

# CHAPTER V EPIDEMIOLOGY AND HEALTH IMPLICATIONS OF OBESITY IN SOUTHERN AFRICA

Dr ARP Walker

## HISTORY

In centuries past, from studies of art and literature, obesity appears to have been very uncommon.<sup>1</sup> The huge majority of people were very poor, and in the main very physically active and muscular. In Biblical times only one man was described as very fat, Ehud. In classical Rome, wealthy patricians made up about a tenth or less of the population; the rest were plebeians and slaves. Among the rich and less active, obesity must have been common. As Shakespeare related, Julius Caesar distrusted lean men like Cassius, and preferred to have those surrounding him who were fat. In Western populations, for example, the UK, obesity only rose markedly from the time of the Industrial Revolution, when there were increases in urbanisation and prosperity.

In Third World populations, from the past until relatively recently, traditionally living rural adults gained little or no weight with age. This was formerly the case in Africa,<sup>2</sup> and still is in the present-day San or hunter-gatherer population in northern Botswana.<sup>3</sup> What a gross contrast to the present time when, with rise in socio-economic state and with increasing changes consequent on urbanisation, prevalence of obesity among black women, although not men, has risen tremendously, to levels exceeding those found in Western populations.

## EPIDEMIOLOGY

Obesity is considered present when a person has a body mass index (BMI) (weight (kg)/height (m)<sup>2</sup>) equal to, or greater than 30. According to the US National Center for Health Statistics (NCHS), overweight is defined as a BMI  $\geq 27,8$  in men, and  $\geq 27,3$  in women, corresponding to approximately  $\geq 20\%$  above desirable weight, as indicated in the 1983 Metropolitan Life Insurance Company Tables.<sup>4</sup>

Prevalences of obesity in South African interethnic men and women, aged mainly 15-64 years are given in Tables I and II.<sup>5-8</sup> For the necessary comparisons, some prevalences in Western and other women are given in Table III.<sup>9-15</sup> To provide additional perspective, in Table IV data are given for various groups of women, of average age 35-40 years, respecting their mean values for BMI, waist/hip ratio, and triceps skinfold thickness.<sup>16,17</sup>

---

*Alexander R P Walker, a graduate of Bristol University, emigrated to South Africa, with his newly married wife, in 1938. After working as biochemist in the City Public Health Laboratories in Johannesburg, he joined the staff of the South African Institute for Medical Research in 1953 as biochemist. He formed the Human Biochemistry Research Unit to investigate the biochemistry, metabolism and other aspects of diseases caused by unsatisfactory nutrition, whether from insufficiency or excess of nutrients. Investigations concerned populations in the four ethnic groups, in rural and urban areas, and in low and higher socioeconomic strata. His research led to obtaining the degrees of Ph.D. and D.Sc. at the University of Cape Town. In 1962 he was a member of the WHO/FAO Expert Committee on Calcium Requirements. Subsequently, in 1988, he was awarded a Citation award at the Third Vahouny Fiber Symposium in Washington; in 1989, the First International Fiber Prize by Farma Food, Copenhagen; in 1991 a gold medal from the Medical Research Council, and in 1993 the International Union of Nutritional Sciences Fellowship award for outstanding work in nutritional research. His research contributions published in about 700 papers, have been supported principally by the MRC, the National Cancer Association, and the South African Sugar Association, and Anglo-American de Beers Chairman Fund.*

Table I. BMI values for interethnic males\*

Age groups (years)						
Study	15-24	25-34	35-44	45-54	55-64	15-64
BRISK <sup>5</sup>						
Africans n	150	110	84	56	42	442
BMI	21,5 ± 3,0	23,4 ± 3,7	24,4 ± 4,8	24,5 ± 4,4	27,4 ± 4,3	23,4 ± 4,2
% BMI > 30	1,3	5,5	10,7	10,7	28,6	7,9
CRISIC <sup>6</sup>						
Coloureds n	94	96	103	95	90	478
BMI	20,9 ± 3,0	23,0 ± 3,0	24,0 ± 3,8	24,4 ± 3,7	24,8 ± 4,8	23,4 ± 3,9
% BMI ≥ 25-30	5,3	19,8	29,2	35,8	26,6	23,8
% BMI > 30	2,1	1,0	5,8	6,3	15,6	6,1
Indians <sup>7</sup> n	108	91	95	69	45	408
% BMI ≥ 30	2,8	1,1	4,2	4,3	4,4	3,2
CORIS <sup>8</sup>						
Whites n	635	634	641	705	742	3357
BMI	22,9 ± 3,3	25,9 ± 3,7	26,4 ± 3,6	27,2 ± 4,1	27,0 ± 4,0	26,0 ± 4,0
% BMI ≥ 24-30	17,0	42,1	49,5	50,0	49,0	41,0
% BMI > 30	3,6	13,2	14,3	20,9	19,8	14,7

\* Means and standard deviations

Table II. BMI values for interethnic females\*

Age groups (years)						
Study	15-24	25-34	35-44	45-54	55-64	15-64
BRISK <sup>5</sup>						
Africans n	171	147	109	64	53	544
BMI	24,8 ± 4,4	27,8 ± 6,2	30,3 ± 6,7	31,7 ± 5,5	31,9 ± 5,1	27,8 ± 6,2
% BMI > 30	12,9	30,6	47,7	59,4	54,7	34,4
CRISIC <sup>6</sup>						
Coloureds n	103	94	112	94	95	498
BMI	22,6 ± 4,4	25,7 ± 5,3	26,8 ± 5,4	30,2 ± 7,8	30,7 ± 7,3	27,1 ± 6,8
% BMI ≥ 25-30	24,3	41,5	46,5	40,4	34,7	37,8
% BMI > 30	6,8	17,0	20,5	42,6	45,3	25,9
Indians <sup>7</sup> n	69	79	103	75	44	370
% BMI ≥ 30	7,2	8,9	23,3	40,0	34,1	21,6
CORIS <sup>8</sup>						
Whites n	658	713	813	845	802	3831
BMI	22,8 ± 4,4	23,2 ± 4,6	25,7 ± 5,1	27,4 ± 5,6	28,1 ± 5,1	25,8 ± 5,3
% BMI ≥ 24-30	23,1	49,9	40,0	48,2	48,0	38,8
% BMI > 30	4,6	10,5	15,6	23,8	31,7	18,0

