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Evaluation of nutritional counselling in HIV-associated malnutrition

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ABSTRACT—In HIV-infected patients, the outcome of counselling as the first step of a nutritional intervention programme was evaluated, in order to identify clinical and nutritional predictors for its efficacy.

75 HIV-infected patients were investigated, most with advanced disease. Nutritional status was determined by body weight, bioelectrical impedance and 7-day food intake record.

Prior mean weight loss was 10% (range = +4% to −31%). Counselling facilitated weight gain in 40/75 patients (1–4 months later, overall mean difference +1.4 ± 6.2%) and in 14/34 patients (8–11 months later, overall mean difference −1.4 ± 9.0%). Weight changes correlated with changes in body cell mass ($r^2 = .69$, $p < .001$) and in body fat ($r^2 = .29$, $p < 0.05$), but not extracellular mass. Underlying conditions such as AIDS definition, fever, and diarrhoea correlated to prior weight loss ($p < .001$) but not to the outcome of counselling. Low energy intake (before counselling, < 31.5 kcal/kg) did not correlate to prior weight loss but it predicted further weight loss ($p < 0.05$ towards normal intake). High energy intake (> 38.5 kcal/kg) correlated ($p < 0.05$) with more prior weight loss but not with further weight changes.

Nutritional counselling may be an effective first-line intervention for malnourished HIV infected patients. More than half of patients gain weight without other nutritional treatment. Whereas the severity of malnutrition is influenced by the underlying disease, fever, and diarrhoea, the course of weight change after nutritional intervention is not. Counselling may reduce the nutritional impact of these risk factors. In patients with low spontaneous intake, efficacy of counselling alone is limited, but it may help to identify those who require more invasive nutritional treatment.

Introduction

Malnutrition is a frequent and severe event in the natural history of Human Immunodeficiency Virus (HIV) infection. In industrialized countries, weight loss is experienced by between 30% of HIV infected patients in the early stages of the disease (1) and over 90% in the late stage (2). Malnutrition may be caused and worsened by some widespread clinical features of HIV infection (3) including:

1. Fever, acute opportunistic infections, recovery, and hypermetabolism which may increase energy needs.
2. Diarrhoea and malabsorption which may induce enteral nutrient loss.
3. Anorexia, dysphagia, and oral/oesophageal disease which may reduce energy intake.

Nutritional treatment strategies have been evaluated only in some small studies (4–14). Decisions for different strategies are often based more on published opinions (15–18) than on data from controlled studies. The natural history of weight changes in HIV infected patients is rather variable, although typical patterns have been identified (19). This variability impedes the assessment of efficacy of any nutritional intervention. At Cologne, a nutritional intervention program was started in 1989 (20), with counselling as the first of four steps, followed by oral supplements, enteral, and parenteral nutrition. The aims of this study were:

1. Evaluation of outcome (weight and body compartment changes) of counselling alone, as first step of nutritional intervention programme.
2. Identification of clinical and nutritional factors influencing nutritional status and outcome.

3. Definition of subgroups candidates for further steps of intervention (e.g. oral supplements, enteral nutrition).

Patients and methods

Patient characteristics

Participation in the nutritional counselling programme was open to all HIV positive patients attending the services of Internal Medicine and of Dermatology at the University of Cologne. 75 patients were evaluable, out of 160 HIV-positive participants from November 1989 to April 1992. However, analysis of outcome was restricted to patients with manifest or incipient malnutrition who received counselling as a single nutritional intervention.

Inclusion criteria were follow-up at least once 1–4 months after the beginning of counselling (n = 140), and HIV infection. Exclusion criteria were deliberate weight loss (n = 1), previous weight gain more than 5% (n = 9), participation in a prospective study (5) on enteral supplements (n = 30), incomplete clinical records (n = 16), additional opportunistic infection during the study period (n = 9). Patients characteristics are given in Table 1. The patients reported here are a subset from a larger group analyzed in a separate paper (21) on clinical risk factors for malnutrition.

