Nutrition and HIV infection

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Nutritional status may have an impact at all stages of HIV disease. Many of the clinical features of HIV infection cause nutritional problems and may also be exacerbated by the presence of malnutrition. Inadequate food intake, due to a wide variety of antiligies, malabsorption and altered metabolism, may all contribute to malnutrition. Additionally, factors in food, including micronutrients, can modulate immune function. Reduced micronutrient levels are documented at all stages of HIV infection although the significance of these findings and how they may relate to HIV disease severity and prognosis are still unclear. Body composition changes in adults include loss of weight with proportionately greater loss of lean mass. Paediatric HIV infection has received far less research attention, but growth failure is a significant nutritional complication seen clinically. Clinical experience suggests that early nutritional intervention may improve prognosis as well as quality of life. Nutritional management in HIV disease depends on the clinical state of the patient. Definition of the benefits of particular food factors and diets, as well as the most appropriate nutrition support modalities, would allow rational nutritional counselling. Better definition of the contribution food makes to health through its social role, and the opportunities this provides in patient care, would complement the biomedical research effort.

Introduction

Nutritional abnormalities are a frequent and characteristic feature of infection with human immunodeficiency virus (HIV) and represent a major determinant of survival.2 Nutritional status may have an impact at all stages of HIV disease from initial acquisition of infection, clinical manifestations, progression of the disease and palliation of advanced disease. Factors such as dietary intake, metabolic changes and absorptive capacity may all contribute to nutritional problems, as can factors such as psychological state, social supports and ethnicity.

HIV infection: an overview

HIV is a retrovirus that targets cell-mediated immunity by destroying a subset of T-lymphocytes with a CD4 molecule on the cell surface. HIV is transmitted by contact with infected body fluids which may occur by unprotected sexual contact, sharing contaminated injecting equipment, transfusion of contaminated blood or blood products or occupational exposure such as a needle-stick injury. Vertical transmission may occur from mother to child during pregnancy, childbirth or breast-feeding.4 Following infection, progressive decline in immune function leads to the development of the manifestations of acquired immune deficiency syndrome (AIDS). The Centers for Disease Control5 have defined a staging system for the classification of HIV infection in adults (Table 1).

Table 1. Classification of HIV infection*

| Group I | Acute infection |
| Group II | Asymptomatic |
| Group III | Persistent generalized lymphadenopathy |
| Group IV | Subgroup A – constitutional disease |
| Subgroup B – neurological disease |
| Subgroup C – secondary infectious diseases |
| Subgroup D – secondary cancers |
| Subgroup E – other conditions |

Source: Centers for Disease Control, USA 1987
*Group IVA is also referred to as AIDS-Related Complex (ARC) and Group IVB, C, D, E may be referred to as Acquired Immune Deficiency Syndrome (AIDS).

It is currently unclear how many people infected with HIV will eventually develop AIDS. The San Francisco Clinic Cohort Study suggested that 48% of those infected with HIV would develop AIDS within 10 years.6 Since that study was published, the widespread use of anti-retroviral medications and more effective prophylactic regimes has probably lengthened that time span considerably.7 Cofactors that influence the progression of asymptomatic HIV infection to development of AIDS are poorly understood. Cofactors that have been suggested include co-infection with cytomegalovirus or other sexually transmitted diseases (particularly ulcer...

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Pathogenesis of wasting
The mechanisms that cause wasting in HIV infection are complex. They include: inadequate intake (due to anorexia, pain, other many factors), altered metabolism (increased energy use or a maladaptive response to hypocaloric state), and malabsorption (failure to absorb ingested nutrients).

Inadequate intake
Anorexia (reduced desire to eat) is an important factor in weight loss and is a common symptom in HIV infection. When actual food intake is measured, there are conflicting reports of the adequacy of nutrient intake. Other factors which may be involved in reduction in food intake include:

Gastrointestinal manifestations. Infection or malignancy in HIV infection can affect any organ in the gastrointestinal tract. Oral and oesophageal lesions (Candida sp; Herpes simplex; Cytomegalovirus, Kapous’s sarcoma; lymphoma, aphthous ulceration) may cause pain, haemorrhage or obstructive lesions. Diarrhoea is a common gastrointestinal symptom in AIDS, reported in up to 90% of all AIDS patients. Fear of exacerbation of symptoms is commonly cited as a cause for decreased food intake in those with diarrhea.

Pharmacotherapy. Most of the medications used in treatment of opportunistic infections can affect food intake by causing nausea, anorexia and taste changes as well as causing side-effects, such as sedation or paresthesiae, which can adversely affect nutritional status. (Table 2).

Psychosocial effects. Depression and anxiety can adversely affect food intake in individuals who are worried financial resources for those unable to work. Illness may affect the physical ability to prepare and purchase food and lack of social support may further exacerbate these problems.

Table 2. Drugs with side effects adversely affecting nutritional status

<table>
<thead>
<tr>
<th>Side Effects</th>
<th>Mug (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal pain</td>
<td>Oral (mg)</td>
</tr>
<tr>
<td>Nausea</td>
<td>Oral (mg)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Oral (mg)</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>Oral (mg)</td>
</tr>
<tr>
<td>Taste alterations</td>
<td>Oral (mg)</td>
</tr>
</tbody>
</table>

(Adapted from: Bowden, Wang)

The social support systems of an HIV-infected individual are often fragmented, with many potential supporters feeling isolated from themselves. They may themselves undermine the practical support that can be provided.

Central nervous system manifestations of AIDS. Pathologic abnormalities in the CNS have been reported in 75-90% of patients who died of AIDS,23,24 Cognitive dysfunction is also common. Immune deficiency may contribute to infections of the CNS and malignancies causing specific neurologic dysfunction. Direct involvement of the brain with HIV can result in AIDS dementia complex, typically causing problems of psychomotor slowing, poor concentration, forgetfulness and social withdrawal. All of these symptoms may undermine the ability of the person to maintain adequate nutrition.

Micronutrient deficiencies such as thiamin or vitamin B12 may conceivably contribute to cognitive dysfunction in HIV-infected persons. These may represent treatable causes of so-called dementia, though no research has been done in this area.

The role of cytokines. The cytokines interleukin-1 (IL-1) and tumour necrosis factor (TNF) have been shown to be anorexigenic in rats. Elevated levels of TNF have also been measured in patients with AIDS,25,26 but no relationship between the degree of weight loss and the levels of circulating TNF was found. The elevations in TNF may have been related to intercurrent infections: subsequent studies have failed to demonstrate elevated TNF in AIDS25,26.

Intriguingly, it has been suggested that not only is cytokine release altered in HIV-infected persons that TNF and possibly other cytokines can themselves activate HIV replication. Cytokines may thus occupy a central role not only in the pathogenesis of AIDS, but also in the wasting and inflammatory response often seen in HIV disease.