\* Mean and standard deviations

Table III. Prevalences of obesity (BMI  $\geq$  30) in various populations\*

Population	% Obese Females	% Obese Males
Whites:		
USA <sup>9</sup>	19	10
UK <sup>9</sup>	13	5
France <sup>9</sup>	9	5
Netherlands <sup>10</sup>	8	6
Spain <sup>11</sup>	25	20
South Africa <sup>8</sup>	18	15
Blacks:		
USA <sup>12</sup>	40-50	16
South Africa <sup>5</sup>	44	8
Other populations:		
Nauruans <sup>14</sup>	35	34
Indians (Bombay) <sup>15</sup>	10	7
Indians (South Africa) <sup>7</sup>	22	3

\* Ages mainly 15-64 years

Table IV. Average BMI, waist/hip ratio and triceps skinfold thickness of various female populations aged 35-40 years

Population	BMI	W/H ratio	Triceps(mm)
Sweden <sup>16</sup>	23,1	0,81	20,2
Poland <sup>16</sup>	23,9	0,80	22,3
Netherlands <sup>16</sup>	23,3	0,79	21,8
Italy (Verona) <sup>16</sup>	24,1	0,78	22,8
Italy (Naples) <sup>16</sup>	27,8	0,82	23,1
Saudi Arabia <sup>17</sup>	32,0	0,96	35,2

Of the South African populations, it will be observed that the proportion of obese black women is double that of white women, the position of Indian and coloured women being intermediate. Also apparent is the fact that, conversely, obesity in white men is much more common than it is in Indian, coloured and black men.

In Table III, it will be apparent that among black women in the USA, obesity is as common or more so than among South African black women. Extremely high proportions of obesity have been reported for Samoan women in the USA, about 90%. In Spain the percentage who are obese, 25%, is higher than that in local white women (18%); both are far higher than that among women in The Netherlands (8%) and France (9%). Regarding Indian women, the proportion locally (22%) is far higher than that reported for women in Bombay (10%). Clearly, among females, there are huge variations, at the extreme a tenfold range, in the occurrence of the disorder, variations which are far from explicable.

Among men, the range of percentages for obesity is less. The proportion for Spanish men (20%) is very high; those in the UK and USA at 5% and 10%<sup>9,11</sup> are lower than that in South African whites (15%).<sup>8</sup> That in black men in the USA (16%),<sup>9</sup> is far higher than that in South African black men (8%).<sup>5</sup>

Obesity is increasing in all populations, developed and developing. The increase is more marked in women. The increase has been more rapid in adolescents. Cross-sectional data in the USA indicate that there was a 58% increase in the prevalence of obesity among 12- to 17-year-old adolescent girls between 1963-1965, and 1976-1980. For black adolescent girls there was a 96% increase in prevalence.<sup>18</sup> Data derived from National Health and Nutrition Examination Surveys Studies I, II and III have revealed that between 1976 and 1980, and 1988 and 1991, BMI rose from 24,3 to 26,3; this approximates to an average gain of 7kg.<sup>19</sup>

## HEALTH HANDICAPS FROM OBESITY

To what extent does obesity promote ill-health?

### In adolescence

In youth, obesity is deemed particularly undesirable, because of the proneness of obese young to become obese adults.<sup>20</sup> Not least of reasons is the fact that about 1% of girls attempting to reduce weight develop anorexia nervosa, an increasing disorder whose mortality is as high as 1 in 30.<sup>21</sup> A related disorder, bulimia nervosa, is increasing in the UK; the lifetime prevalence in women ranges from 4% to 8%. These disorders impair physical and psychological health as well as social functioning.<sup>22</sup>

### In adulthood

As repeatedly pointed out, obesity favours the development of hypertension and hypertension-related diseases, diabetes, coronary heart disease, gallbladder disease, and certain cancers (breast, colon).<sup>23-25</sup> The relative risk of the obese has been considered to be double for hypertension, coronary heart disease and breast cancer (postmenopausal); the risk is treble or quadruple for gallbladder disease and diabetes. In terms of total mortality, it has been stated that among men and women who are 30-40% heavier than the average, i.e. those who are very obese, the mortality rate is nearly 50% higher than among those of average weight; furthermore, among those who are more than 40% heavier, i.e. extremely obese, the rate is nearly 90% higher compared with those of average weight.<sup>24</sup> Obesity also contributes to dyslipidaemias.<sup>26</sup>

The site of obesity is very important. Abdominal ('apple') obesity, more common in men, is more noxious than gynoid ('pear') obesity, more common in women.<sup>27</sup> It has been reported that fat distribution is a better predictor of cardiovascular morbidity and mortality than BMI.<sup>28</sup> In relation to this parameter, it has been shown that a 0,1 increase in waist/hip ratio can lead to a 30% decrease in the possibility for women of conception per menstrual cycle.<sup>30</sup>

It is noteworthy that obesity appears more life-threatening in some populations than in others. Thus, in the USA, obesity is more promotive of elevated serum cholesterol levels in white than in black children.<sup>31</sup> In adolescents, the correlation coefficient of association of obesity and serum HDL cholesterol level is far lower in blacks than in whites.<sup>32</sup> Black women who are obese are at lesser risk of developing diabetes;<sup>33</sup> moreover, they have longer survival,<sup>34</sup> compared with obese white women. In South Africa, some studies on elderly black women have indicated that the obese are indeed at a lower risk to the ill-effects mentioned.<sup>35</sup> However, also in the USA, among those aged 45-65 years, prevalence of cardiovascular disease has been found to be associated with obesity equally in blacks and whites.<sup>36</sup> Hence, the somewhat lesser disadvantage depicted does not negate the fact that obesity in the black population, both in the USA and South Africa, is undoubtedly a serious health problem, and should be combated accordingly.

There are other drawbacks. Certainly there is diminution in self-esteem. Consider the recent case of the Tennessee woman who sued a movie theatre in which she was refused a seat on the alleged grounds that she was a fire hazard!<sup>37</sup> Attention has recently been drawn to the fact that there is discrimination against the obese in employment.<sup>37-39</sup> In one study in the USA, obese men made

4000 dollars less per year, a differential which increased with time.<sup>40</sup>

A point of some mitigating importance is that elderly obese compared with non-obese women, are *less* prone to hip fractures in later life.<sup>41</sup> However, prospective data indicate that a high BMI is a strong predictor of long-term risk for mobility disability in older women, and that this risk persists even to very old age.<sup>42</sup>

Studies of immunological function in obese humans and experimental animals indicate that excess adiposity, in general, is associated with impairments in host defense mechanisms. However, definitive studies of immune function in human obesity are still needed.<sup>43</sup>

## **RISK FACTORS**

There are numerous influencing factors.