Intervention

The practice of our nutritional counselling programme has been described elsewhere (22). The following components are obligatory:

1. Comprehensive nutritional history, including food intake record (prospective, 1 week), and evaluation of specific food intolerance.
2. Estimation of energy and nutrient needs, adapted to clinical situation (enteral loss, increased needs, or recovery).
3. Basic information on food safety and hygiene.
4. Basic information on needs for balanced diet (vitamin, macro- and micronutrient needs).
5. Information (and mostly discouragement) on 'alternative' diet regimens.
6. Training on choice and preparation of energy-dense, non-satiating food, avoidance of nausea and food-associated diarrhoea.
7. Training of self control techniques towards anorexia and nausea.
8. Emotional support.

Table 1 Patient characteristics (n = 75)

Age and sex	mean 41 years (20–70), 73 men, 2 women.	
Infection risk situation	71 homosexual contacts, no intravenous drug use, 4 other	
CD4 cell count	median 60 (0–790)* 10^6/l	
Stage of HIV disease	8	CDC stage A (asymptomatic)
	21	CDC stage B (symptoms, not AIDS defining)
	46	CDC stage C (AIDS defining symptoms)
History of AIDS defining events		time since first AIDS defining event: mean 8 months (0–36 months)
	20	1 episode
	16	2 episodes
	10	3–5 episodes

Assessment of nutritional status

Timepoints are referred to as T0, time before beginning of weight loss; T1, beginning of nutritional counselling; T2, 1–4 months after T1; and T3, 8–12 months after T1.

The main parameter for efficacy of nutritional intervention was weight gain or loss from T1 to T2. Follow up data at T3 are available from 34 patients. In the case of multiple measurements within the defined time, the lowest weight was chosen. Duration between timepoints was median 56 days ± 32 (T1 to T2), and median 283 days ±52 (T1 to T3). It had no measurable influence on the amount of weight changes in this time ($r = 0.09$, $p = 0.4$ for T1 to T2, $r = 0.29$, $p = 0.23$ for T1 to T3). Reasons for exclusion or loss to follow-up include death in 14 patients, additional opportunistic infection or neoplasm in 10, and refusal of follow-up by 4 patients.

A tendency of regression to the mean (23) was observed. Weight gain (T1 to T2) was higher (2.9% ± 8.0) in patients who previously had lost more than 10% than in patients with lower weight loss (0.1% ± 3.8, $p < 0.05$), but it was not correlated ($r = .22$, $p > 0.05$) to the amount of previous weight loss (T0 to T1). To account for this confounding tendency (see discussion), weight changes were calculated both in relation to weight at T0 and to weight at T1.

Body composition measurements by tetrapolar bioelectrical impedance analysis (24, 25) (Akern/RJL BIA 103 analyzer, Data Input, Frankfurt, Germany) were performed in 41 patients at T1, 37 patients at T1 and T2, and 21 patients at T1 and T3. 2 women were excluded from the analysis. The control group consisted of 281 male participants in an longitudinal study on the epidemiology of cardiovascular risk factors (26), who were equally distributed in age and height. Energy intake was determined by prospective 7 day intake record in household measures (27) in 61

out of 75 patients and was analyzed using software comprising data on 13 000 food items (28). Patients with and without available data on body composition or energy intake were not different concerning their course of body weight.

Clinical data were determined by retrospective analysis of medical records. Definition criteria were as follows:

1. Stage of disease: according to the revised Centers for Disease Control (CDC) stage definition from 1993 (29), patients were attributed to stages (A) asymptomatic other than generalized lymphadenopathy, (B) symptomatic without AIDS-defining event, or (C) fulfilling the AIDS definition.
2. Fever: more than 38.5°C for ≥ 2 weeks at ≥ 3 days per week.
3. Diarrhoea: more than 5 loose stools for ≥ 2 weeks at ≥ 3 days per week.
4. Acute opportunistic infection: infection listed in CDC classification C, ongoing symptoms and/or treatment at T1.
5. Reduced energy intake: reported intake below 31.5 kcal per kg previous body weight (i.e. < 90% of 35 kcal/kg, taken arbitrarily as 'standard' energy requirement).
6. Increased energy intake: reported intake above 38.5 kcal per kg previous body weight (i.e. > 110% of 'standard' requirement).
7. Anorexia and other symptoms related to food intake: subjective rating (absent – present – severe) by the patients.

