR</td>
<td>Median SIQR</td>
<td>Median SIQR</td>
</tr>
<tr>
<td>HYPNOSIS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Susceptible</td>
<td>14 4.5</td>
<td>10.5 2.5</td>
<td>42.5 24</td>
<td>7.5 4.5</td>
</tr>
<tr>
<td>Unsusceptible</td>
<td>14 3.5</td>
<td>11.5 3.8</td>
<td>55. 2.25</td>
<td>12.5 4.0</td>
</tr>
<tr>
<td>Combined</td>
<td>14 4.5</td>
<td>10.5 3.0</td>
<td>46.5 29</td>
<td>11 6.0</td>
</tr>
<tr>
<td>RELAXATION</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Susceptible</td>
<td>10 1.5</td>
<td>7.5 1.5</td>
<td>3 5.5</td>
<td>0 0</td>
</tr>
<tr>
<td>Unsusceptible</td>
<td>14 2.5</td>
<td>12.0 1.8</td>
<td>24.5 15.0</td>
<td>14 7.5</td>
</tr>
<tr>
<td>Combined</td>
<td>10 3.5</td>
<td>8.5 2.5</td>
<td>14 9.0</td>
<td>0 7.5</td>
</tr>
</tbody>
</table>

SIQR = semi interquartile range.
indicated a significant difference among the groups \( (H = 7.97) \). Multiple comparisons again showed that subjects in the Susceptible Relaxation group produced significantly fewer non specific fluctuations than subjects in both Hypnosis groups. The comparison with the Unsusceptible Relaxation group fell short of significance, but the difference was consistent with the data pertaining to elicited responses. Subjects in the latter group also gave significantly fewer non specific fluctuations than Unsusceptible subjects in the Hypnosis group. Since trials to habituation and numbers of elicited responses were significantly correlated with numbers of non specific fluctuations in both Session \( (\rho = 0.82 \text{ and } 0.81) \) and Session II \( (\rho = 0.80 \text{ and } 0.85) \), it seems that the impression of a treatment effect is unfounded. The differences reflect allocation, which took account of phasic responses but not tonic measures.

Table 3 summarises data for response amplitudes and skin conductance levels in Session I and II. In an ANOVA on mean log +1 response amplitudes with two between subject factors of Treatment and Susceptibility and one within subject factor of Session, there was a significant main effect of Session \( (F(1,16) = 7.32) \) which was due to the lower amplitudes in Session II \( (\bar{x} = 0.19, \ SD = 0.18; \ \text{Session II } \bar{x} = 0.10, \ SD = 0.14) \). There was neither a main effect of Treatment \( (F(1,16) = 2.92) \) nor any interaction of this with Session, Susceptibility, or both (all F ratios < 1.30).

In an ANOVA on log skin conductance levels with two between factors of Treatment and Susceptibility and three within factors of Hand, Initial/Final Level and Session, there was a significant main effect of treatment \( (F(1,16) = 11.53) \) and an unreliable interaction of
### Table 3: Skin conductance response amplitudes and levels in Session I and II by treatment group and hypnotic susceptibility

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Susceptible</th>
<th>Unsusceptible</th>
<th>Susceptible</th>
<th>Unsusceptible</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean Log $+1$ Skin conductance response amplitude</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Session I</td>
<td>Session II</td>
<td>Session I</td>
<td>Session II</td>
</tr>
<tr>
<td></td>
<td>$\bar{x}$</td>
<td>SD</td>
<td>$\bar{x}$</td>
<td>SD</td>
</tr>
<tr>
<td><strong>HYPNOSIS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Susceptible</td>
<td>0.26</td>
<td>0.25</td>
<td>0.21</td>
<td>0.20</td>
</tr>
<tr>
<td>Unsusceptible</td>
<td>0.20</td>
<td>0.13</td>
<td>0.13</td>
<td>0.12</td>
</tr>
<tr>
<td><strong>RELAXATION</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Susceptible</td>
<td>0.15</td>
<td>0.19</td>
<td>0.003</td>
<td>0.008</td>
</tr>
<tr>
<td>Unsusceptible</td>
<td>0.17</td>
<td>0.10</td>
<td>0.03</td>
<td>0.024</td>
</tr>
</tbody>
</table>

| Log skin conductance levels | | | |
| Session I | Initial | Final | Session II | Initial | Final |
| $\bar{x}$ | SD | $\bar{x}$ | SD | $\bar{x}$ | SD | $\bar{x}$ | SD |
| **HYPNOSIS** | | | | |
| Susceptible | 1.74 | 0.57 | 1.68 | 0.72 | 1.62 | 0.67 | 1.55 | 0.64 |
| Unsusceptible | 1.70 | 0.64 | 1.65 | 0.70 | 1.58 | 0.28 | 1.50 | 0.39 |
| **RELAXATION** | | | | |
| Susceptible | 0.55 | 0.33 | 0.50 | 0.22 | 0.59 | 0.40 | 0.36 | 0.27 |
| Unsusceptible | 1.43 | 0.41 | 1.40 | 0.34 | 1.26 | 0.15 | 1.04 | 0.08 |