### **Genetic**

Certainly, there is a very strong genetic influence. A child with one or, worse, two obese parents, is at a considerably increased risk of becoming obese. Although some reports have indicated the responsibility of the genetic component to be a quarter or so, in a recent review it was concluded that 'an appreciable part of human obesity, perhaps as much as 79%, has a genetic component'.<sup>20</sup>

### **Diet**

Basically, a habitual energy intake greater than energy expenditure promotes the disorder.<sup>20,23-25</sup> As to sources of energy, the popular belief that a diet high in 'starch' or carbohydrate per se is promotive of obesity is incorrect. Actually, as noted, for traditionally living Africans a very high consumption of carbohydrate is consistent with no gain in weight with age.<sup>2</sup> A high sugar intake is almost invariably blamed, but again incorrectly so.<sup>44</sup> Although the typical American diet provides about 11% of energy from added sugars, 34-38% of energy comes from fat, which is now regarded as the most important dietary component in the aetiology of obesity.<sup>45</sup> It is illuminating that from an analysis of 55 000 Brazilian households, an investigation revealed that 85% of the variance in weight for height was explained by the fat content of the diet, even after allowing for the effect of income and of other variables.<sup>46</sup> In this connection, it has been reported that individuals with a genetic predisposition to obesity have been found to respond with more marked weight gain than control subjects when exposed to an increased dietary fat content; this may be attributable to a lower capacity to oxidise lipids. Additionally, cross-sectional studies have demonstrated that obese subjects, viewed as a whole, consume a more fat-rich diet than normal-weight subjects.<sup>47</sup> As to weight loss, it has been shown that such is more strongly associated with change in percentage energy from fat than with change in total energy intake.<sup>48</sup> Other findings indicate that dietary fat intake may play a role in obesity beyond the diet's fat content.<sup>49</sup>

Concerning other aspects of diet, the practice of snacking has often been blamed. A recent French study showed that environmental factors which were associated with obesity in children included snacking, excessive television viewing and, more importantly, short sleep duration (relative risk of the latter, 4,9).<sup>50</sup>

### **Physical activity**

A low level of activity is also a very powerful contributory factor. Western women are now almost sedentary compared with their very active ancestors (as recently as in Victorian times).<sup>51</sup> In the US, poorer women - the more prone to obesity - have a lower level of physical activity than women in higher socio-economic circumstances.<sup>52</sup> As a measure of the present-day low physical ability in the young, a study made on US high school pupils revealed that only 50% of boys and 30% of girls could do more than one pull-up.<sup>53</sup> In South Africa, while the previously high level of activity of black

women in both rural and urban areas has decreased, compared with such in the past, their level is still higher than that usual among white women.

### Socio-demographic aspects

*Socio-economic state:* This factor is very influential. Obesity is more common among the poor than among those better circumstanced with a higher level of education. However, in the USA, it has been found that black and white women in the same social class have much the same frequency of obesity.<sup>54</sup>

*Neglect in childhood:* It has been found that parental neglect during childhood predicts an increased risk of obesity in young adulthood, which is independent of age and BMI, sex, and social background.<sup>55</sup>

*Marital state:* It is intriguing that obesity is twice as common in married as in single men, although this is not the case among women.<sup>56</sup>

*Smoking:* This practice is strongly influential; ironically, it retards weight gain.<sup>57</sup> One unwanted result of stopping smoking is the frequent subsequent gain in weight. A study on Swedish women showed that those who gave up smoking experienced a gain in lower body fat, i.e. in the region not associated with increased cardiovascular risk.<sup>58</sup>

*Alcohol:* The obesity-regulating capacity of alcohol consumption is variable.<sup>59</sup> Whereas in men in the USA an inverse association has been found between obesity and alcohol consumption, this was not found to be the case in the UK nor in France.<sup>9</sup> In women in the three countries mentioned obesity prevalence varied according to level of exercise, income and alcohol consumption. An Australian study on overweight men revealed that the combination of reduced alcohol consumption and weight loss from energy restriction led to substantial and sustained reductions in blood pressure and improved serum lipid profiles.<sup>60</sup>

*Pregnancy:* It is widely believed that childbearing is associated with obesity. In young adolescent females particularly there is a risk of retaining weight gained during pregnancy. In the USA, among a population of low-income black adolescents there was significantly higher weight retention at an average of 6 weeks postpartum among those who had gained what was defined as an excessive amount of subsequent weight ( $>0,4$  kg/wk), compared with those who gained an average amount of weight during pregnancy ( $0,33$  kg/wk).<sup>61</sup> The mean amount of weight gain for each group was as follows: excessive  $9,6 \pm 5,6$  kg; average  $2,9 \pm 2,9$  kg; and small  $1,7 \pm 2,9$  kg ( $P > 0,0001$ ). The prepregnancy BMIs of the three groups of adolescents were identical.

*Puzzling features:* Although levels of knowledge of risk factors and of their significance has increased enormously in recent years, such do not fully explain the variation in the occurrence of obesity, nor individual susceptibility to the disorder. Many questions obtrude: Why should obesity be so high in women in Spain?<sup>11</sup> Why should the latter be three times more common than the prevalence among French and Netherlands women? Why are there often huge disparities in siblings in the same family? In particular, why is the disorder so common in non-traditionally living black women? Locally, from unpublished observations, it has been noted that many obese black women have short and thin children whose state of growth lies under the 5th percentile of USA National Center for Health Statistics reference standards. In such contexts, the obese mother is certainly not overeating at the expense of food for her children. Moreover, to reiterate, a high carbohydrate diet, usual to the black population, far from being promotive, is consistent with an absence of the disorder.<sup>2</sup> Turning to men, why is obesity relatively less common in Indian, and in coloured and black men, than in white men? Why should local black men have half the prevalence of obesity of black men in the USA?