Altered metabolism
Hypermetabolism (raised basal energy expenditure – BEE) is commonly found in the cachexia of fever or malignancy. As febrile illnesses and malignancies are common in infection with HIV, an elevation in BEE would be expected in people with this disease. Even in the absence of sepsis or malabsorption, studies have demonstrated elevated BEE in both asymptomatic and advanced HIV infection.27,28 An undetermined role for cytokines in this phenomenon has been suggested.

Grunfeld’s group2 postulated that some of the elevation in BEE could be explained by undetected opportunistic infections, but as elevated BEE was not present in asymptomatic subjects, they suggested that some of the BEE elevation could be related to the underlying HIV infection itself. In addition, it is clear that the hormonotopic adaptations to energy deficiency states observed in normal subjects, there is a maladaptive response seen in infection and inflammation. Responses such as inhibition of ketosis, increased pyruvate oxidation, and failure to decrease the percentage of energy expenditure represented by protein oxidation, have been observed in septic catabolic states. In HIV infection, alterations in lipid metabolism have been observed. No correlations were found between elevated triglyceride levels and body cell weight. Severe weight loss changes appear to contribute to hypertriglyceridemia seen in HIV infection. Firstly, there is an increase in de novo hepatic lipogenesis and secondary hypertriglyceridemia is reduced due to inhibition of lipoprotein lipase activity. These changes in lipid metabolism probably only account for a small amount of inappropriate energy use and do not explain the whole impact on HIV infection.

Notwithstanding the studies reviewed here, the role of hypermetabolism remains poorly understood. Many HIV-infected individuals are able to maintain constant body weight for long periods of time with significant increases in food intake. Clearly in some patients an adaptation occurs compensating for metabolic changes. The mechanism whereby this can occur still need to be explored.

Adrenal insufficiency is an uncommon, but treatable cause of weight loss in AIDS patients. It has mostly been described as a consequence of cytomegalovirus infection of the adenals.

Malabsorption
Malabsorption is common in HIV infection. Aetiology includes infections, malignancy, pancreatic disease and pharmacotherapy.

Infectious causes. HIV has been demonstrated in the small and large intestinal mucosa of patients with AIDS.29,30 Detection of the virus does not necessarily imply a pathogenic role: AIDS enteroenteropathy remains a diagnosis of exclusion. Elevated diosamine (D) has been decreased acid secretion has been reported in AIDS. Its cause is not clear, but it has been speculated that the resultant hypochlorhydria could contribute to the entry of enteropathogenic organisms through the gastric mucosa.32 Impaired absorptive function can occur in HIV-infected patients, even when no pathogens are detected. At a morphological level, partial villous atrophy has been reported.33 Whether this is due to HIV infection itself, or to an as yet unidentified pathogen, is still unclear. Involvement of the intestinal autonomic nervous system by HIV may also contribute to gastrointestinal dysfunction by impairing motility and absorption.

Many pathogens causing diarrhoea and malabsorption have been isolated in HIV-infected patients.33 (Table 3). Chronic syndromes produced by these infections include diarrhoea, colitis and specific malabsorption syndromes. Any infectious agent that causes diarrhoea is also capable of producing malabsorption of both macro and micronutrients.

Pathogens

Virus

Cytopneumovirus (CMV)
Herpes simplex virus (HSV)

Bacteria

Salmonella sp.
Shigella sp.
Campylobacter sp.
Chlamydia trachomatis
Vibrio parahaemolyticus

Protozoa

Cryptosporidium sp.
Microsporidium sp.
Giardia lamblia
Entamoeba histolytica
Isospora bell

No infective agent found

AIDS enteropathy

(Adapted from Simon et al,46 Keusch GT & Farthing MIO)

Non-infectious causes: malignancy. Kaposis’s sarcoma (KS) lesions have been detected throughout the gastrointestinal tract. Gastrointestinal KS is most likely to cause symptoms when it occurs in the oropharynx and oesophagus. Occasionally, the tumour has been associated with a protein-losing enteropathy in the lower bowel, as well as massive haemorrhage and bowel obstruction.35,36 Non-Kaposis’s lymphoma occurs in the intestinal tract of HIV-infected patients, but is far less common than KS in homosexual men. The tumours are almost all of B-cell origin.37 Clinical symptoms include diarrhoea, haemorrhage, pain and intestinal obstruction.

Pancratic insufficiency resulting in malabsorption has occasionally been described in HIV infection. It may result from either chronic pancreatitis unrelated to HIV infection, opportunistic infection of the pancreas, or being drug-induced. Pancreatitis is a recognized adverse effect of diosamine (D).32 Increased duodenal antibiotic use may result in bacterial overgrowth in the gut. Infections with bacteria such as Clostridium difficile are well documented in this context and may cause malabsorption.
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NUTRITION AND HIV INFECTION

The interaction between nutritional status and increased disease susceptibility is well known.22 Many of the immunological abnormalities seen in HIV infection are very similar to those seen in people with disease and in single nutrient deficiencies.33,34 An interaction between nutrient deficiencies and HIV-induced immune deficiency has been proposed.25 Reduced mucosal defences secondary to zinc deficiency has been suggested as one way in which malnutrition may interact with HIV acquisition.35 There has been no formal study of such hypotheses to date.

Pathogenesis of wasting

The mechanisms that cause wasting in HIV infection are complex. They include: inadequate intake (due to anorexia, pain, or other many factors), altered metabolism (increased catabolism or a maladaptive response to hypocaloric state), and malabsorption (failure to absorb ingested nutrients).

Inadequate intake

Anorexia (reduced desire to eat) is an important factor in weight loss and is a common symptom in HIV infection. When assessment of food intake is measured, there are conflicting reports of the adequacy of nutrient intake.36-39 Other factors which may be involved in reduction in food intake include:

Gastrointestinal manifestations. Infection or malignancy in HIV infection can affect any organ in the gastrointestinal tract. Oral and oesophageal lesions (Candida sp; Herpes simplex; Cytomegalovirus, Kapoos’s sarcoma; lymphoma, aphthous ulceration) may cause pain, harm to the absorptive state.26 Diarrhoea is a common gastrointestinal symptom in AIDS, reported in up to 90% of all AIDS patients.27 Fear of exacerbation of symptoms is commonly cited as a cause for decreased food intake in those with diarrhoea.