Treatment and Susceptibility ($F(1,16) = 3.63$, $p < 0.08$). Comparisons of means indicated that, consistent with the measures of non specific activity, this was due to the lower levels throughout of subjects in the Susceptible Relaxation group (Table 3). There was no interaction of either Treatment or Susceptibility with Session (both $F$ ratios $< 0.63$) suggesting that the effects again reflected allocation policy.
There was a significant main effect of Initial/Final level ($F(1,16) = 21.2$) which interacted with Session ($F(1,16) = 4.79$). Further analysis indicated that Final levels in Session II were lower than all others (Table 3). A significant main effect of Hand ($F(1,16) = 5.39$) was due to higher levels in the preferred hand and did not interact with any of the other factors (Preferred hand $\bar{x} = 1.25$, $SD = 0.68$, Non-Preferred hand $\bar{x} = 1.19$, $SD = 0.68$).

**Dishabitation**

In Session I, ten of the twenty subjects gave a larger response to the change stimulus and seven showed dishabituation on the following trial. In Session II only five subjects gave a larger response on Trial 15 and six showed dishabituation. In an ANOVA of log +1 response amplitudes for trials 14, 15 and 16 with two between subject factors of Treatment and Susceptibility and two within subject factors of Session and Trial number there was a main effect of Session ($F(1,16) = 14.64$) due to the lower amplitudes in Session II (Session I $\bar{x} = 0.13$, $SD = 0.23$; Session II $\bar{x} = 0.05$, $SD = 0.17$). There were no other significant main effects nor any significant interactions among the factors (all $F$ ratios $< 1.76$). Thus there was no main effect of dishabituation in either session.

**Anxiety Ratings** (Table 4)

In an ANOVA on State anxiety with two between factors of Treatment and Susceptibility and one within factor of Session there were no significant main effects or interactions among the factors (all $F$ ratios $< 1.23$).

In a similar ANOVA on Trait anxiety there was a significant main effect of Session ($F(1,16) = 13.03$) such that Trait anxiety was rated
Table 4: State and Trait anxiety (T-scores) in Session I and II by treatment group and hypnotic susceptibility

<table>
<thead>
<tr>
<th></th>
<th>State</th>
<th></th>
<th>Trait</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Session I</td>
<td>Session II</td>
<td></td>
<td>Session I</td>
</tr>
<tr>
<td></td>
<td>X</td>
<td>SD</td>
<td>X</td>
<td>SD</td>
</tr>
<tr>
<td>HYPNOSIS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Susceptible</td>
<td>49.6</td>
<td>8.3</td>
<td>47.0</td>
<td>10.7</td>
</tr>
<tr>
<td>Unsusceptible</td>
<td>46.5</td>
<td>8.5</td>
<td>48.5</td>
<td>7.9</td>
</tr>
<tr>
<td>RELAXATION</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Susceptible</td>
<td>48.3</td>
<td>6.4</td>
<td>41.8</td>
<td>5.6</td>
</tr>
<tr>
<td>Unsusceptible</td>
<td>52.8</td>
<td>8.8</td>
<td>43.8</td>
<td>16.7</td>
</tr>
</tbody>
</table>

higher in Session I than in Session II. However, there was a marginally significant interaction of Session with Treatment (F(1,16) = 4.15, p < 0.06). Further investigation of this indicated that subjects in the Relaxation group gave higher self-ratings of Trait anxiety in Session I (X = 58.0, SD = 7.3) than did subjects in the Hypnosis group I (X = 54, SD = 6.4) and than either Treatment group in Session II (Hypnosis X = 51.3, SD = 7.3; Relaxation X = 50.9, SD = 6.1).

There was no correlation of either State or Trait anxiety with trials to habituation, response amplitudes, skin conductance levels or non specific fluctuations in either Session I or II (all rho < 4.0). There was therefore no relationship of self-rated anxiety to either tonic or phasic electrodermal measures.

Hypnotic Susceptibility

The correlation between the two measures of susceptibility among subjects in the Hypnosis group fell short of significance (rho = 0.41, p < 0.1 (1 tailed)). This was largely due to one subject whose score on the Barber Suggestibility Scale before Session I was much higher.
than that achieved in Session II. The adequacy of the Barber measure was confirmed by showing that no subject was reclassified when an average of the two measures of susceptibility was calculated.

Treatment outcome

Although treatment outcome was not a planned dependent variable, since follow-up data were available, it is included for completeness.