Descriptive statistics were calculated with SPSS PC + 4.0 (30). Differences between subgroups were tested with Mann-Whitney-Wilcoxon rank sum test. Correlations were calculated with Spearman's rank correlation coefficient. Values are given as median [minimum – maximum, standard deviation (SD) from the mean] in square brackets, or as mean ± standard deviation in round brackets for comparisons between subgroups.

Results

Prior to nutritional intervention (T0 to T1), patients had lost a median of 10% [4% gain to 31% loss, SD 8.7%] of their body weight. Body weight was stable (±5%) in 23 patients (30.7%). During and after nutritional intervention (T1 to T2), weight changes averaged at plus 1.4 ± 6.2% (mean ± SD). 40 of 75 patients achieved some weight gain. Only 7 of 75 patients lost more than 5%, but 15 patients gained more than 5% of body weight.

From T1 to T3, more patients experienced significant weight loss: weight changes averaged at −1.4 ± 9.0%. 14 of 34 patients achieved some weight gain. 10 patients had lost more than 5%, but 8 patients still had gained more than 5%. No follow-up at T3 is available for 41 out of 75 patients. Weight changes from T0/T1 to T2 were not different between patients with and without follow-up at T3 ($p > 0.7$). For complete data, see Table 2 and Figure 1.

Body composition

At T1, patients had a lower body mass index than healthy controls (20.4 kg/m² [15.3 to 29.6, SD 2.8] vs. 23.1 kg/m² [15.9 to 33.6, SD 3.2] $p < 0.001$). Body composition of patients is characterized by deficiency of body fat (BF) more than of body cell mass (BCM) and by an increase in extracellular mass (ECM), compared with controls:

1. The ratio ECM/BCM was higher in patients (1.0 [0.7 to 2.0, SD 0.3]) than in controls (0.8 [0.6 to 1.5, SD 0.1], $p < 0.001$).
2. The proportion of BF to weight (%BF) was lower in patients (15.0% [8.0 to 34.5, SD 4.8] than in controls (22.4% [10.7 to 39.3, SD 5.7], $p < 0.001$).
3. The proportion of BCM to body weight (%BCM) was equal between patients (42.5% [30.3 to 49.1, SD 4.3]) and controls (42.4% [30.8 to 52.3, SD 4.6], n.s.).

Changes in body compartments during nutritional intervention are given in Table 2. From T1 to T2, there was more interindividual variability in body fat changes than in BCM or ECM changes, and BCM tended to increase more than ECM and BF. Body weight changes were strongly correlated to changes of body cell mass ($r^2 = .69$, $p < 0.001$). There was only a loose correlation to changes in body fat ($r^2 = .26$, $p < 0.05$) and no correlation to changes in extracellular mass ($r^2 = 0.01$, n.s.). In most cases, weight loss was combined with BCM and/or BF gain and ECM loss, and vice versa:

1. Among patients gaining weight, 17/23 gained more BCM than BF, 7/23 gained more BF than BCM. 15/23 had also a decrease in ECM/BCM ratio, and no patient had an increase > 0.1 in ECM/BCM ratio.
2. Among patients losing weight or weight stable, 7/14 lost more BCM, 5 lost more BF, and 1 lost equal amounts of BF and BCM. 7 also had an increase in ECM/BCM index, and no patient had an decrease > 0.1 in ECM/BCM ratio.

Similar relations of changes in body weight and composition were seen from T1 to T3, except a disproportionate loss of body fat.

Table 2 Changes in body weight and body composition

Weight changes	Median	Minimum – Maximum
T1 to T2 (75 patients)		
body weight at T2 as % of body weight at T1	100.7%	87.8% to 123.3%
body weight at T2 as % of body weight at T0	92.4%	68.6% to 112.4%
T1 to T3 (34 patients)		
body weight at T3 as % of body weight at T1	98.1%	78.0% to 117.4%
body weight at T3 at % of body weight at T0	92.3%	65.0% to 107.1%
Body composition changes		
T1 to T2 (37 patients)		
BCM at T2 as % of BCM at T1	101.5%	82.9% to 118.3%
ECM at T2 at % of ECM at T1	100.7%	86.9% to 113.3%
body fat at T2 as % of body fat at T1	99.5%	81.4% to 147.7%
T1 to T3 (21 patients)		
BCM at T3 at % of BCM at T1	98.1%	58.8% to 114.1%
ECM at T3 as % of ECM at T1	101.1%	82.8% to 127.3%
body fat at T3 as % of body fat at T1	94.0%	40.6% to 132.4%
Body mass index		
T0	22.6	18.6 to 29.6 kg/m^2
T1	20.4	15.3 to 29.6 kg/m^2
T2	20.6	15.5 to 28.6 kg/m^2
T3	20.6	14.7 to 30.2 kg/m^2

