

The Asia-Pacific  
perspective:

Redefining  
**obesity**  
and its treatment

Steering Committee

Co-chairmen

Prof S Inoue *Japan*

Prof P Zimmet *Australia*

Members

Prof I Caterson *Australia*

Prof Chen Chunming *China*

Prof Y Ikeda *Japan*

Prof Dato' Dr AK Khalid *Malaysia*

Prof YS Kim *Korea*

Research coordinator

Dr J Bassett PhD

This document has been coordinated by the International Diabetes Institute,  
a World Health Organisation Collaborating Centre for the Epidemiology  
of Diabetes Mellitus and Health Promotion for Non-communicable Diseases,  
with an education grant from Knoll.

Co-sponsored jointly by the Regional Office for the Western Pacific,  
World Health Organisation, the International Association for the Study of Obesity  
and the International Obesity Task Force.

### Acknowledgements

Dr Chen Jidi *China*

Prof Clive Cockram *Hong Kong*

Dr Hwang Kau Gin *Taiwan*

Prof Mohd Ismail Noor *Malaysia*

Prof Tai Hee Lee *Korea*

Prof Augusto Litonjua *Philippines*

Dr Su Youn Nam *Korea*

Prof Shi Yi-fan *China*

Dr Kate Steinbeck *Australia*

A/Prof Boyd Swinburn *New Zealand*

Prof Vichai Tanphaichitr *Thailand*

Prof Tim Welborn *Australia*

Prof Xiang Kum-san *China*

Dr L.T. Cavalli-Sforza *WHO Philippines*

Dr Han Tieru *WHO Philippines*

© February 2000

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system,  
or transmitted in any form or by any means electronic, mechanical, photocopying, recording or otherwise  
without the written permission of the copyright owner.

Published by Health Communications Australia Pty Limited on behalf of the Steering Committee.

# Preface

The prevalence and incidence of obesity are increasing rapidly in both developed and developing countries in the Western Pacific region of World Health Organisation. This has led to an increase in obesity-related morbidity which has imposed a heavy burden on health care systems and lowered the quality of life. Obesity is related to several chronic conditions such as Type 2 diabetes, hypertension and cardiovascular diseases. The prevalence of such obesity-related conditions is likely to increase as obesity continues to rise and prevention and control of non-communicable diseases is a high priority for World Health Organisation.

While in some Asian populations the prevalence of obesity is lower than that in European nations, the health risks associated with obesity occur at a lower body mass index (BMI) in Asian populations. In contrast, Polynesians tend to be muscular and have a higher body mass index (BMI) than Europeans, but lower body fat levels for the same BMI. These observations suggest that the current WHO criteria to define overweight and obesity using BMI may not be appropriate for some populations in the Western Pacific region. Thus new criteria to define overweight and obesity in the Asia-Pacific region have been proposed in this book.

*The Asia-Pacific Perspective: Redefining Obesity and its Treatment* is a joint enterprise of the Regional Office for the Western Pacific of the World Health Organisation, the International Association for the Study of Obesity and the International Obesity Task Force. The preparation of the report has been coordinated by the World Health Organisation Collaborating Centre for the Epidemiology of Diabetes and Health Promotion for Non-communicable Diseases in Melbourne, Australia. I am grateful for the work of Professors Paul Zimmet AM and Shuji Inoue for chairing the working group and also their committee. I would also like to thank Knoll for the education grant that provided the funding to undertake this important initiative.

I believe that these guidelines will strengthen obesity management in countries of the region and will be useful to all health professionals interested and involved in the diagnosis, management and prevention of obesity.

**Dr Shigeru Omi**  
Regional Director  
WHO Western Pacific Region

# Foreword

One of the greatest public health challenges in the first half of the 21st century is preventing the epidemic of obesity. While it is obvious that this is necessary in many western countries, the obesity issue in the Asia-Pacific region has thus far not been high on either public health or treatment agendas for countries in our region. Yet as this region becomes more affluent, the prevalence of disease associated with obesity is increasing dramatically, while obesity itself is increasing as well. It is only in recent years that data on the prevalence of obesity has begun to be reported in our region. With increased obesity prevalence comes increased metabolic disease. The need therefore to prevent and treat obesity becomes obvious to reduce its incidence and costly associated metabolic disease.