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Altered metabolism

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In contrast to the hypermetabolic adaptations to energy deficiency states observed in normal subjects, there is a maladaptive response seen in infection and inflammation.34 Responses such as inhibition of ketosis, increased pyruvate oxidation, and failure to decrease the percentage of energy expenditure represented by protein oxidation, have been observed in septic catastopic states. In HIV infection, alterations in lipid metabolism have been observed.40 No correlations were found between elevated triglyceride levels and body cell wasting. Several metabolic changes appear to contribute to hyperlipidermicemia seen in HIV infection. Firstly, there is an increase in de novo hepatic lipogenesis and secondary to hypertriglyceridaemia of triglycerides in HIV infection. Secondly, there is an increase in de novo hepatic lipogenesis and secondary to hypertriglyceridaemia of triglycerides in HIV infection.40-42 These changes in lipid metabolism probably only account for a small amount of inappropriate energy use and do not fully explain the result in protein wasting.40

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Malabsorption

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Infectious causes. HIV has been demonstrated in the small and large intestinal mucosa of patients with AIDS.9-15 Detection of the virus does not necessarily imply a pathogenic role: AIDS enteropathy remains a diagnosis of exclusion.17 Cytokine release altering the activity of intestinal mucosa enzymes, such as dipeptidyl peptidase (DPP) and possibly other cytokines can themselves activate HIV replication.37 Cytokines may thus occupy a central role not only in the pathogenesis of AIDS, but also in the through the gastric mucosa.20 Impaired absorptive function can occur in HIV-infected patients, even when no pathogens are detected. At a morphological level, partial villous atrophy has been reported.20-22 Whether this is due to HIV infection itself, or to an as yet unidentified pathogen, is still unclear. Involvement of the intestinal autonomic nervous system by HIV may also contribute to gastrointestinal dysfunction by impairing motility and absorption.18,19 Many pathogens causing diarrhoea and malabsorption have been isolated in HIV-infected patients.19,23 (Table 3). Clinical syndromes produced by these infections include diarrhoea, colitis and specific malabsorption syndromes. Any infectious agent that causes diarrhoea is also capable of producing malabsorption of both macro- and microminerals.

Table 3. Enteric pathogens causing diarrhoea and malabsorption in HIV infection.

Pathogen

Virus

Cytomegalovirus (CMV)
Herpes simplex virus (HSV)
Bacteria
Salmonella sp.
Shigella sp.
Campylobacter sp.
Chlamydia trachomatis
Vibrio parahemolyticus
Clostridium difficile
Protozoa
Cryptosporidium sp.
Microsporidium sp.
Giardia lamblia
Encephalitis histolytica
Isospora belli

No infective agent found

AIDS enteropathy

(Adapted from Simon et al.48, Keusch & Farthing MJO20)

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(Adapted from Simon et al.48, Keusch & Farthing MJO20)
Anthropological aspects of nutrition

Nutritional status has many determinants. The pathological manifestations of an illness such as HIV infection, are but one of these. Sociocultural aspects have profound influences on food intake, behaviour and beliefs, and hence nutritional status, in both health and illness. Epidemiological studies have revealed that particular systems of food and health beliefs and religious beliefs may also determine food intake. Economic status is important in determining food choice. Poverty affects the ability to purchase food as well as the type of food purchased.

Nutritional education may be lacking in lower socio-economic groups. There are good correllates between the prevalence of diseases such as obesity and socio-economic and educational status4. Food economics and politics may be the main factors in the development of malnutrition, particularly in developing countries. Indigenous peoples in developed nations may not only deal with poor economic conditions, but also face a multitude of other factors such as dispossession, poor sanitary conditions and lack of education. Low social activity has been shown to be strongly predictive for premature death. It has been postulated that as many social activities in our society revolve around eating, it is the meal that determines the outcome5. Certainly, social isolation, such as the single parent or single homosexual may experience, may influence food intake behaviour. Skills to food preparation are an important determinant of nutritional status. Male gender is still an important contributor to lack of food preparation skills in our society6. This may also apply to many individuals in the gay community.

HIV disease is largely a disease of young people, and the nutritional peculiarities peculiar to young people will influence nutritional status and response to interventions. Young people may be particularly concerned with maintaining a culturally desirable body image. Loss of the body image (imagined) can be a major source of anxiety to HIV positive individuals as well as adversely affecting efforts to increase body weight8. Children's food choices have many determinants. Young people use food for many purposes other than sustenance, such as to underline their own identity as children and to defy the adult world9. This may be particularly true for the child who is chronically ill and has reduced opportunity to assert themselves in other areas. Recreational drugs and alcohol may influence nutritional status in the opposite direction. The infection or organ failure associated with abuse of these substances can lead to adverse nutritional consequences. In addition, the intoxicated person is unlikely to be capable of maintaining a nutritional diet being neither able to prepare food nor to eat it.

Nutritional consequences of HIV infection

Changes in body composition
The body composition changes associated with the wasting of HIV infection have been reported in several papers, most of which have only been documented in HIV infection33,34,35. There have been several more sophisticated descriptions of body composition changes in HIV infection. Kotler et al studied a group of male and female patients with AIDS. Depletion of body cell mass, derived from measurement of total body potassium (TBK), was present in all subjects. Body fat content, measured by anthropometry (skin-fold thickness), was also diminished although the value of percentage body fat in both body fat was significantly different between males and females. The data obtained in healthy male homosexuals. Depletion of relative fat mass was more marked in the small group of patients with disease. The pattern of body composition changes seen in the males resembled the wasting seen in sepsis or injury, the pattern in women, where fat depletion was greater than loss of lean body mass, resembled the pattern of wasting seen in starvation. The women studied suffered from reduced food intake due to severe upper gastrointestinal candidiasis, but had fewer sexual activities than the males. Their body composition changes thus appeared to reflect starvation rather than changes inherent to infection with HIV.

Kotler et al subsequently studied the relationship of depletion of body cell mass and survival time. They found that whilst reduction in body weight paralleled body cell mass depletion, the regression line for TBK/height was a better predictor of death. The calculated body fat content showed no correlation with survival time and varied considerably in individual patients.

Table 4. Summary of body composition changes.

<table>
<thead>
<tr>
<th>Category</th>
<th>Definition</th>
<th>Change in HIV infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass</td>
<td>Weight loss</td>
<td>LBM reduced</td>
</tr>
<tr>
<td></td>
<td>Relative fat mass reduced</td>
<td></td>
</tr>
<tr>
<td></td>
<td>AIDS</td>
<td>LBM reduced (proportionally &gt; simple starvation)</td>
</tr>
<tr>
<td></td>
<td>Relative fat mass reduced</td>
<td>Increased ECF and reduced ICW; Overall decreased total body water</td>
</tr>
</tbody>
</table>

*The term ‘asymptomatic’ is used here to imply ‘otherwise asymptomatic’, as weight loss can be a symptom itself.

Table 5. Altered micronutrient levels in HIV infection.