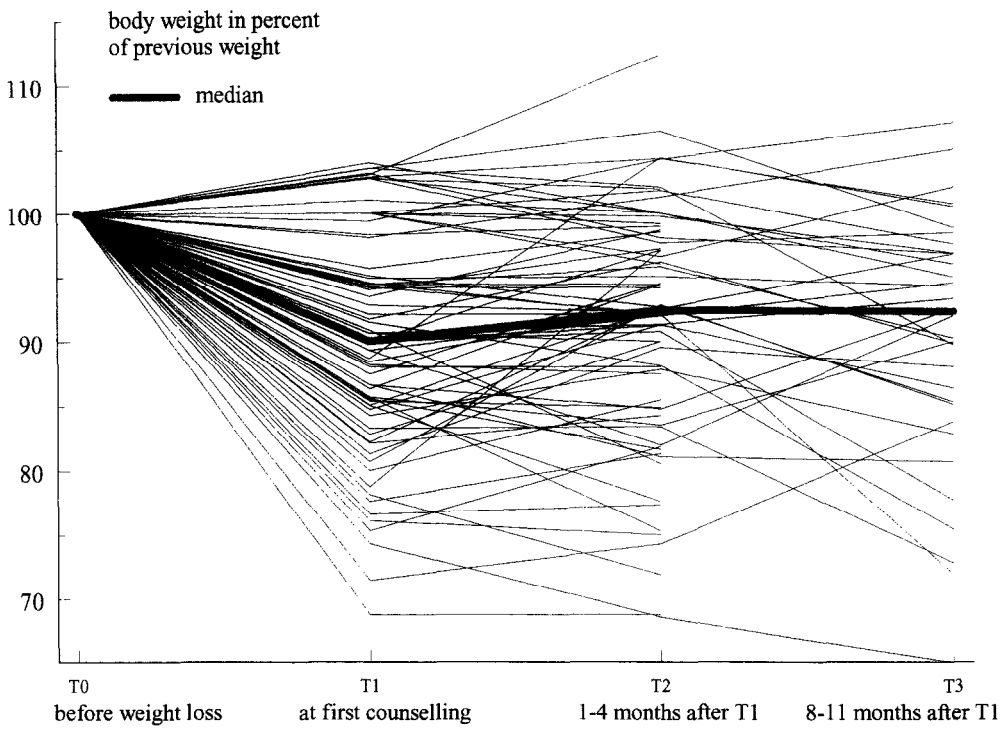

Fig. 1. Weight course before and during counselling.

Determinants of malnutrition and of outcome

Severity of malnutrition, measured by previous weight loss (T0 to T1), was strongly correlated to several clinical and nutritional risk factors. However, these clinical risk factors had much less influence on weight gain/loss T0 to T2 and no such influence on the outcome of nutritional counselling (T1 to T2). Figure 2 displays weight changes between T1 and T2 for several subgroups of patients. For the same subgroups, Figure 3 displays the percentage of initial (T0) weight achieved at T2.