There are numerous other puzzling features. As an index of the need for continuing research, 8 of

the 12 US National Institutes of Health are now carrying out obesity-related research projects.<sup>62</sup> In 1992 Bernadine Healy,<sup>63</sup> then Head of the Institutes, convened a conference to: (i) identify the practices being used to achieve weight loss and control; (ii) evaluate the evidence for the success of various methods for weight loss and control; and (iii) assess the beneficial and adverse effects of weight loss, and provide the best possible advice to the public on methods for voluntary weight loss and control. The 27 presentations made at the conference were published in a special issue (October 1993) of the *Annals of Internal Medicine*. Many of these papers are referred to in this contribution.

## CAN OBESITY REALLY BE COMBATED?

### General Considerations

Given this background of obesity's occurrence, and of its risk factors, who's for trying to lose weight? Can a sustained loss really be accomplished?

First, there should be no minimising of difficulties. In this connection, arising from a UK publication, *The Health of the Nation*, it was contended that there could be a 30% reduction in the prevalence of obesity from three measures: by informing the public about the weights associated with appreciable risk to health; by providing affordable slimming clinics; and by targeting children over the 90th percentile of weight for height.<sup>64</sup> However, this view was criticised; it was contended that knowledge of health disadvantages is already widespread among the public, that clinics demonstrably are largely ineffective, and that virtually all fat children are eager to lose weight.<sup>65</sup>

Which BMI should be aimed at? Studies on Japanese men and women with BMIs of 22,2 and 21,9 kg/m<sup>2</sup> respectively revealed that such values were associated with minimum morbidity from maladies which involved numerous organs.<sup>66</sup> These values are almost identical to those noted in the US Framingham Study.<sup>67</sup>

As to weight reduction, to be realistic, as repeatedly stated, it must be faced that almost all efforts to lose weight in the longterm result in total failure or in very short-term success, for as few as 5% or less are ultimately successful.

It is not, of course, from the want of trying, nor indeed from paying. In the USA, as many as 40% of women and 24% of men are trying to lose weight at any given time.<sup>68</sup> As to cost, in the USA, nation-wide, commercial weight reduction programmes have been stated to cost as much as 30-40 billion dollars annually (about 1400 US\$ (R4 500) per person annually), 8% of all illness costs.<sup>69</sup> This amount is about a quarter of the cost for treating all cardiovascular disease. As a specific example of high cost, in Australia, at an in-patient clinic, it cost 700 dollars (1700 rand) to achieve each kilogramme of weight loss.<sup>70</sup>

Even from historical times, when obesity did occur, weight reduction was known to be difficult. In early Grecian writings, fat people desirous of losing weight were exhorted to eat once a day, perform hard work, take no baths, sleep on a hard bed, and walk naked for as long as possible.<sup>71</sup> These robust and intriguing recommendations are matched by some current heroic practices. For example, in the USA, it has been stated that in an effort to lose weight, 14% of female students admitted to self-induced vomiting. Similar proportions have been reported from other Western populations. Nor is this practice limited to such populations. In 1992, in Ilorin, Nigeria, among high school and university students trying to lose weight, 21% had engaged in binge eating, 19% used laxatives, and in 26% vomiting.<sup>72</sup> In a corresponding interethnic study carried out in South Africa, among white obese adolescent girls, 32% were dieting, 46% had practised bulimia, 25% fasting, 7% laxatives, and 5% vomiting. However, the corresponding proportions were much lower in obese adolescent black girls.<sup>73</sup>

## Practical Issues

What are the blunt facts? To lose 0,5kg of weight per week implies taking in 3500 kcal less energy than she/he expends. Thus, to lose 0,5kg each week means having a negative energy balance of 500 kcal daily.<sup>74</sup> To do this by exercise alone, the person, daily, would have to do one of the following or equivalent - run for 45 minutes, play tennis for 1 hour, take a brisk walk for 1 ¼ hours, or play golf for 2 hours. The most reasonable way to lose weight is to decrease energy intake *and* increase energy expenditure through exercise. Experience has indicated that most people begin to lose if they decrease their energy intake below 1500 kcal a day.<sup>75</sup> But experience also indicates that most moderately to severely obese persons are unable to tolerate such a diet for long enough to reach ideal weight. Moreover, of patients who do reach this goal, as already indicated, the overwhelming majority regain the weight lost within the next 1 to 2 years or more. A point of importance is that several studies have shown that among the obese, weight cycling is not without risk.

In the USA, in a review by the National Task Force on the Prevention and Treatment of Obesity (1994) the conclusion reached regarding weight cycling is as follows.<sup>76</sup>

'There is no convincing evidence that weight cycling in humans has adverse effects on body composition, energy expenditure, risk factors for cardiovascular disease, or the effectiveness of future efforts at weight loss.'

'The currently available evidence regarding increased morbidity and mortality with variation in body weight is not sufficiently compelling to override the potential benefits of moderate weight loss in significantly obese patients. Therefore, obese individuals should not allow concerns about hazards of weight cycling to deter them from efforts to control their body weight.'

'Determination of the psychological impact of weight cycling requires further investigation.'  
'Individuals who are not obese and who have no risk factors for obesity-related illness should not undertake weight loss efforts, but should focus on the prevention of weight gain by increasing physical activity and consuming a healthful diet as recommended by the *Dietary Guidelines for Americans*.'

'Although conclusive data regarding longterm health effects of weight cycling are lacking, obese individuals who undertake weight loss efforts should be ready to commit to lifelong changes in the behavioural patterns, diet, and physical activity.'

General advice, for the determined:<sup>77</sup> (i) eat three meals a day at specified times and places; (ii) use small plates; (iii) eat predetermined amounts of food; (iv) eat slowly, and put utensils down after each bite; (v) never eat alone; (vi) keep food out of sight in the home; and (vii) shop for groceries just *after* eating, using a prepared list. In this scenario, it has been claimed that in the control of obesity the most important person in the household is one who does the shopping and cooking.<sup>78</sup> It has also been claimed that obese men benefit from obese wives earnestly trying to reduce their weight.<sup>79</sup>

Of the huge number of diets recommended, several have been demonstrated to be ineffective. At the extreme, there are the Low and Very Low Calorie Diets, which provide 1200 kcal and 400-800 kcal daily respectively. On the conventional 1200 kcal daily, combined with behavioural modification, the obese can lose about 8,5 kg in 20 weeks.<sup>80</sup> They can maintain one-half to two-thirds of this loss in the following year. However, despite many reports of its success, such diets are not without dangers.