<table>
<thead>
<tr>
<th>Micronutrient</th>
<th>Commentary</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zinc</td>
<td>-70% body stores when Alb. ↓ in AIDS</td>
<td>Palta et al 1986</td>
</tr>
<tr>
<td></td>
<td>-70% body stores with whole body stores and wide variation</td>
<td>Malcolm et al 1992</td>
</tr>
<tr>
<td></td>
<td>Asymptomatic: serum Zn</td>
<td>Moraglia et al 1988</td>
</tr>
<tr>
<td></td>
<td>Serum Zn</td>
<td>Beach et al 1972</td>
</tr>
<tr>
<td></td>
<td>Serum Zn</td>
<td>Graham et al 1972</td>
</tr>
<tr>
<td>Selenium</td>
<td>50% serum and very low</td>
<td>Dwek et al 1989</td>
</tr>
<tr>
<td></td>
<td>Serum selenium</td>
<td>Dwek et al 1989</td>
</tr>
<tr>
<td></td>
<td>Serum and very low</td>
<td>Harris &amp; Calendine 1981</td>
</tr>
<tr>
<td></td>
<td>Serum and very low</td>
<td>Herbert 1983</td>
</tr>
<tr>
<td></td>
<td>Serum and very low</td>
<td>Mantor-Aieta 1986</td>
</tr>
<tr>
<td></td>
<td>Serum and very low</td>
<td>Beach et al 1972</td>
</tr>
<tr>
<td></td>
<td>Serum and very low</td>
<td>Bogden et al 1990</td>
</tr>
<tr>
<td></td>
<td>Serum and very low</td>
<td>Baum et al 1994</td>
</tr>
<tr>
<td></td>
<td>Serum and very low</td>
<td>Kartter et al 1998</td>
</tr>
<tr>
<td></td>
<td>Serum and very low</td>
<td>Bogden et al 1990</td>
</tr>
</tbody>
</table>

(Adapted from Hyde & Oliver16)

Changes in micronutrient status
Micronutrients are important for maintaining normal immune function. Reduced levels of many micronutrients have been documented in HIV infection. These include zinc, iron, copper, selenium, B6, B12, folic acid and many others (see Table 5).

The significance of these reduced levels is not clear and does not necessarily imply deficiency at a cellular level. A decreased blood level may, for example, reflect reduced carrier protein levels rather than true deficiency of the micronutrient. Reduced red cell folate has been demonstrated in the case of zinc levels in HIV infection: lowered plasma zinc was not associated with reduced cellular zinc. Cellular zinc was better correlated with disease severity than plasma levels18. Another suggestion has been that the reduced levels of micronutrients documented in HIV infection may reflect an acute phase response rather than true deficiency. Thus interpretation of reduced levels of micronutrients should be viewed with caution. Such levels do not necessarily reflect deficiency states. Few researches have been done in this and the literature abounds with rigid acceptance of deficiencies. Further research is clearly required in this area so as better to define micronutrient status in HIV infection and the role that micronutrient supplementation might play.

Zinc
Finders et al found significant reductions in serum zinc in AIDS patients and postulated that the aetiology was multi-factorial. But they questioned the validity of measuring serum zinc as a reflection of body stores of zinc in patients who commonly also have reduced serum albumin, which may contribute to low levels of serum zinc. Additionally, acute infection itself may cause an apparent decrease in serum zinc concentration by intra-hepatic sequestration mediated by interleukin-17. Similarly, Malcolm et al questioned the significance of plasma zinc levels in AIDS patients.

In asymptomatic individuals, studies are conflicting. Moraglia et al18 found a wide variance in the zinc status of their asymptomatic subjects. However, Beach et al found significantly greater prevalence of reduced zinc levels in HIV positive patients compared to controls. In another study, reduced levels of serum zinc (and copper) were found to predict progression to AIDS independent of CD4 counts and dietary intake of these nutrients.

Patients with known AIDS or patients with asymptomatic plasma zinc levels is questioned, it has been suggested that zinc replacement can improve immunological function in early infection. In AIDS patients where fever is increased in HIV infection, zinc requirements may be increased and supplementation could be helpful, despite these caveats on interpretation of test results36.

Selenium
Selenium has been studied in only a few small studies. Reduced selenium in plasmas, whole blood and red blood cells in patients with AIDS and ARC7,72. Selenium supplementation has been reported to result in increased levels of whole blood selenium7,72, as well as improvement in subjective well-being77. Kavanaugh-McHugh et al18 reported a case of selenium deficiency associated with cardiomyopathy in a child with AIDS where cardiac function improved with selenium supplementation. Dwockin et al reported reduced selenium in cardiac muscle biopsies.

There have not been clinical trials to support the routine use of selenium replacement or the use of large doses of selenium, as advocated by many alternative therapists.

Vitamin B6 and folate
Reduced levels of serum vitamin B6 were found in a study of 27 HIV-infected patients with megabloblastic anemia and normal serum folate levels37. Interestingly, three of these patients were found to have intrinsic factor antibodies. Whilst the authors cited several explanations for folate and vitamin B12 levels, including reabsorption due to HIV terminal ileitis or hypochlohydroxy and subsequent bacterial overgrowth, they postulated that intrinsic factor auto-antibodies represent a pathogenic mechanism resulting in B12 deficiency in AIDS. Herbert et al similarly documented reduced levels of vitamin B12 in approximately one-third of their sample of 80 in-hospital patients who were already being supplemented with transcobalamin II, presumably reflecting malabsorption. Other studies26,38 have similarly documented reduced serum vitamin B12.

Reduced cerebrospinal folate levels have been found in AIDS patients even when serum and erythrocyte levels were normal39. Tilikkan postulated mechanisms including co-exist B12 deficiency and significant catalasm to explain the lack of documented folate deficiency in the literature.

Vitamin B12 (pyridoxine)
Vitamin B12 deficiency has been reported in asymptomatic subjects31. In this study, 4% of all patients positive for HIV were found to have vitamin B12 deficiency, although those consuming the lowest amounts of dietary vitamin B12 had levels in the deficient range. In contrast, all the subjects consuming more than 20 mg is (approximately 20 mg is a good standard) had B12 levels that are normally considered adequate. There were significant associations between reduced vitamin B12 levels and parameters of immune function in
Anthropological aspects of nutrition

Nutritional status has many determinants. The pathological manifestations of an illness such as HIV infection, are but one of these. Socio-cultural aspects have profound influences on food intake, behaviour and beliefs, and hence nutritional status, in both health and illness. Ethnographic surveys have evinced particular systems of food and health beliefs and religious beliefs may also determine food intake. Economic status is important in determining food choice. Poverty affects the ability to purchase food as well as the type of food purchased. Nutritional education may be lacking in lower socio-economic groups. There are good correlates between the prevalence of diseases such as obesity and socio-economic and educational status25. Food economics and politics may be the main factors in the development of malnutrition, particularly in developing countries. Indigenous peoples in developed nations may not only deal with poor economic conditions, but also face a multitude of other factors such as dispossession, poor sanitary conditions and lack of education. Low social activity has been shown to be strongly predictive for premature death. It has been postulated that as many social activities in our society revolve around eating, it is the meal that determines the outcome26. Certainly, social isolation, such as the single parent or single homosexual may experience, may influence food intake behaviour. Skills in food preparation are also important determinants of nutritional status. Male gender is still an important contributor to lack of food preparation skills in our society27. This may also apply to many individuals in the gay community.