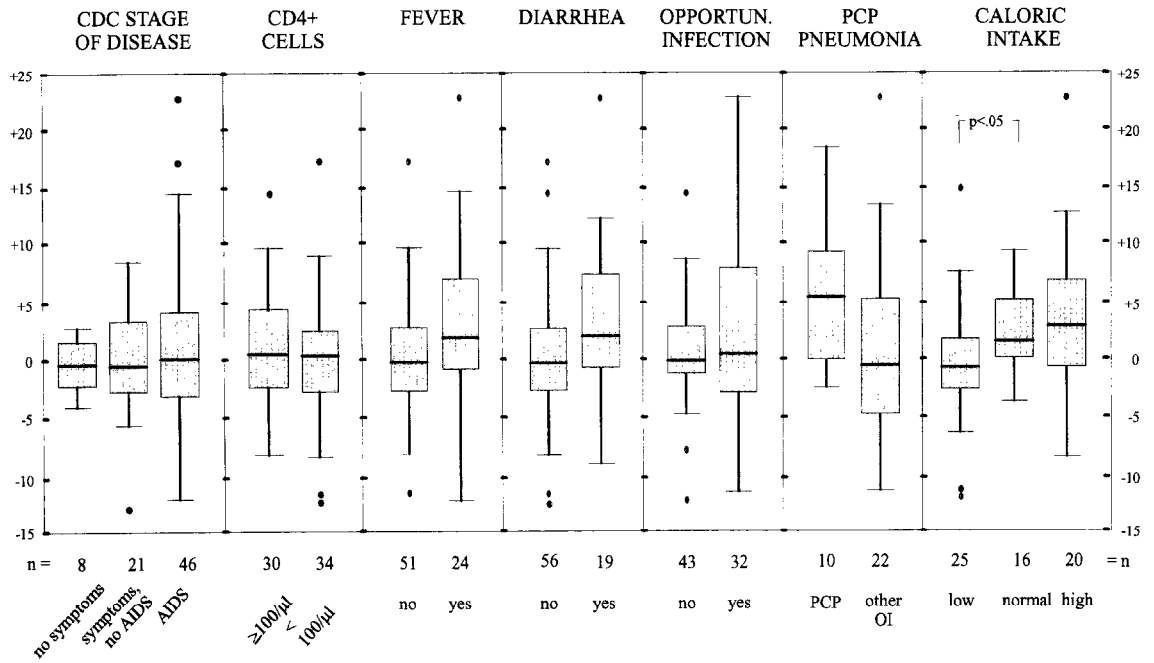

Fig. 2. Weight gain/loss after counselling, depending on clinical conditions. For each group, weight changes from T1 to T2 are given as percentage of T1 weight. Box denotes 25th to 75th percentile; lines denote range without outliers, points denote outliers (i.e., 1 to 1.5 box lengths outside of the box). Abbreviations see text. Level of significance by Mann-Whitney test; all other comparisons are nonsignificant.

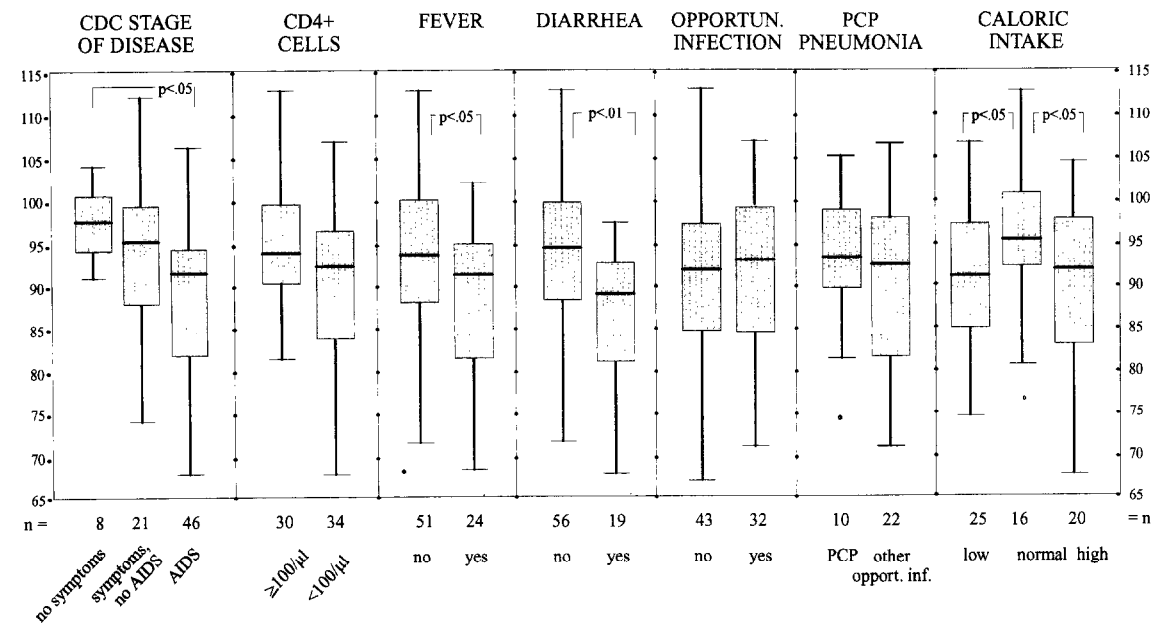

Fig. 3. Percentage of initial weight achieved after counselling, depending on clinical conditions. For each group, weight at T2 is given as percentage of T0 weight. For explanation of boxplot see Fig. 2. Abbreviations see text. Levels of significance by Mann-Whitney test; all other comparisons are nonsignificant.