The International Obesity Task Force (IOTF) has been working intensely during the past several years to establish a clear public focus on the huge cost of obesity. The work of this group has been bolstered with the publication of the WHO Interim Report on Obesity. The region however lacks reliable data with much of the available data derived from Europe and America. Therefore, the findings may lack relevance in Asia. Expert meetings organised by IOTF and others have heard that obesity prevalence is lower in Asia, although metabolic disease tends to occur at lower BMIs. The pattern of metabolic disease is different and perhaps Asians tend to put on abdominal fat preferentially. Conversely, those from the Pacific Islands tend to get disease at greater BMIs, but are prone to diabetes. It was concluded that the approach to obesity needed to be considered in a regional context.

From these realisations has come this Asia-Pacific perspective on obesity. They are welcome as they provide a regional emphasis on the importance of obesity, its prevention and treatment. It is hoped they will be used to encourage the development of effective obesity prevention programs and policies. This document constitutes a useful starting point whose management approaches must be implemented in conjunction with existing national guidelines. Data collection on obesity prevalence, consequences and costs throughout the region must be begun and continued, and used to implement effective obesity prevention and treatment strategies. It is important for all of us in the region to collaborate in developing and redefining obesity treatment and prevention. The IOTF, IASO and WHO will remain closely involved in this process. Congratulations on this regional perspective on obesity, an important first step!

# Chairmen's Statement

In recent years there has been a dramatic increase in the prevalence of obesity in the Asia-Pacific region as well as in other regions of the world. In some developing countries both obesity and under-nutrition co-exist. In such countries, rates of infectious diseases have declined but chronic diseases such as Type 2 diabetes, cardiovascular diseases and cancer have increased dramatically. Many of these chronic diseases are related to obesity. For example, the Asia-Pacific region appears to be at the forefront of the Type 2 diabetes epidemic, and this has occurred in parallel with the increasing rates of obesity.

It has been recognised that the current WHO criteria to classify overweight and obesity in adult Europeans using the body mass index (BMI) or waist circumference, may not be appropriate in Asian or Pacific Island populations. Indeed, it has been demonstrated that the increased risks associated with obesity occur at lower BMIs in Asians, and that these populations are predisposed to visceral or abdominal obesity. In contrast, Pacific Islanders are muscular and have lower levels of body fat than Europeans for the same BMI. However, while data now are available from Asian populations to suggest changes in criteria for overweight and obesity, there are only sparse data for Pacific Islanders.

*The Asia-Pacific Perspective: Redefining Obesity and its Treatment* suggests diagnostic criteria to identify overweight and obesity in Asian populations using the body mass index and waist circumference. It also provides information on prevention and treatment of obesity in Asian and Pacific communities.

These guidelines have been produced specifically for the needs of the Asia-Pacific region. They should be used to complement, rather than replace National Guidelines where these exist.

We hope that this book will be valuable and widely used by health care professionals within the Asia-Pacific region. We are particularly grateful to Professor I. Caterson and the other panel members, Professors Chen Chunming, Y. Ikeda, A.K. Khalid and Y.S. Kim as well as Dr J. Bassett and Knoll for their support by way of an education grant.

**Prof Shuji Inoue**  
Japan

**Prof Paul Zimmet**  
Australia

# Contents

<b>1</b>	<b>Introduction</b>	<b>8</b>
1.1	Epidemiology of obesity in the Asia-Pacific region	9
1.1.1	Temporal trends in obesity prevalence	11
1.2	Modernisation, acculturation and socioeconomic status	13
<b>2</b>	<b>Assessment / Diagnosis</b>	<b>15</b>
2.1	Measurement of obesity	17
2.2	Body fat distribution	19
2.3	Obesity in children	21
<b>3</b>	<b>Consequences</b>	<b>22</b>
3.1	General mortality and morbidity risk	22
3.2	Obesity and cardiovascular risk factors	23
3.2.1	Hypertension	23
3.2.2	Dyslipidaemia	23
3.3	Obesity and Type 2 diabetes	24
3.4	The Metabolic Syndrome	25
3.5	Stroke	26
3.6	Female reproductive health	27
3.7	Respiratory function	27
3.8	Musculo-skeletal disorders	27
3.9	Cancer	28
3.10	Gastrointestinal diseases	28
3.11	Psychosocial problems	29
3.12	Consequences of childhood/adolescent obesity	29