In dissatisfaction at the poor long-term results of even extreme food restriction, a 'non-dieting' or rather a non-rigorous approach to dieting and weight management has been advanced.<sup>81</sup> It involves behavioural modification, i.e. essentially a kind of oral interaction between a patient and a therapist

or a counsellor in which ways of avoiding excess eating are discussed. The procedure involves five components, which are applied in the following manner: (i) development of therapist and peer support; (ii) cessation of strict dieting and normalising of eating patterns; (iii) gradual increase in exercise; (iv) gradual reduction of fat intake; and (v) acceptance of whatever weight is achieved by the individual's 'prudent' eating and exercise habits. Using this behavioural approach, some reports indicate weight losses of the order of 10% less than baseline weight, which have been maintained.<sup>82</sup> In other studies, of the same type, the average length of treatment is 18 weeks, and the average weight loss somewhat higher, 9,9 kg. About two-thirds of these weight losses were reported to be maintained at 1 year of follow-up.

In the USA, weight control for black women has been shown to be difficult, for illiteracy is common, especially among the unemployed, the poor and the old.<sup>83</sup> Black women may be less likely to attend weight control classes for several reasons. Fewer black women than white women perceive themselves as overweight. The percentage who do perceive themselves as overweight increases as education and income levels increase. Poverty creates barriers to attending classes, and the learning environment creates stress for adults who fear disclosures of their illiteracy. The need for obesity intervention among many black populations is compelling, but appropriate nutrition education materials are scarce.

### Pharmacological Treatment

*Drugs that reduce hunger.* These drugs e.g. amphetamines, have limited value, although they have been stated to induce significantly greater weight loss than placebo controls, especially over short periods.<sup>37</sup> Amphetamines can cause eating disorders because tolerance to the appetite-suppressing effect develops rapidly, and when the drug is withdrawn there is a rebounding effect, resulting in increased appetite.

*Drugs that enhance satiety.* Drugs of this type, e.g. fenfluramine, are widely used by medical practitioners. Side-effects are minor and short-lived.<sup>37</sup>

*Drugs that increase energy expenditure.* These drugs, e.g. dexfenfluramine, as well as inducing satiety tend to increase the thermic effect of feeding. Results in some studies have been beneficial, but in others less so.<sup>84,85</sup> Results of a 14-month Dutch study using dexfenfluramine as an appetite suppressant showed that although weight loss was not enhanced, subjects showed greater dietary compliance and were less anxious about eating behaviours than placebo-treated counterparts. It was concluded that adjunctive dietary drug therapy may be useful for clinicians working with obese patients in long-term weight loss trials, and may show promise in the treatment of bulimia.<sup>86</sup>

*Bulking agents.* These include high-fibre food concentrates, also fibre components such as methyl cellulose, and have been shown to attain a measure of success.<sup>87</sup> They produce a measure of malabsorption, i.e. the patient does not absorb all the calories taken in.

### Limitations of drug therapy

Drugs in the treatment of obesity are useful only if given in conjunction with an energy-modified diet and exercise programme.<sup>84,85</sup> They are of no benefit whatever as a quick cure. On the contrary, they can potentiate low self-esteem and other negative psychological aspects usually associated with obesity, especially when neither short-term nor long-term goals are met.

Many high authorities, as related by Saul,<sup>88</sup> such as Bray, consider that drugs certainly have a place but that they must be used long term, and continued even when weight reduction has been successfully attained. This is analogous to the continued pharmacological treatment of hypertension or of diabetes. Bray is also confident that more effective drugs will be forthcoming. Other authorities who were cited, such as Garrow, consider that the drugs are being misused. Then there are others, at the extreme, Collier was mentioned, who believe that the drugs should be banned.

"These drugs are useless ... They should only ever be used in excessively overweight people and even so, they don't really work. In the whole of St Georges Hospital (London) - with medical staff of 600 - they are not used at all."

Rarely, if ever mentioned, is the cost of treatment with drugs. For a year's course of, say, dexfenfluramine the current cost is about R1800. For long-term use, the need for which has been acknowledged by some authorities, the cost would be several thousands, which no poor person, who are the more susceptible to obesity, especially in developing populations, could afford.

## **BENEFITS AND ADVERSE EFFECTS OF WEIGHT LOSS**

In the short-term, weight loss reduces many of the health hazards associated with obesity, including insulin resistance, diabetes, hypertension and hyperlipidaemia. Potential adverse effects include a greater risk for gallstone formation and cholecystitis, excessive loss of lean body mass, and elevated uric acid levels. However, the short-term adverse effects do not outweigh the short-term benefits.<sup>89</sup>

Repeated weight changes are associated with increased cardiovascular mortality.<sup>90</sup> In the longterm, benefits from weight loss are variable. In one investigation, in moderately overweight women weight loss was associated with a 37% decrease in mortality rate. However, in severely overweight women the mortality reduction was less, at 16%.<sup>28</sup> A study in the USA revealed that at maximum BMIs of 29 or more, weight losses of 5-14% appears to be protective for men, but not for women. Among men and women whose maximum BMI was less than 29, the risk for death increased with increasing weight loss. Among participants who had been moderately overweight (maximum BMIs between 26 and 29), those who lost 15% or more had more than twice the mortality risk of those losing less than 5%.<sup>29</sup> A recent examination of several long-term studies has provided only limited information on the magnitude of the weight loss associated with changes in longevity.<sup>28</sup> This uncertainty over the long-term benefits of weight loss should caution against overclaiming in this regard. It is noteworthy that in respect of the long-term benefits from other important components of the 'prudent' lifestyle, such as stopping smoking,<sup>91</sup> and increasing physical exercise,<sup>92</sup> caution has also been expressed against the overclaiming of benefits.