Thirteen subjects had further treatment: seven in the Hypnosis group (\( \bar{x} \) Number of sessions = 3.3) and six in the Relaxation group (\( \bar{x} \) Number of sessions = 4). There was some suggestion that Susceptibility was a factor in outcome since only half of the susceptible subjects required more treatment compared with seven out of eight unsusceptible subjects. However, there were no significant differences among the groups defined by treatment, susceptibility or susceptibility within treatment (Fisher's exact probability tests all \( p > 0.2 \)). There was no relationship between trials to habituation or between self rated-anxiety in Sessions I or II and treatment outcome (all \( U > 36 \)).
DISCUSSION

Trials to habituation, numbers of elicited responses and non-specific fluctuations to auditory tones were significantly reduced among subjects treated with muscle relaxation and autohypnosis compared with pre-treatment measures. This implies an effect of treatment although in order to verify this, a no-treatment control group would be required. Both treatments also produced lower response amplitudes and skin conductance levels were reduced at the end of the final session. However, no differences were found among the groups defined by treatment and susceptibility with the exception of the susceptible relaxation group. This group showed significantly greater reductions in phasic measures of electrodermal responsivity. Despite the fact that the allocation measure of trials to habituation revealed no significant differences among the groups before treatment, the treatment effect found in this group appears to be artefactual. Tonic measures of electrodermal activity indicated a lower level of responsivity among individuals in this group before treatment. The relationship between tonic and phasic measures during habituation is debatable. Martin and Rust (1976) however, in their investigation of intercorrelations among fifteen skin conductance variables during habituation, suggest that there is no completely independent measure of change which can be attributed to habituation, as distinct from reactivity. They consider that interpretation of measures of habituation should take account of individual differences in responsivity. In this study, one pre-treatment measure, the main dependent variable, was not sufficient to preempt the problem of individual differences in reactivity. This suggests that the results of many studies in which comparisons are made between groups which are
not previously equated for any electrodermal variable (e.g. the work of Ohman and his associates (1971 onwards) should be viewed with caution.

Within the hypnosis group, susceptibility did not distinguish between subjects on any electrodermal measure. Although the results of this study are not strictly comparable with previous work in this laboratory, where subjects received a single session of autohypnosis, anxious patients did not show the same effects of susceptibility to hypnosis as normal subjects.

The low tone presented in Trial 15 failed to elicit a larger response in either session and there was no evidence of dishabituation to the subsequent presentation of the original stimulus. This failure to produce dishabituation has been observed among normal subjects (Connolly and Frith, 1978) and among clinical populations, including anxious patients (Frith et al., 1982). In the latter study, increases in responding to the change stimulus were found in all subject groups. These effects appear to depend upon the particular stimulus parameters used and in the present experiment, an insufficient number of subjects responded to the relevant stimuli to allow any conclusions to be drawn regarding sensitisation.

There were no significant laterality effects. In the first session among the fourteen slow habituators, only six showed the asymmetry previously found among non clinically anxious subjects. In the Hypnosis group, five subjects showed reversals in asymmetry in Session II. Four of these changed to larger responses in the left hand, which is the opposite shift to that found by Brow and Gruzelier (1982). Among subjects in the relaxation group, five showed the asymmetries associated with faster habituation in Session II. No conclusions regarding laterality can be drawn but there was very tentative support
for the proposition that relaxation influences habituation in a lawful manner. Subjects in the hypnosis group appeared, if anything, to change in a manner opposite to the prediction.

Subjects in the relaxation group gave higher self-ratings of Trait anxiety before treatment than did those allocated to hypnosis and consequently showed the greatest decrease in Session II. Although Trait anxiety is generally thought to reflect a relatively stable individual characteristic, similar reductions have been reported among anxious patients after relaxation treatment (Turner, 1978). The lack of change in self-ratings of State anxiety is surprising since this measure would be expected to be more sensitive to the effects of treatment (Stoudemire, 1975). It may be that, although subjects were feeling less anxious in general, they attributed an evaluative aspect to the second laboratory session and increased their self-ratings on that occasion. The lack of a relationship between anxiety ratings and measures of electrodermal activity is at variance with previous findings reviewed by Lader (1975). Self reported anxiety was found to be associated with increased tonic levels of electrodermal activity. It is possible that ceiling effects in the self-ratings in this study obscured any differences.

Evaluation of treatment outcome was not the object of this study, however it is interesting to note that seven subjects required no other treatment and a further three needed only one follow-up session. In view of the fact that relaxation is rarely given as sole treatment, this suggests that clinical psychologists may sometimes be giving more elaborate treatment to anxious patients than is necessary. This possibility was also implied by the results of Hutchings et al. (1980). Unfortunately, the ten subjects who required more than one extra
treatment session did not seem to be distinguishable in any obvious way from those that did not.

In summary, no conclusions regarding differences between autohypnosis and muscle relaxation in their effects on habituation of electrodermal orienting responses can be drawn from this study. The implications for research using psychophysiological measures seem to be that groups to be compared must be equated for tonic as well as phasic levels of activity before the introduction of the independent variable.

Acknowledgements

I should like to thank John Gruzelier who supervised this research and made his laboratory available to me. Thanks are due to Sara Turner who provided clinical supervision and generously allowed me to base many details of procedure directly on those in her MSc dissertation. James Kennedy gave many hours help in the laboratory and Frank Eves gave statistical advice. Thanks are also due to Martin Thomas who was Therapist B and drafted the autohypnosis training, and to Tricia Carroll who typed the dissertation.
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