<b>4</b>	<b>Economic Costs</b>	<b>30</b>
<b>5</b>	<b>Prevention</b>	<b>31</b>
5.1	Universal prevention	32
5.2	Selective prevention	32
5.3	Targeted prevention	32
<b>6</b>	<b>Treatment Outline</b>	<b>34</b>
6.1	Goals for obesity therapy	34
6.2	Lifestyle approaches	34
6.3	Overall approach to treatment	35
6.4	Dietary measures	35
6.5	Physical activity	37
6.6	Pharmacotherapy	37
	6.6.1 Types of anti-obesity drugs	38
	6.6.2 Drugs not appropriate for the treatment of obesity	39
	6.6.3 Contra-indications to the use of anti-obesity drugs	40
6.7	Very low calorie diets (VLCD)	40
6.8	Surgery	41
6.9	Management of obesity in children	41
<b>7</b>	<b>Risk Assessment</b>	<b>42</b>
<b>8</b>	<b>Clinical Monitoring Outline</b>	<b>43</b>
<b>9</b>	<b>Future Research</b>	<b>44</b>
9.1	Assessment of obesity	44
9.2	Attitudes to obesity and body image	45
	<b>References</b>	<b>46</b>
	<b>Appendices</b>	<b>50</b>

# 1 Introduction

Obesity has reached epidemic proportions globally, and all this evidence suggests that the situation is likely to get worse. In developed regions such as Europe, the United States of America, and in Australasia, the prevalence is high and increasing but in some developing countries even more extreme situations exist. For example, using World Health Organisation standards for obesity (see Figure 1.1), more than 70% of the adult Polynesian population in Samoa are considered obese (WHO 1998).

Coincident with the high rates of obesity, the prevalence of Type 2 diabetes is also escalating, and this increase is expected to continue. Currently, the number of people in the Asia-Pacific region with Type 2 diabetes is estimated to be 30 million and worldwide it is estimated to be 120 million (Amos *et al* 1997). By the year 2010 AD, it is predicted that a total of 216 million people worldwide will have Type 2 diabetes and there will be 130 million cases in the Asia-Pacific region (Amos *et al* 1997).

This report focuses on the dramatic rise in prevalence of obesity in developing and newly developed nations in the Asia-Pacific and Indian Ocean regions. This has resulted in an epidemic that has occurred concurrently with modernisation of lifestyle (Zimmet 1999). Obesity is now epidemic in developed nations including Australia, New Zealand and Singapore and is rapidly becoming so in many developing populations, particularly Pacific Islanders and certain Asian nations (WHO 1998). This rapid process is also seen in minority groups in developed societies eg. Maoris in New Zealand, Indians in the UK, Malaysia and Singapore, Australian Aborigines and Torres Strait Islanders (WHO 1998). Secondly, this report discusses current management issues in the Asia-Pacific region given that the co-morbidities occur at lower BMIs.

Most studies that examine the risk of adverse health associated with obesity have been based on data from Europe or the United States. However the increased health risks associated with obesity occur in people with lower BMIs in the Asia-Pacific region. Although there are no current local standards in this document we quote cut-offs identified by WHO (WHO 1998) but also acknowledge the need for different standards that are culturally specific.

In Pacific Island populations eg. Samoa, the recommended BMI standards should be higher than those recommended by WHO (Swinburn 1998), whereas in certain other Asian populations such as Chinese and Japanese it is likely that they should be lower.

The actual costs of obesity and its co-morbidities in Asia and the Pacific have not been assessed in any detail to date. In the United States the morbidity and mortality from cardiovascular disease (CVD), Type 2 diabetes, gallbladder disease, cancer and musculo-skeletal disorders was costed at approximately US\$69 billion per year (Wolf & Colditz 1994) and is likely to be much higher now. The importance of obesity as a risk factor for a number of diseases including Type 2 diabetes, cardiovascular disease, hypertension, gallstones and certain cancers, is well documented (WHO 1998).

Those with low birthweight (or more specifically, thinness at birth) and who become obese as adults have a particularly high risk of the Metabolic Syndrome (central obesity, glucose intolerance, insulin resistance, dyslipidaemia and hypertension) in later life (Hales & Barker 1992).

This syndrome has a high prevalence in communities where people with poor foetal nutrition subsequently become obese in later life.