Patients fulfilling the AIDS definition (CDC stage C) had lost more weight than patients with CDC stages A or B ($p < 0.001$). However, course of body weight during nutritional intervention (T1 to T2) was not different, although the variance was higher in symptomatic patients. CDC stage C patients achieved

less of their previous body weight (T0 to T2) than stage A patients ($p < 0.05$). Patients diagnosed with AIDS for more than 1 year ($n = 12$) had not lost more weight (T0 to T1) than other AIDS patients, but gained nonsignificantly less weight T1 to T2 (0.8% ± 6.7 vs. 2.0% ± 8.7, n.s.).

Patients with fever had lost more weight than their counterparts (T0 to T1, $p = 0.001$), regained less of their previous body weight (T0 to T2; $p < 0.05$), but weight gain T1 to T2 was not different. Similarly, patients with diarrhoea had lost more weight previously ($p < 0.001$), regained less of their weight (T0 to T2, $p < 0.01$), but weight gain T1 to T2 was not different.

Patients with and without acute opportunistic infection (OI) had lost the same percentage of weight ($p = 1.0$). A difference in weight gain was found neither from T0 to T2 nor from T1 to T2 ($p > 0.5$), but body cell mass gain was higher in OI patients than in patients without acute OI (+4.1% ± 5.8 vs. –0.2% ± 4.5, $p < 0.05$). The 10 patients with acute *Pneumocystis carinii* pneumonia (PCP) had lost insignificantly more weight than patients with other opportunistic infections, but they gained more weight (T1 to T2). This did not reach statistical significance due to the small patient number. The 23 patients with gastrointestinal OI had lost more weight than their counterparts (13.7% ± 8.8 vs. 7.9% ± 8.2, $p < 0.05$), but weight gain T1 to T2 was not different.

Nutritional factors had more influence on outcome than the clinical factors mentioned above (see Figs 2 & 3). Food intake is the factor that correlates the best with outcome. Before nutritional intervention, patients reported an intake of median 33.7 [15.8 to 52.9, SD 9.3] kcal per kg previous body weight. Previous weight loss was not different between 25 patients with an intake below 31.5 kcal/kg ('low' group) and 16 patients with a normal intake ('normal' group), but was higher in 20 patients with an intake more than 38.5 kcal/kg ('high' group). Weight gain (T1 to T2) was smaller in the low group than in the normal group ($p < 0.05$), and larger in the high group. Weight at T2, compared to weight at T0, was lower in both the low ($p < 0.05$) and the high ($p < 0.05$) group than in the normal group. These data may be confounded by the presence of opportunistic infections: 7/25 (28.0%) patients in the low group, 6/16 (37.5%) in the normal group, and 12/20 (60.0%) in the high group had a recent or acute opportunistic infection. No association was found between energy intake and presence of diarrhoea or fever.

Subjective complaint of anorexia was not always congruent with reported energy intake, with 5/61 patients reporting both severe anorexia and high energy intake, and 10/61 patients denying anorexia but reporting low energy intake. Anorexia was present in 52/75 (69.3%) patients and severe in 30/75 (40.0%) patients. Patients with severe anorexia had lost slightly more weight than their counterparts ($p = 0.07$), were regaining less of their previous body weight (T0 to T2, 88.9% ± 7.5% vs. 93.1% ± 9.6%, $p < 0.05$), but weight gain T1 to T2 was not different (0.9% ± 5.9% vs. 1.7% ± 6.4%, n.s.).

Other symptoms diminishing food intake (dysphagia in 24, nausea in 32, and altered taste sensation in 35 patients) were mostly combined with anorexia. Patients free from all these complaints ($n = 29$) had lost nonsignificantly less weight than patients with any nutritional complaint (T0 to T1, 6.0% ± 8.3 vs. 11.0% ± 9.5, n.s.), but weight course from T1 to T2 was not different ($p > .9$).