HIV disease is largely a disease of young people, and the nutritional beliefs peculiar to young people will influence nutritional status and response to interventions. Young people may be particularly concerned with maintaining a culturally desirable body image. Loss of this body image (imagined) can be a major source of anxiety to HIV positive individuals as well as adversely affecting efforts to increase body weight28. Children's food choices have many determinants. Young people use food for many purposes other than sustenance, such as to underline their own identity as children and to defy the adult world29. This may be particularly true for the child who is chronically ill and has reduced opportunity to assert themselves in other areas. Recreational drugs and alcohol may influence nutritional status in these young people. The infection or organ failure associated with abuse of these substances can lead to adverse nutritional consequences. In addition, the intoxicated person is unlikely to be capable of maintaining a nutritional diet being neither able to prepare food nor to eat it.

Nutritional consequences of HIV infection

Changes in body composition

The body composition changes associated with the wasting of HIV infection have been reported in several papers. Body composition studies done in HIV infected30,31 have revealed that there are several more sophisticated described changes in body composition changes in HIV infection. Kotler et al32 studied a group of male and female patients with AIDS. Depletion of body cell mass, derived from measurement of total body potassium (TBK), was present in all subjects. Body fat content, measured by anthropometry (skin-fold thickness), was also diminished although the value of percentage body fat in male subjects was not significantly different from that obtained in healthy male homosexuals. Depletion of relative fat mass was more marked in the small group of female subjects, where the pattern of body composition changes seen in the males resembled the wasting seen in sepsis or injury, the pattern in women, where fat depletion was greater than loss of lean body mass, resembled the pattern of wasting seen in starvation. The women studied suffered from reduced food intake due to severe upper gastrointestinal candidiasis, but had fewer severe symptoms of AIDS than the males. Their body composition changes thus appeared to reflect starvation rather than changes inherent to infection with HIV. Kotler et al32 subsequently studied the relationship of depletion of body cell mass and survival time. They found that whilst reduction in body weight paralleled body cell mass depletion, the regression line for TBK/height was a better predictor of death. The calculated body fat content showed no correlation with survival time and varied considerably in individual patients. 

In a cohort of asymptomatic patients followed prospectively for 12 months33, individuals were unable to demonstrate changes in protein stores as measured by 15N in vivo neutron activation (AIA) when weight and CD4 count remained stable. As it has been postulated that loss of lean body mass may occur early in HIV infection, these findings were surprising.

Asymptomatic18 symptomatic HIV-positive and AIDS patients bioelectrical impedance (BPI) was evaluated as a measure of body cell mass30. Kotler et al34 proposed a BPI dilution techniques in the AIDS patients who had significantly lower body weights than the asymptomatic controls. The decrease in body weight was attributed to a decrease in both body cell mass and fat mass. Bioelectrical impedance was used to assess lean body mass in the Australian HIV Community Study35, a cohort of asymptomatic individuals. All the literature shows that infection were correlated with reduction in lean body mass.

Summary of body composition changes. The various body composition techniques used in research described above have been used to quantify some of the changes that occur in HIV infection. Table 4 summarizes the findings.

| Table 4. Summary of body composition changes. |

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The term ‘asymptomatic’ is used to imply ‘otherwise asymptomatic’, as clearly weight loss can be a symptom itself.

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on immune status in their own right, so that evaluation of the effect of a single nutrient deficiency, such as vitamin B6, on immune function can only be interpreted cautiously.

Vitamin A and beta-carotene
Reduced vitamin A (retinol) levels have been reported in a study of patients with AIDS. They were not taking any vitamin A supplements and, compared to healthy controls, had a significant lower retinol level. Even when dietary intake of retinol equivalents was normal, 27% had hypoaesthesia. Malabsorption may be deemed to contribute to the risk of vitamin A deficiency in infants with advanced HIV infection in infants. Beach et al. found significantly reduced vitamin A levels in their study of asymptomatic subjects. High intake of vitamin A has been proposed as an immune-modulator in HIV infection. An early study demonstrated that high intake of vitamin A enhanced survival in murine AIDS. Beta-carotene is a carotenoid that is metabolized to retinol and vitamin A and has been associated with immune modulation in both animal and human models. In HIV infection, beta-carotene has been shown to have clinical trials. Eleven subjects with HIV infection of various clinical stages were supplemented with 60 mg daily of beta-carotene for four months. Increases in the percentage of cells expressing CD8 (T cell) and CD4 (T cell) markers in antigen and transferrin receptor (activated lymphocytes) were observed.

Vitamin E
In their group of HIV positive subjects with a range of stages of infection, Bogden et al. found reduced levels of vitamin E, but in a study of asymptomatic subjects, vitamin E was in the normal range. As vitamin E is a fat-soluble vitamin, and may not be well absorbed in HIV infection, it would be expected that the prevalence of vitamin E deficiency would be similar to that found for vitamin A. The conflicting reports above probably reflect the small samples used as well as variability dependent on clinical manifestations of HIV infection.

Summary of micronutrient changes. There is considerable literature that documents reduced micronutrient levels in HIV infection. However, there is still a poor understanding of the significance of such reduced levels: they may be due to deficiency states at a cellular or, indeed, functional level. Further research is required to establish recommended intake of micronutrients in HIV infection. Such research should be considered if some of the caveats to describing reduced levels of micronutrients in HIV infection and thus also to describing clinically significant changes in such levels.

It is also important to recognize the potential of other factors in food, besides micronutrients, which may interact with immune status. There is evidence that components such as glutathione enhance or omega-3 fatty acids may suppress immune function. There has also been much recent literature examining these factors in HIV infection.

Children
Childhood HIV infection: epidemiology
The World Health Organization states that, worldwide, over one million children are infected with HIV, half of whom have already developed AIDS. It is predicted that over 10 million children will be infected with HIV by the year 2004.

In Australia, the prevalence of HIV infection in children is low. To September 1992, there were 136 notified cases of HIV infection in Australia. Initially the predominant mode of HIV transmission in Australian children was via breast-feeding. This transitory mode of transmission has been replaced by blood-borne modes of transmission, however, probably in response to the high number of cases reported. Breast-feeding is the mode of transmission to Australian children is now vertical transmission.

Nutrition in childhood HIV infection
Pediatric HIV infection often leads to growth retardation, marked failure to thrive and multiple nutritional deficiencies, particularly protein-energy malnutrition. The aetiological factors that cause this remain unclear. The process is probably serious. The aetiological factors are thought to be chronic disease, easy tiring, depression, respiratory distress, mouth infections, food insapporitive for a child's developmental stage, neurological impairment, psychological and social problems and food refusal may contribute to inadequate energy intake. Gastrointestinal dysfunction is a major problem in children with pediatric HIV infection and is also in the adult population. Enteral infections are common and include a similar spectrum of pathogens. An enteropathy occurring in the Peyer's patches has been described in HIV infected children and may result in malabsorption and in increased gastrointestinal nutrient losses. Carbohydrate malabsorption has been reported in several papers. Specific studies of micronutrient status in children with HIV infection are rare. Mantero-Aiienza et al. have studied selenium status in infected infants and found a high prevalence of reduced selenium levels, similar to findings in adult populations. The same covariates of reduced micronutrient levels have been applied to children as they do to adults (see Micronutrient status above). Paediatric guidelines on micronutrient supplementation have relied on extrapolations from adult studies.