## **COMMENT**

Millennia ago, Hippocrates, in a masterpiece of brevity, contended that "repletion must be followed by depletion."<sup>93</sup> In this respect the conclusion has been reached that "the problem of obesity could be eliminated in a few generations if our children were raised to know and practice prudent eating and exercise habits".<sup>94</sup> "If only it were that simple" is the heart-felt cry of millions, especially of adolescent girls and older women. Of the lifestyle components which militate against success, it must be faced that food is likely to become even more attractive than it is at present, and that the consumption of plant foods, which are less energy dense, is barely increasing despite repeated strong authoritative recommendations.<sup>95</sup> It is illuminating that long ago John Seldon (1584-1654) maintained: "'Tis not the eating, and 'tis not the drinking that is to be blamed, but the excess." Benjamin Franklin (1707-1790) concurred: "In general, mankind, since the improvement in cookery, eats twice as much as nature requires." Of the other primary lifestyle components, which militate against success, physical activity, apart from that followed by a small minority, is unlikely to increase, at least to a level meaningful to health. In this respect it is interesting that in a recent review it was concluded that a low level of physical activity "must play an important, perhaps dominant, role in the development of obesity by greatly reducing energy needs".<sup>96</sup>

A sceptic could ask, since obesity in measure is a built-in sequel closely linked with Western lifestyle, and since it is a life-long challenge to millions - are the months, or the years, of continuous struggle against the disorder really worth it? After all, despite a high and rising frequency of obesity, Western populations, particularly women, now enjoy the longest expectations of life ever (80 years in France,<sup>97</sup> 82 years in Japan<sup>98</sup>). The answer is that although some authorities such as Wooley and Garner<sup>99</sup> are greatly sceptical of benefits, the struggle must certainly go on, at least for

the benefit of the very obese, and assuredly for the benefit of all of those whose self-esteem and joie de vivre are markedly diminished by their obesity. Ways and means of change, and of encouragement, must continue to be offered, even if they simply amount to urges to eat less and to become more physically active, counsels which physicians have expressed from time immemorial.

## SUMMARY AND RESEARCH PROPOSALS

Obesity is very common, particularly among women, in both developed and developing populations. World-wide, the disorder is increasing, although more so in some populations than in others, for reasons unclear. Causally, there is a strong genetic factor. Principal amenable risk factors are level of energy intake, diet composition, physical activity, and smoking practice. Dietarily, apart from energy intake, fat consumption is deemed the most important influencing factor. The obese are more prone to hypertension, diabetes, coronary heart disease, and certain diet-related cancers. Their survival time is shortened. Socially, and job-wise, the obese are discriminated against. Although huge proportions, especially of adolescent girls and young and middle-aged women, attempt to lose weight, and most do so initially, only 5% or so succeed in attaining sustained weight loss. Basically, energy intake must be reduced and expenditure increased. Some drug treatments can enhance weight loss, but the benefits are limited. Although the value of short-term benefits is undoubted, the long-term benefits are variable. While individuals differ from each other in response, there is no short-cut to combat this ubiquitous disorder, which in most ways may be regarded as a near inevitable accompaniment to present-day lifestyle.

As to research proposals, there are three types of problems which require clarification and elucidation. (1) The first concerns learning of the reason/s why women and men in all of South African populations manifest much higher prevalences of obesity than do women and men in France and The Netherlands. What are South Africans doing wrong? What are the populations of France and of The Netherlands doing right? (2) The prevalence of obesity in African women is over five times that in African men. The prevalence in Indian women is seven times that in Indian men. Yet in the white population there is only a slight difference in obesity experience between the sexes. Can these contrasting phenomena be accounted for? (3) Observations made on Afro-American and local African women indicate that their sequelae of ageing are less severe than such in white women. Is it conceivable that there is a state of 'healthy obesity', at least in individuals among whom adverse sequelae are absent or minor?

## ACKNOWLEDGEMENTS

Grateful acknowledgements are made to the Medical Research Council, South African Sugar Association, Kellogg's South Africa, and the National Cancer Association of South Africa for grants. Appreciation for typing the manuscript goes to Miss F.I. Sookaria.

## REFERENCES

1. Trowell H. Obesity in the western world. *Plant Foods for Man* 1975;1:157-168.
2. Walker ARP. Overweight and hypertension in emerging populations. *Am Heart J* 1964;68:581-585.
3. Hansen JDL, Dunn DS, Lee PJ, Jenkins T. Hunter-Gatherers to pastoral way of life: Effects of the transition on health, growth and nutritional status. *S Afr J Sci* 1993;89:559-564.
4. Williamson DF. Descriptive epidemiology of body weight and weight change in US adults. *Ann Intern Med* 1993;119:646-649.
5. Steyn K, Jooste PL, Bourne L, *et al.* Risk factors for coronary heart disease in the black population of the Cape Peninsula. The BRISK study. *SAMJ* 1991;79:480-485.