Discussion

Malnutrition has a strong impact on morbidity and quality of life, and probably on survival (31) of HIV infected patients. A wide array of nutritional interventions have been proposed (16), but data on their feasability, indication and outcome are scanty. Treatment strategies from nutritional counselling to enteral and parenteral nutrition have been tested in small patient samples (4–14). The validity of these studies is limited by several problems. No controlled study comparing different treatment strategies in the same patient population has hitherto been published. For ethical and practical reasons, placebo controlled studies are rarely feasible to evaluate nutritional interventions other than drugs. Clinical characteristics of participants are often reported incompletely. However, changes in nutritional status are likely to depend on the clinical course, stage of HIV infection and secondary diseases. Prior nutritional treatment, e.g. concomitant nutritional counselling, and the decision process for a given intervention are rarely reported. The amount of prior weight loss is likely to influence subsequent weight changes (tendency of regression to the mean, see below), which is not considered in most studies. Body composition is not measured in many studies. Weight changes by rehydration (e.g. in parenteral nutrition), and preferential gain in body fat or body cell mass may thus be overlooked.

The participants in this study represent all stages of HIV associated malnutrition, from appetite disorders in otherwise asymptomatic, weight stable HIV positive patients to severe, debilitating wasting syndrome associated with diarrhoea and fever. The majority were severely malnourished, with a median weight loss of 10% body weight. The relative frequency and influence of clinical and nutritional risk

factors in a larger group of our participants has been analyzed elsewhere (21). Differences between subgroups in the amount of weight loss were similar in the subset of patients analyzed here and the whole group presented in the former paper (21). Therefore, data on weight loss from T0 to T1 are not repeated in this paper.

In the first months after the start of nutritional counselling, more than half of the patients gained weight. 15 patients (20%) gained more than 5% weight (up to 23.3%), and only 7 (9.3%) lost more than 5% (up to 12.2%). Even in comparison to their weight before HIV-associated weight loss, 10 patients (13.4%) achieved a higher body weight at T2, and 18 patients (24.0%) had a weight deficit of less than 5%.

Analysis of changes in body composition demonstrate that weight gain is not due to rehydration or oedema, as the ECM/BCM ratio neither increases with weight gain nor decreases with weight loss. In weight gaining patients, the relative increase of body cell mass is larger than that of body fat, but both are highly variable. In weight losing patients, body cell mass and body fat are lost in comparable but highly variable amounts. However, bioelectrical impedance analysis is not very reliable in estimating body fat, particularly in malnourished patients (32, 33) and analysis of changes in body composition requires further study.

In the absence of a control group, these weight changes may not simply be attributed to nutritional counselling. However, these results are approximately equal to those from studies on more invasive and costly interventions such as enteral or parenteral nutrition or appetite stimulants (4–14). Counselling alone seems to be effective in a considerable part of malnourished HIV positive patients, and may be the basis for more invasive interventions in other patients.

Clinical and nutritional risk factors for malnutrition

The outcome of nutritional intervention in a patient would be expected to depend on the pathogenesis of the malnutrition. In our study, the following clinical risk factors were correlated to previous weight loss: AIDS definition (CDC stage C), chronic fever, and diarrhoea (21). However, these factors did not influence weight gain and loss after nutritional intervention (see Fig. 2). Nutritional counselling seems to be effective independently from clinical risk factors such as chronic fever, diarrhoea, and AIDS diagnosis. Moreover, these data suggest that nutritional counselling reduces the influence of these risk factors on nutritional status.

The presence of acute opportunistic infection influenced neither the amount of weight loss T0 to T1, nor the course of body weight T1 to T2 in our patient sample. However, this data should be interpreted with caution. In clinical experience, rapid weight loss is often a sign of underlying opportunistic disease which may be otherwise asymptomatic. Our data suggest that some of these diseases (e.g. atypical mycobacteriosis) may have been underdiagnosed in the past.