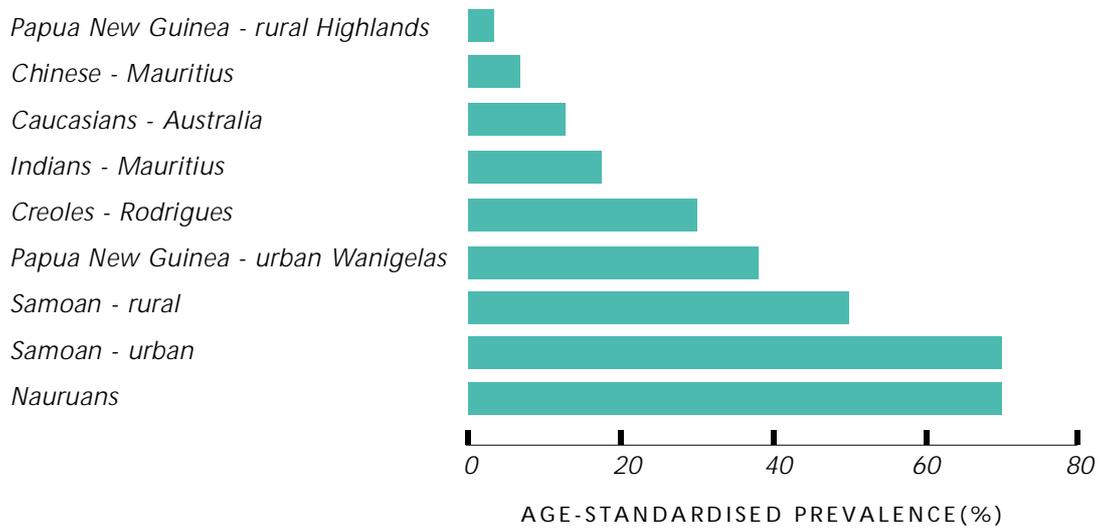
Well-designed epidemiological studies of representative population samples provide the basis for understanding the extent and public health indications of obesity. Epidemiology will also facilitate standardised measurements and definitions of obesity and overweight, to allow comparison within and between populations.

## **1.1**

### **Epidemiology of obesity in the Asia-Pacific region**

Early European explorers of the Pacific Ocean were impressed by the large and muscular physiques of the island inhabitants, particularly in Polynesia and Micronesia. In more recent times (Dowse *et al* 1995, Zimmet *et al* 1990), attention has turned to the heightened susceptibility of these populations to obesity associated with modernisation of lifestyle. The International Diabetes Institute team has collected data on the prevalence of obesity in 10 Pacific Island populations and in the multi-ethnic populations of the Indian Ocean islands of Mauritius (Asian Indians, Chinese and Creoles) and Rodrigues (mainly Creoles) over the last 20 years. Some of these data (using WHO criteria) are shown in Figure 1.1 for women aged 25-69 years. Although Mauritius is not a country of the Asia-Pacific region, the ethnic origins of two of its three main sub-groups are Asian. Thus, because of the scarcity of data from Asia, the data from Mauritius has been used to support some of the recommendations in this report.

**Figure 1.1. Obesity prevalence (BMI > 30 kg/m<sup>2</sup>)  
in women 25-69 years from Pacific and Indian Ocean populations**



The extremely high age-standardised prevalence of obesity observed in many of these populations is notable in comparison with Australians (Europids). Urban versus rural differences in the prevalence of obesity are highlighted in Papua New Guinea and Samoa. Ethnic differences are evident in Mauritius where Chinese tend to have much less obesity than Asian Indians and Creoles (African origin).

The Republic of Korea's National Nutrition Survey of 1995, found only 1.5% of the population was classified as obese (BMI > 30 kg/m<sup>2</sup>), and 20.5% were overweight (BMI 25-29.9 kg/m<sup>2</sup>). In Thailand, 4% were obese and 16% overweight. In Malaysia, 4.7% of men and 7.7% of women were obese (Ismail *et al* 1995). Among Malaysian women, ethnic differences were evident: 16.5% of Indian women were obese compared to 4.3% of Chinese and 8.6% of Malays. Urban-rural differences in the prevalence of obesity are also evident in Malaysia: 5.6% of urban men were obese compared to 1.8% of rural men, and 8.8% of urban women compared to 2.6% of rural women. In China, in the National Nutrition Survey of 1992, low rates of obesity were found among men and women (< 2%) (Chen - personal communication, Ge 1997). In urban regions (excluding Beijing, Shanghai and Tianjin), the prevalence of overweight was 12.3% of men and 14.4% of women (comparable figures for rural regions are 5.3% and 9.8%). In Japan, in the National Nutrition Survey of 1990-94, less than 3% of the population was classified as obese (BMI > 30 kg/m<sup>2</sup>), with approximately 24.3% of men and 20.2% of women being

classified as overweight (BMI 25-29.9 kg/m<sup>2</sup>) (Yoshiike *et al* 1998). The above figures show the marked ethnic and cultural variations in the prevalence of obesity and overweight across the region (see Appendix 1 for further details).