6. Steyn K, Fourie J, Rossouw JE, Langenhoven ML, Joubert G, Chalton DO. Anthropometric profile of the coloured population of the Cape Peninsula. *SAMJ* 1990;78:68-72.
7. Seedat YK, Mayet FGH, Khan S, Somers SR, Joubert G. Risk factors for coronary heart disease in the Indians in Durban. *SAMJ* 1990;78:447-454.
8. Jooste PL, Steenkamp HJ, Benadé AJS, Rossouw JE. Prevalence of overweight and obesity and its relation to coronary heart disease in the CORIS study. *SAMJ* 1988;74:101-104.
9. Laurier D, Guiguet M, Chau NP, Wells JA, Valleron AJ. Prevalence of obesity: a comparative survey in France, the United Kingdom and the United States. *Int J Obes* 1992;16:565-572.
10. Blokstra A, Kromhout D. Trends in obesity in young adults in The Netherlands from 1974 to 1986. *Int J Obes* 1992;15:513-521.
11. Alvarez-Torices JC, Franch-Nadal J, Alvarez-Guisasola F, Hernandez-Mejia R, Cueto-Espinar A. Self-reported height and weight and prevalence of obesity. Study in a Spanish population. *Int J Obesity* 1993;17:663-667.
12. Garrow JS. Indices of adiposity. *Nutr Abstr Rev* 1983;53:697-708.
13. Price RA, Lunetta K, Ness R, et al. Obesity in Pima Indians. Distribution characteristics and possible thresholds for genetic studies. *Int J Obesity* 1992;16:851-857.
14. Hodge HM, Dowse GK, Zimmet PZ. Association of body mass index and waist-hip circumference ratio with cardiovascular disease risk factors in Micronesian Nauruans. *Int J Obesity* 1993;17:399-407.
15. Dhurandhar NV, Kulkarni PR. Out-patient weight management in India: a comparison with the west. *Internat J Food Sci Nutr* 1993;44:73-83.
16. Seidell JC, Cigolini M, Charzewska J, et al. Indicators of fat distribution study, serum lipids, and blood pressure in European women born in 1948. The European Fat Distribution Study. *Am J Epidemiol* 1989;130:53-65.
17. Al-Rehaimi AAR, Björntorp P. Obesity and fat distribution in women from Saudi Arabia. *Int J Obesity* 1992;16:1017-1019.
18. McAnarney ER, Stevens-Simon C. First, do no harm. Low birth weight and adolescent obesity. *AJDC* 1993;147:983-984.
19. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults: The National Health and Nutrition Examination Surveys, 1960 to 1991. *JAMA* 1994;272:205-211.
20. Van Itallie TB, Simopoulos AP. Summary of the National Obesity and Weight Control Symposium. *Nutr Today* 1993;Jul/Aug:33-35.
21. Patton G. The course of anorexia nervosa. About one in 30 die, and half recover fully after six years. *BMJ* 1989;299:139-140.
22. Treasure J, Schmidt U, Troop N, et al. First step in managing bulimia nervosa: controlled trial of therapeutic manual. *BMJ* 1992;308:686-689.
23. Bray GA, York B, DeLany J. A survey of the opinions of obesity experts on the causes and treatment of obesity. *Am J Clin Nutr* 1992;55(Suppl 1):151S-154S.
24. Flynn MAT, Gibney MJ. Obesity and health: why slim? *Proc Nutr Soc* 1991;50:413-432.
25. Pi-Sunyer FX. Medical hazards of obesity. *Ann Intern Med* 1993;119:655-660.
26. Denke MA, Sempos CT, Grundy SM. Excess body weight. An under-recognized contributor to dyslipidemia in white American women. *Arch Intern Med* 1994;154:401-410.
27. Anonymous. The metabolic basis for the 'apple' and the 'pear' body habitus. *Nutr Rev* 1991;49:84-86.
28. Williamson DF, Pamuk ER. The association between weight loss and increased longevity. A review of the evidence. *Ann Intern Med* 1993;119:731-736.
29. Pamuk ER, Williamson DF, Serdula MK, Madans J, Byers TE. Weight loss and subsequent death in a cohort of US adults. *Ann Intern Med* 1993;119:744-748.
30. Zaadstra BM, Seidell JC, Van Noord PAH, et al. Fat and female fecundity: prospective study of effect of body fat distribution on conception rates. *BMJ* 1993;306:484-487.
31. Resnicow K, Morabia A. The relation between body mass index and plasma total cholesterol in a multiracial sample of US schoolchildren. *Am J Epidemiol* 1990;132:1083-1090.
32. Wattigney WA, Harsha DW, Srinivasan SR, Webber LS, Berenson GS. Increasing impact of obesity on serum lipids and lipoproteins in young adults. *Arch Intern Med* 1991;151:2017-2022.
33. O'Brien TR, Flanders WD, Decoufle P, Boyle CA, DeStefano F, Teutsch S. Are racial differences in the prevalence of diabetes in adults explained by differences in obesity? *JAMA* 1989;262:1485-1488.
34. Stevens J, Keil KE, Rust PF, Tyroler HA, Gazes PC. Body mass index and body girths as predictors of mortality in black and white women. *Arch Intern Med* 1992;152:1257-1262.
35. Walker ARP, Walker BF, Manetsi N, Tsotetsi NG, Walker AJ. Obesity in black women in Soweto, South Africa. Minimal effects of hypertension, hyperlipidaemia, and hyperglycaemia. *J Roy Soc Health* 1990;110:101-103.
36. Flegal KM, Harlan WR, Landis JR. Secular trends in body mass index and skinfold thickness with socioeconomic factors in young adult women. *Am J Clin Nutr* 1988;48:535-543.
37. Allison DB, Pi-Sunyer FX. Fleshing out obesity. *The Sciences* 1994;34:38-43.
38. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. *N Engl J Med* 1993;329:1008-1012.
39. Stunkard AJ, Sorensen TIA. Obesity and socioeconomic status - a complex relation. *N Engl J Med* 1993;329:1036-1037.
40. Rodin J. Cultural and psychosocial determinants of weight concerns. *Ann Intern Med* 1993;119:643-645.
41. Dempster DW, Lindsay R. Pathogenesis of osteoporosis. *Lancet* 1993;341:797-801.
42. Launer LJ, Harris T, Rumpel C, Madans J. Body mass index, weight change, and risk of mobility disability in middle-aged and older women. The epidemiologic follow-up study of NHANES I. *JAMA* 1994;271:1093-1098.
43. Stallone DD. The influence of obesity and its treatments on the immune system. *Nutr Rev* 1994;52:37-50.
44. COMA Report. *Dietary Sugars and Human Disease: Conclusions*. Department of Health and Social Subjects No 37. London: Her Majesty's Stationery Office, 1990.
45. Drewnowski A, Garn SM. Pathophysiology of obesity. *Lancet* 1993;341:55-56.
46. James WPT. Epidemiology of obesity. *Int J Obes* 1992;55:641-644.
47. Astrup A, Buemann B, Western P, Toubro S, Raben A, Christensen NJ. Obesity as an adaptation to a high-fat diet: evidence from a cross-sectional study. *Am J Clin Nutr* 1994;59:350-355.
48. Sheppard L, Kristal AR, Kushi LH. Weight loss in women participating in a randomized trial of low-fat diets. *Am J Clin Nutr* 1991;54:821-828.
49. Tucker LA, Kano MJ. Dietary fat and body fat: a multivariate study of 205 adult females. *Am J Clin Nutr* 1992;56:616-622.