However, recovery from an opportunistic infection seems to facilitate body cell mass gain. This should be attributed to medical rather than to nutritional treatment. The nutritional efficacy of successful medical treatment has been documented for *Cytomegalovirus* colitis (34). In our study, patients with *Pneumocystis carinii* pneumonia, an infection with a highly effective standard treatment, seem to have gained more weight than patients with other opportunistic infections (not significant). As an attempt to exclude confounding clinical events, patients with a new opportunistic infection during the period T1 to T2 were excluded from this analysis. However, ongoing or undetected infections may have influenced the course of body weight.

Nutritional risk factors for malnutrition have a much stronger influence on outcome than clinical risk factors. Patients with appetite disorders may be overrepresented among participants in nutritional counselling (52/75 patients, 69.3%), but certainly loss of appetite and taste, dysphagia, and nausea are among the most frequent symptoms of HIV infected patients. These symptoms impair both the quality of life of patients and their ability to cope with increased energy needs. Patients reporting low energy intake gained significantly less weight during nutritional counselling than their counterparts. The presence of subjective anorexia, however, is predictive neither for reported food intake nor for outcome. These data confirm results from a prospective study on energy metabolism and intake in hospitalized HIV infected patients (35) where short-term weight changes were not correlated to resting energy expenditure but to energy intake. This was due to a very low intake in patients with acute infections who also lost more weight than other patients. The influences of low energy intake and of acute infection may have been confounded in that study. However, in our patients, opportunistic infections were even more frequent in patients with higher intake.

The correlation between caloric intake and outcome may reveal a limitation of nutritional counselling as single intervention. Many patients with severe anorexia may be unable to raise food intake sufficiently to gain weight, whereas patients with higher energy

needs (e.g. fever) or enteral nutrient loss (e.g. diarrhoea) are more likely to increase their food intake if they are supported by nutritional counselling. This underscores the importance of a comprehensive evaluation of medical and nutritional status, including a prospective intake protocol. Such a nutritional history may identify those patients who need more invasive intervention like enteral tube feeding.

Limitations of this study

The interpretation of our data should consider some of the limitations of the study design. There is no control group, as the intervention programme was offered to all HIV positive patients attending our outpatient and inpatient services. Data were collected retrospectively, so some clinical data may have been missed due to documentation failure, non-standardized or incomplete follow up. The influence of time since initiation of medical treatment (e.g. antiretroviral drugs) could not be evaluated because of too much missing data. Only the minority of patients could be followed until T3; thus interpretation of weight changes from T1 to T3 had to be very cautious. The velocity of weight changes could not be determined, as most patients remember neither the time of beginning of weight loss nor the body weight at a given timepoint.

Patients with more severe weight loss before any intervention are more likely to gain weight subsequently, independent from the intervention. This biological principle of 'regression to the mean' was first described by Sir Francis Galton (1822–1911) (23). The most desirable method accounting for this, i.e. logistic regression analysis with previous weight loss as first independent variable, was not feasible due to the small patient number, and to the highly variable risk factors and severity of malnutrition (36). Instead, we analyzed both weight gain during intervention (T1 to T2) and achievement of prior weight (T0 to T2).

Implications for future intervention studies

The data presented here may be helpful for designing future studies on nutritional interventions in malnourished HIV infected patients. We suggest that new interventions should be evaluated in controlled studies preferentially, with nutritional counselling as standard treatment. Outside of controlled studies, invasive nutritional treatment (enteral tube feeding, parenteral nutrition etc.) should be administered only to those patients who failed to gain weight after nutritional counselling or who suffer from severe disorders impeding oral food intake.

For a better comparability between studies, patient samples should be described exactly, considering factors like previous weight loss, fever, diarrhoea, and acute opportunistic disease. Body composition should be measured in every intervention study to distinguish extracellular water or fat accumulation from body cell mass gain. The roles of metabolic disorders and asymptomatic malabsorption, in comparison to clinically evident risk factors like diarrhoea and fever, require further investigation. Efficacy of nutritional intervention should be determined not only by course of body weight and body composition but also by its impact on life quality and performance status.

Nutritional counselling should become an integral part of routine medical care for HIV infected patients. It may help to restore nutritional status, even in patients suffering from severe fever or diarrhoea or in advanced stage of HIV disease. In patients with severe appetite disorders, its effect may be limited, but counselling is necessary to identify needs for more invasive nutritional interventions.

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