Nutritional support may be indicated when growth fails, or during an acute infectious episode. Oral supplements are available. The guidelines for oral supplements are available. There are Guidelines for Nutritional Support of HIV-Infected Children. In the convalescent stage following an illness when 'catch-up' growth is usual, oral supplements have also been recommended.

When enteral or parenteral nutrition support is untenable, the guidelines stated should be adhered to wherever possible by oral intake, so as to maintain oral motor capacities.

Perinatal infection: breast-feeding
The European Community Study suggests that the overall risk of vertical transmission via breast-feeding (excluding the risk of transmission in the period of delivery) is 14.4% (95% confidence interval 12.1–17.7%) for infants born to women known to be HIV positive prenatally. When maternal HIV infection has occurred in the perinatal period, the estimated risk of vertical transmission is 29%.

The recent WHO/UNICEF Consensus Statement on HIV Infection and Breast-feeding [42, 43] has clarified the situation regarding recommendations for breast-feeding in HIV infection (see Table 6).


<table>
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<td>Where infections are not the primary cause of death in infancy, HIV-infected pregnant women should be advised not to breast-feed. Women whose infection status is unknown should be advised to breast-feed.</td>
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<td>When a baby is to be artificially fed, the choice of substitute feeding method and product should not be influenced by commercial pressures.</td>
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<td>Where antiretroviral treatment for the mother is required, breast-feeding should be considered.</td>
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<td>Prevention of children with severe malnutrition should be achieved.</td>
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<td>Prevention of women of child-bearing age becoming infected with HIV in the first place is the over-riding priority in preventing HIV transmission from mother to child.</td>
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<td>Breast-feeding should continue to be promoted and supported in all populations, irrespective of HIV infection rates.</td>
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Nutritional management in HIV infection
Although guidelines for nutritional management of HIV infection have been published, there is little research in the literature of the outcome of nutritional interventions. Most recommendations are based on the existing data that identify methods and consequences of malnutrition in HIV infection as well as on clinical experience.

Specific therapies
Treatment with zidovudine (AZT) may contribute to weight gain, presumably by improving immune function and by decreasing the incidence of opportunistic infections. Conversely, zidovudine use in children may have adverse effects on nutritional status. It appears that treatment of secondary infections in patients with AIDS may modify the wasting process, a very careful search for a treatable cause must be sought when assessing patients with weight loss.

A number of therapies has been used experimentally in the management of wasting in AIDS. Mostly these agents have proved to be disappointing in clinical trials. One single approach has proved useful, presumably reflecting the multifactorial and poorly understood underlying aetiology.

Megestrol acetate was noted to promote weight gain in AIDS patients. Flynn et al. compared megestrol acetate with a placebo in a study of 65 subjects with AIDS. After 12 weeks treatment, the subjects receiving megestrol acetate gained significantly more weight (comprising increases in both lean and fat mass), had improved appetite and improved assessments of well-being.

Dronabinol (delta-9-THC), a derivative of cannabis, has been used in the USA for management of HIV-related anorexia and weight loss. A short double-blind trial in children under 13 years of age showed that AIDS or ARC produced improved appetite and dietary energy intake, as well as reduction in nausea. The changes were significantly different in the group treated with dronabinol, this may have been due to the small sample size that was studied. Dronabinol has recently been approved for use in Australia.

Physical exercise may be an effective intervention as a means of preserving or increasing lean body mass. A 54-year-old male with AIDS undertook a physical training programme over a one-year period. Body composition measurements showed preservation of body cell mass (as measured by total body potassium) in another randomized, controlled clinical trial. Amongst subjects such effects were indicated on the basis of suggestions that preservation of lean mass might be an important determinant of disease modification, and that exercise is beneficial because it maintains or promotes lean body mass.

Growth hormone may be another therapy which could promote weight gain. To date, it has only been investigated in very small study.

Nutrition support
In absence of active symptomatic patients, the nutritional recommendations are: (1) maintenance of an adequate intake of essential nutrients from a variety of foods, and (2) essential micronutrient supplementation (recommended by some groups). Clinicians have suggested that early nutritional intervention appears to be beneficial. Not only is severe nutritional depletion (and its concomitant debility) prevented, but suggestions have been made that early treatment could slow disease progression. Whilst no formal research is available to support this, some authorities currently support such intervention: improved quality of life is cited as an important and tangible benefit. The small amount of research carried out on micronutrient interventions has concentrated on single nutrient supplementation. Some of this research suggests that deficiencies can be corrected and that some immunosuppressive effects may be reversed. However, there are no clear guidelines on routine supplementation as research is not yet available to support any particular regimen. Clinicians' prescribing practices cover a wide
his study. In a related study, Fordyce-Baum et al. reported the complete dietary assessments of these HIV positive women. However, micronutrient supplementation was prevalent, often at doses far in excess of the RDA. Similar findings have been reported in other studies. Many of the micronutrients taken have impact on immune status in their own right, so that evaluation of the effect of a single nutrient deficiency, such as vitamin B12, on immune function can only be interpreted cautiously.

**Vitamin A and beta-carotene**

Reduced vitamin A (retinol) levels have been found in a study of patients with AIDS. They were not taking any vitamin A supplements and, compared to healthy controls, had a significantly lower retinol level. Even when dietary intake of retinol equivalents was normal, 27% had hypothermia. Malabsorption may be deemed to contribute to the risk of vitamin A deficiency in infants. With advanced HIV infection in pregnant women, beach et al. found significantly reduced vitamin A levels in their study of asymptomatic subjects. High intake of vitamin A has been proposed as an immune-modulator in HIV infection. An early study demonstrated that high intake of vitamin A enhanced survival in murine AIDS. Beta-carotene is a carotenoid that is metabolized to retinol and vitamin A and has been associated with immune modulation in both animal and human models. In HIV infection, beta-carotene has been used in clinical trials. Eleven subjects with HIV infection of various clinical stages were supplemented with 60 mg daily of beta-carotene for 4 months. Increases in the percentage of cells expressing LGL (natural killer cell) in antigen and transferrin receptor (activated lymphocytes) were observed.

**Vitamin E**

In their group of HIV positive subjects with a range of stages of infection, Bogden et al. found reduced levels of vitamin E, but in another study of asymptomatic patients, vitamin E was in the normal range. As vitamin E is a fat-soluble vitamin, and may not be well absorbed in HIV infection, it would be expected that the prevalence of deficiency would be similar to that found for vitamin A. The conflicting reports above probably reflect the small samples used as well as variability dependent on clinical manifestations of HIV infection.