50. Locard E, Mamelie N, Billette A, Miginiac M, Munoz F, Rey S. Risk factors of obesity in a five year old population. Parental versus environmental factors. *Int J Obesity* 1992;16:721-729.
51. Fussell GE, Fussell KR. *The English Countrywoman*. London: Orbis Publishing, 1981.
52. Ford ES, Merritt RK, Heath GW, et al. Physical activity behaviors in lower and higher socio-economic status population. *Am J Epidemiol* 1991;133:1246-1256.
53. Dymont PG, Goldberg B, Haefele B, et al. Physical fitness and the schools. *Pediatr* 1987;80:449-450.
54. Flegal KM, Harlan WR, Landis JR. Secular trends in body mass index and skinfold thickness with socio-economic factors in young adult women. *Am J Clin Nutr* 1988;48:535-543.
55. Lissau I, Sorensen TIA. Parental neglect during childhood and increased risk of obesity in young adulthood. *Lancet* 1994;343:324-327.
56. Sobal J, Rauschenbach BS, Frongillo EA. Marital status, fatness and obesity. *Soc Sci Med* 1992;35:915-923.
57. Cursiter M, Holmes M, Jennett S. The effect of cessation of smoking on eating patterns, energy intake and body weight. *Proc Nutr Soc* 1992;51:154A.
58. Lissner L, Bengtsson C, Lapidus L, Björkelund C. Smoking initiation and cessation in relation to body fat distribution based on data from a study of Swedish women. *Am J Public Health* 1992;82:273-275.
59. Glass-Crome IB. Alcohol misuse as challenge to medical education: a belated remedy. *Br Med Bull* 1994;50:164-170.
60. Puddey IB, Parker M, Beilin LJ, Vandongen R, Masarei JRL. Effects of alcohol and caloric restrictions on blood pressure and serum lipids in overweight men. *Hypertension* 1992;20:533-541.
61. Lederman SA. The effect of prepregnancy weight gain on later obesity. *Obstet Gynecol* 1993;82:148-155.
62. Byrne G. A huge problem for NIH? *Science* 1988;241:907.
63. Healy B. Foreword. *Ann Int Med* 1993;119:641.
64. Garrow J. Importance of obesity. *BMJ* 1991;303:704-706.
65. Wardle J. Health of the nation. *BMJ* 1991;303:1333.
66. Tokunaga K, Matsuzawa Y, Kotani K, et al. Ideal body weight estimated from the body mass index with lowest morbidity. *Int J Obes* 1991;15:1-5.
67. Garrison RJ, Kannel WB. A new approach for estimating healthy body weights. *Int J Obes* 1993;17:417-423.
68. NIH Technology Assessment Conference Panel. Methods for voluntary weight loss and control. *Ann Intern Med* 1993;119:764-770.
69. Meade V. Conference examines weight loss in America. *American Pharmacy* 1992;NS32:30-32.
70. Bradley PJ. Very low energy diets and treating severe obesity. *Med J Austr* 1992;157:286.
71. Atkinson RL. Treatment of obesity. *Nutr Rev* 1992;50:338-339.
72. Oyewumi LK, Kazarian SS. Abnormal eating attitudes among a group of Nigerian youths: I. Bulimic behaviour. *East Afr Med J* 1992;69:663-666.
73. Walker ARP, Walker BF, Locke MM, Cassim FA, Molefe O. Body image and eating behaviour in interethnic adolescent girls. *J Roy Soc Health* 1991;111:12-16.
74. Friedman RB. Fad diets. Evaluation of five common types. *Postgrad Med* 1986;79:249-258.
75. Lissner L, Odell OM, D'Agostino RB, et al. Variability of body weight and health outcomes in the Framingham population. *N Engl J Med* 1991;324:1839-1844.
76. National Task Force on the Prevention and Treatment of Obesity. Weight cycling. *JAMA* 1994;272:1196-1202.
77. Straw WE. The dilemma of obesity. Current concepts of causes and management. *Postgrad Med* 1982;72:121-126.
78. Kennedy A. Pathophysiology of obesity. *Lancet* 1992;340:1420.
79. Shattuck AL, White E, Kristal AR. How women's adopted low-fat diets affect their husbands. *Am J Public Health* 1992;82:273-275.
80. Foster GD, Wadden TA, Peterson FJ, Letizia KA, Bartlett SJ, Conill AM. A controlled comparison of three very-low-calorie diets: effects on weight, body composition, and symptoms. *Am J Clin Nutr* 1992;55:811-817.
81. Foreyt JP, Goodrick GK. Evidence for success of behavior modification in weight loss and control. *Ann Intern Med* 1993;119:698-701.
82. Foreyt JP, Goodrick K. Weight management without dieting. *Nutr Today* 1993;March/April:4-9.
83. Kumanyika S. Obesity in black women. *Epidemiologic Rev* 1987;9:31-50.
84. Bray GA. Drug treatment of obesity. *Am J Clin Nutr* 1992;55:538S-544S.
85. Berger M. Pharmacological treatment of obesity: digestion and absorption inhibitors - clinical perspective. *Am J Clin Nutr* 1992;55:318S-319S.
86. Anonymous. Dexfenfluramine influences dietary compliance and eating behaviors of obese subjects. *Nutr Rev* 1994;52:65-68.
87. Rytting KR, Larsen S, Hægh L. Treatment of slightly to moderately overweight persons. *Tidsskrift for Den norske lægeforening* 1984;104:989-991.
88. Saul H. Fat is a pharmaceutical issue. *New Scientist* 1993;139:28-31.
89. Pi-Sunyer FX. Short-term medical benefits and adverse effects of weight loss. *Ann Intern Med* 1993;119:722-726.
90. Blair SN, Shaten J, Brownell K, Collins G, Lissner L. Body weight change, all-cause mortality, and cause-specific in the multiple risk factor intervention trial. *Ann Intern Med* 1993;119:749-757.
91. Skrabanek P. Smoking and statistical overkill. *Lancet* 1992;340:1208-1209.
92. Curfman GD. The health benefits of exercise. A critical reappraisal. *N Engl J Med* 1993;328:574-576.
93. Adams F. *The Genuine Works of Hippocrates*. Williams and Wilkins, Baltimore: 1939.
94. Peterkin BB. Dietary guidelines for Americans. *JAMA* 1990;90:1725-1727.
95. Foreyt J, Goodrick K. The ultimate triumph of obesity. *Lancet* 1995;346:134-135.
96. Chesnais JC. La duree de la vie dans les pays industrialises. *La Recherche* 1983;14:1041-1048.
97. Prentice AM, Jebb SA. Obesity in Britain: gluttony or sloth? *BMJ* 1995;311:437-439.
98. Iglehart JK. Japan's medical care system II. *N Engl J Med* 1988;319:1166-1172.
99. Wooley SC, Garner DM. Dietary treatments for obesity are ineffective. *BMJ* 1994;309:655-656.