There is a paucity of data on the prevalence of childhood obesity in the Asia-Pacific region (Appendix 1). Different measures and definitions used to identify obese children further hinder comparisons between countries. Recent Malaysian data demonstrate increasing prevalence of obesity with increasing age: 6.6% among 7 year olds, rising to 13.8% of 10 year-olds (Ismail & Tan 1998). Obesity among these 7-10 year olds was higher among boys than girls (12.5% compared to 5%). Ethnic differences were also found, especially among boys, where 16.8% of Malays were obese compared to approximately 11.0% of Chinese and Indians. In China, about 10% of school children were obese in 1992 (Chen - personal communication). Recent data from Japanese school children aged 6-14 years old, shows the prevalence of obesity ranging between 5% to 11% (Ito & Murata 1999).

### 1.1.1 Temporal trends in obesity prevalence

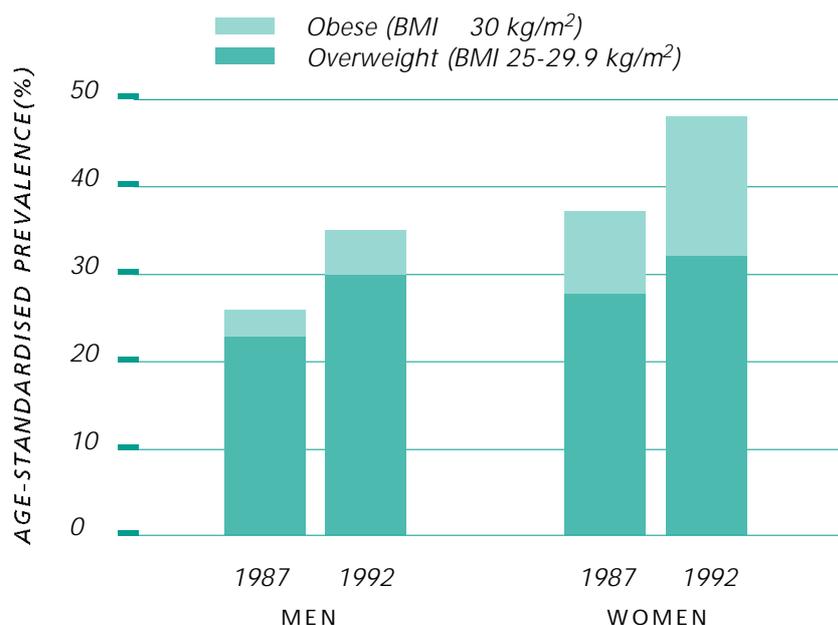
Trends over time can be seen, for example, in Samoa (1978-1991) (Hodge *et al* 1994) and Mauritius (1987-1992) (Hodge *et al* 1996) where marked increases in the prevalence of obesity have occurred, especially in rural Samoa. In 1978, a survey conducted in three areas in Samoa showed large differences in the prevalence of obesity (BMI > 30 kg/m<sup>2</sup>) between rural and urban dwellers (Dowse *et al* 1995). There were higher levels of obesity in urban vs rural areas: 74% of women in Apia were obese compared with 62% in Poutasi and 56% in Tuasivi. In men, comparable figures were 57%, 44% and 36% for Apia, Poutasi and Tuasivi respectively. Mean BMIs followed the same pattern. Even in subjects aged 25-34 years, more than 50% of women in all locations, and 45% of urban men were obese. Increasing physical activity in men, but not women, was associated with lower mean BMI. Prevalence of obesity increased dramatically between 1978 and 1991 in all locations, but especially in rural Tuasivi, where in males the increase was 297% and in females 115% (Hodge *et al* 1994).

Similarly in Mauritius, between 1987 and 1992, the prevalence of overweight or obesity (BMI > 25 kg/m<sup>2</sup>) (see Figure 1.2) increased from 26.1% to 35.7% in women (Hodge *et al* 1996). The prevalence of abdominal obesity (WHR > 85 percentile in 1987

for each sex) also increased. The cumulative incidence of overweight or obesity in men ranged from 10.8% in Chinese to 18.2% in Creoles, and in women from 16.1% to 27.5% in Chinese and Creoles, respectively. The incidence of abdominal obesity exceeded 20% in Indian men, and Indian and Creole women.