**Summary of micronutrient changes**

There is considerable literature that documents reduced micronutrient levels in HIV infection. However, there is still a poor understanding of the significance of such reduced levels; they may represent deficiency states at a cellular or, indeed, functional level. Further research is required to establish recommended intake of micronutrients in HIV infection. Such research should consider some of the caveats to describing reduced levels of micronutrients in HIV infection and thus also to describing clinically significant changes.

It is also important to recognize the potential of other factors in food, besides micronutrients, which may interact with immune status. There is evidence that components such as glutathione enhance or omega-3 fatty acids may suppress immune function. There has also been some interest in the literature examining these factors in HIV infection.

**Children**

**Childhood HIV infection: epidemiology**

The World Health Organization states that, worldwide, over one million children are infected with HIV, half of whom have already developed AIDS. It is predicted that over 10 million children will be infected with HIV by the year 2000.

In Australia, the prevalence of HIV infection in children is low. To September 1992, there were 156 children under 13 years of age with HIV infection in Australia. Initially the predominant mode of HIV transmission in Australian children was via transfusion, but this mode of transmission has decreased since 1985 and the major source of HIV transmission to Australian children is now vertical transmission.

**Nutrition in childhood HIV infection**

Pediatric HIV infection often leads to growth retardation, marked failure to thrive and multiple nutritional deficiencies, particularly protein-energy malnutrition (PEM).

The protein-energy nutrition status of children with HIV infection is probably similar to the pathogenic mechanisms in adults, although there has been far less research in the paediatric population. A wide variety of clinical problems can be related to chronic disease, easy tiring, depression, respiratory distress, mouth infections, food inappropriateness for a child’s developmental level, neuropsychic impairment, psychosocial problems and food refusal may contribute to inadequate energy intake.

Gastrointestinal dysfunction is a major problem in HIV-infected children in the adult population. Enteric infections are common and include a similar spectrum of pathogens. An enteropathy occurring in the HIV-infected infant has been described in HIV-infected children and may result in malabsorption and in increased gastrointestinal nutrient loss. Carbohydrate malabsorption has been reported in several papers.

Specific studies of micronutrient status in children with HIV infection are rare. Mantero-Atienza et al. have studied selenium status in infected infants and found a high prevalence of reduced selenium levels, similar to findings in adult populations. The same caveats to describing reduced levels of micronutrients in HIV infection and thus also to describing clinically significant changes apply to children as they do to adults (see Micronutrient status above). Paediatric guidelines on micronutrient supplementation have relied on extrapolations from adult sources.

**Nutritional support**

Nutritional support may be indicated when growth fails, or during an acute infectious episode. Oral nutritional supplements are not always practical. There are Guidelines for Nutritional Support of HIV-Infected Children. In the convalescent stage following an illness when ‘catch-up’ growth is usual, oral supplements have also been recommended.

When enteral or parenteral nutrition support is indicated, it should be administered at the point where possible by oral intake, so as to maintain oral motor capabilities.

**Perinatal infection: breast-feeding**

The European Collaborative Study suggests that the overall risk of vertical transmission via breast-feeding (exclusive breastfeeding in two feedings during delivery) is 14.4% (95% confidence interval 12.1–17.7) for infants born to women known to be HIV positive prenatally.

When maternal HIV infection has occurred in the perinatal period, the estimated risk of vertical transmission is 29%.

The recent WHO/UNICEF/UNAIDS Statement on HIV Infection and Breast-feeding has confirmed that breastfeeding should continue to be the preferred means of feeding for the infant of an HIV-infected mother, as has been known for many years. However, the most recent WHO/UNICEF/UNAIDS Statement has clarified the situation regarding recommendations for breast-feeding in HIV infection (see Table 6).

**Table 6. Summary of the WHO/UNICEF/UNAIDS Statement on HIV Infection and Breast-feeding.**

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<td>Where neonatal symptoms of infant mortality are infectious diseases and malnutrition, breast-feeding should remain the standard advice to pregnant women, including those known to be HIV positive or whose baby’s risk of becoming infected through breast milk is likely to be less than its risk of dying in infancy of other causes if deprived of breast-feeding.</td>
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**Nutritional management in HIV infection**

Although guidelines for nutritional management of HIV infection have been published, there is little research in the literature of the outcome of nutritional interventions. Most recommendations are based on the existing data that identify the pathogenesis and consequences of malnutrition in HIV infection as well as on clinical experience.

**Specific therapies**

Treatment with zidovudine (AZT) may contribute to weight gain, presumably by improving immune function and appetite. There is currently debate on such intervention: improved quality of life is cited as an important and tangible benefit.

The small amount of research carried out on micro- nutrient interventions has concentrated on single nutrient supplementation. Some of this research suggests that deficiencies can be corrected and that some immunosuppressive symptoms can be relieved. However, there are no clear guidelines on routine supplementation as research is not yet available to support any particular regimen. Clinicians’ prescribing practices cover a wide
range from micronutrient supplementation through to extensive vaccination. In patients with AIDS, nutrition support will depend on clinical symptoms at a given time. Applications of general principles of nutritional management have been limited. This review develops guidelines for the nutritional management of diarrhoea. Strategies such as restriction of fat or lactose or use of formula supplements containing medium-chain triglycerides have been utilized. This review reports a reduction in frequency of diarrhoea in patients on a low-fat, low-lactose diet. Bulk-forming agents including Metamucil, bran, or psyllium may be effective treatments.

Enteral and parenteral nutrition support has been used in patients with AIDS. The route of administration will depend, in the main, on the clinical problems of an individual patient. Enteral nutrition (either via a naso-gastric tube or via an entero-stomy for more long-term support) was assessed in a recent study. Kolter et al. proposed that HIV-infected patients may benefit from a small body of knowledge gained from research work.

Further research is needed in the many aspects of nutrition and diarrhoea, but considerable advances have been made in understanding the potential to enhance quality and length of life for those with HIV infection.

Acknowledgments — The comments and criticism provided by members of the Australian Nutrition & HIV Project Steering Committee, as well as by a number of practising clinicians, are gratefully acknowledged.

References

5. Centers for Disease Control, Center for Infectious Diseases, September 1987.

Discussion


range from micronutrient supplementation through to extensive supplementation. In patients with AIDS, nutrition support will depend on clinical symptoms at a given time. Applications of general principles of nutritional management have been used for developing guidelines for the nutritional management of diarrhoea. Strategies such as restriction of fat or lactose or use of formula supplements containing medium-chain triglycerides have been utilized in the treatment of diarrhoea. Supportive reduction in frequency of diarrhoea in patients on a low-fat elemental diet. Bulk-forming agents including Metamucil, bran, and gelatinized maize (cornstarch) are used as diarrhoea treatments. Enteral and parenteral nutrition support has been used in patients with AIDS. The route of administration will depend on the patient, on the clinical problems of an individual patient. Enteral nutrition (via a naso- gastric tube or via an enteroenterostomy for more long-term support) was assessed in a recent study. Koiter et al. proposed with HIV-related cachexia, they have highly malnourished AIDS patients who were treated with enteral feeding. They found significant repletion in body cell mass, as measured by total body potassium, as well as marked improvement in mental well-being and functional capacity. All but one of their previously bed-ridden patients was able to be discharged from hospital. One limiting factor in the use of enteral feeding in AIDS patients is the presence of severe malabsorption.

A comparative trial to assess the most appropriate enteric route of administration and the use of parenteral nutrition in AIDS patients is not universally accepted. The two factors are often used in a variety of clinical situations. The theoretical risk of catheter sepsis in immunocompromised patients is often raised by clinicians. In practice, the risk becomes less as the amount of clinical experience and the numbers of patients treated increases. But as more of the problem in HIV disease than in other immunocompromized states such as haematological malignancies. Catheter sepsis rates are determined predominantly by the technique of catheter insertion and by the techniques used. The use of peripheral intravenous lines for parenteral nutrition infusions significantly lessens the risk of catheter sepsis and is viewed by many as the preferred method of administration of parenteral nutrition for all clinical indications including those found in HIV infection.

Another argument against the use of parenteral nutrition in HIV infection might be seen as discriminatory. They include citing the ‘terminal nature’ of the infection. When parenteral nutrition is clinically indicated, such arguments alone are not considered in the management of other medical conditions and should be the same true for HIV infection.

In advanced and terminal stages of AIDS, manage- ment shifts towards palliation. The emphasis should be on maintaining dignity and comfort for the individual. Nutrition support is less likely to continue to play a role in these stages of HIV disease.

Conclusion

There has been speculation that nutritional status is one of the cofactors which determine whether the HIV

will become established and cause infection when an individual is exposed to it. Many of the clinical symp- toms associated with HIV infection are caused by the nutritional requirements of the virus. These include fever, cough, diarrhea, and weight loss.

Early malnutrition may delay progression to AIDS-related death and increase the duration of HIV infection. Factors such as body composition and nutritional status may improve prognosis, but further research is required in this area. Current guidelines for nutritional management in HIV infection rely on extrapolation from other disease mechanisms and a small body of knowledge gained from research work.

Further research is needed in the many aspects of nutritional problems and treatments. Such research will con- tribute to a better understanding of pathogenesis and progression. Definition of the effects of food and diet, nutritional formulations and microorganisms, on HIV infec- tion will provide an essential tool for the rational development of guidelines. The examination of nutritional status will provide an essential tool for the potential to enhance quality and length of life for those with HIV infections.

Acknowledgments — The comments and criticism provided by members of the Australian Nutrition & HIV Project Steering Committee, as well as by a number of practicing clinicians, are gratefully acknowled- ged.

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NUTRITION AND HIV INFECTION


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摘要
營養與人類免疫缺陷病毒（HIV）傳染：論評

營養狀況亦影響愛滋病毒的各個時期都有影響。人類免疫缺陷病毒（HIV）傳染的許多臨床特徵會引起營養問題，同時由于營養不良也影響病情惡化。由廣泛的病原，吸收不良和病變代謝改變引起的逆轉不足，均可致營養不良。此外，食物中的因素，包括微
覈營養素可抑制免疫功能。雖然這些發現的臨床意義和其它與愛滋病的嚴重程度和病後的關係仍未明確，但是已經證實人類免疫缺陷病毒傳染的各個時期，微觀營養素的水平下降。成人身體組成的改
變，包括體重下降和較多喪失非脂肪組織。兒童的人類免疫缺陷病毒傳染研究研究少，但生長障礙是一個明顯的營養缺乏症。營養組合
指出早期營養干預也許可以改變預後和生長療效。愛滋病的營養遺
憾可通過先後營養聲的在診斷、確定食物的治療因素，進食和最適合營
養支持方式的效益。考慮合理的營養計劃是很重要的，同時，考慮食
物在社會活動中對營養的效益，提供病人護理也很重要的，它將有
助于生物醫學的研究。

Uses of anthropometry in the elderly in the field setting
with notes on screening in developing countries

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A field setting can be defined as any setting outside of a fixed, permanent, and sophisticated health facility or research laboratory. The most important applications of anthropometry at field level include biological anthropology, epidemiology, clinical application, and metabolic research. Data collection in the field setting requires different levels of accuracy and precision; the standardization should also consider intra- and inter-observer variability due to the possibility of more than one observer participating in a given survey. A field setting, in contrast to the laboratory setting, involves special conditions that challenge the application of anthropometry. The required equipment is different and the conditions of data collection are less rigorous. Issues intrinsic to the target group—of education, culture and sophistication—might be limiting factors for carrying out anthropometric surveys in field settings.

Another issue is related to interpretation of the biological, nutritional and health significance of anthropometric findings in relationship to the elderly. Uncertainty regarding the accuracy of chronological age, and geography and differential survival of the elderly should be considered when designing a survey. In addition, because the majority of the elderly now live in developing countries, short stature should be a common finding in the age groups from these regions. It is in these short-stature elderly populations, that there is a need interpreting and applying anthropometric norms or references for height or weight derived from elderly populations of developed countries.

In conclusion, although the application of anthropometry to the field setting is feasible, given its enormous importance to gerontological biology, nutrition and health, researchers should consider a series of factors and paradigms when designing and carrying out anthropometric surveys at the field level.

Introduction

What is a field setting?
International research has a romantic mystique about it, and the term ‘field setting’ conjures up images of Jane Goodall studying ape colonies, or some path-helmet adorned archeologist scraping dirt from an Egyptian burial site.

Field setting should be defined by exclusion, and includes, for us: ‘Any setting outside of a fixed, perma-nent and sophisticated health facility or research labora-tory’. In a US perspective, the entire operation of the National Health and Nutrition Examination Surveys (NHANES) studies based in mobile trailers is a valid example of a field setting.

Applications of anthropometry in the field setting
Given this broad and comprehensive definition of the field setting, a large number of applications are conceivable. Table 1 lists a series of field applications of anthropometry which could involve elderly populations.

Biological anthropology
The most creative and exploratory application of anthropometrics may be in the field of biological anthrop-

| Table 1. Field applications of anthropometry involving elderly populations |
|-----------------------------|-----------------------------|
| Biological anthropology     | Health epidemiology         |
| - prevalence studies        | - cross-sectional studies of associations |
| - longitudinal studies      | - intervention studies      |
| - surveillance and monitoring of populations |
| - geriatric practice        | - disease research/c clinical investigation |

Metabolic research

Since this discipline has traditionally used the dimensions of mineralized structures (bones, dentition) as the subjects of study in a living population, the strategy would be to try to compare measurements of comparative indicators of the living body with skeletal and fossilized skeletal specimens. To some extent,