In Japan, current prevalence of obesity (BMI > 30 kg/m<sup>2</sup>) is relatively low (1.9% of men and 2.9% of women) (Yoshiike et al 1998), with little increase over the last 30 years. However there has been a 2-4 times increase in overweight men (BMI 25-29.9 kg/m<sup>2</sup>), especially in rural areas. In China, between 1982 and 1992, the prevalence of overweight and obesity increased from 9.7% to 14.9% in urban regions and from 6.8% to 8.4% in rural regions (Ge 1997). Between 1992 and 1998 the prevalence of obesity among Singaporeans remained unchanged at 6%, however, secular trends in increasing obesity prevalence were evident among certain ethnic groups. Among Malay women, 11.1% were classified as obese in 1992, and 16.2% in 1998, comparable figures for Indian women are 12.5% and 17.5%. Therefore, there are regional, ethnic and cultural variations in the temporal trends for obesity and overweight. In some areas there has been a major increase in those who are obese, in others it is the overweight group that is increasing in the Asia-Pacific region (see Appendix 1 for further details).

**Figure 1.2. Trend in prevalence of overweight and obesity - Mauritius**



Temporal trends in increasing obesity among children in the Asia-Pacific region have also been reported. In Malaysia, obesity has increased from 1% in 1990 to 6% in 1997 among 13-17 year-olds (Ismail & Zulkifli 1996, Ismail & Vickneswary 1999). In Japan, the prevalence of obesity among nine year old boys has increased from 2.9% in 1970 to 9.7% in 1997, comparative figures among nine year old girls are, 3.4% and 8.0% (Ito & Murata 1999). These extreme increases in the prevalence of obesity, even in young adults, over the relatively short 13 year study period in Samoa (Hodge *et al* 1994), and the increase in those who are overweight in Asia, suggests an increasing burden of future chronic diseases facing this region. This emphasises the need for effective intervention to bring about lifestyle modification. While in Western societies, obesity is generally socially unacceptable, in Pacific Islanders, obesity was, and in some cases remains, a symbol of wealth and increased social status (Dowse *et al* 1995). Such social and cultural factors need to be considered when developing intervention strategies. It appears that in Asian countries the problem is an increase in those who are overweight, and are already at risk of obesity-related diseases.

## 1.2

### **Modernisation, acculturation and socioeconomic status**

Rural-urban comparisons and migration studies provide evidence for an effect of acculturation or modernisation in increasing the prevalence of obesity. This effect has been attributed to reduced levels of physical activity, and the dietary changes that occur with westernisation. Decreased physical activity has been observed in urban compared with rural groups in Kiribati (King *et al* 1984), Samoa (Hodge *et al* 1994) and Fiji (Zimmet *et al* 1983). Differences in diet are less clear. As populations become more westernised, dietary composition changes to include more saturated fat and less fibre. Although this leads to more energy-dense diets, the actual energy intake may not be greater (Taylor *et al* 1992). The National Nutrition Survey in Japan, showed that the total caloric intake remained unchanged in Japanese adults over the past 30 years, although there has been an increase in fat intake in parallel with the increase in overweight. Other factors that vary with modernisation are occupation and education, as well as stresses from an urban society.

It is clear that there are socio-economic, ethnic and cultural differences between countries within the region. In the Pacific Islands there are some groups who are relatively wealthy and with it has come obesity. For example, Nauru is an affluent, westernised and

isolated Pacific Island, with very high rates of obesity. Other affluent societies (eg. Japan) have not experienced an increase in obesity, but there has been an increase in prevalence of overweight. Malaysia lies between the rates in Nauru and Japan. It is affluent, with increasing rates of obesity, and this obesity varies between genders and ethnic groups. Singapore displays a similar phenomenon, where there has been a two-fold increase in the prevalence of diabetes over eight years (Tan *et al* 1999) associated with the increase in overweight and obesity. In Indonesia, diabetes is a major disease in terms of health costs, and this is replacing infectious diseases. This phenomenon of increasing prevalence of obesity-related chronic diseases and lower prevalence of infectious diseases, is also evident in China. Whether the increase in obesity is due to increased energy intake, increase in specific foods or more sedentary lifestyle, needs to be clarified in each country or ethnic group.

# Assessment 2 Diagnosis

Obesity is defined as a condition in which there is an excess of body fat. The operational definitions of obesity and overweight however are based on body size (BMI) which is closely correlated with body fatness. These BMI cut-off points are arbitrary along a continuum of increasing risk with increasing BMI. Cut-offs used for the definition of obesity can be based on (1) statistical data from reference populations or (2) on the excess morbidity and mortality associated with increasing body fat content.

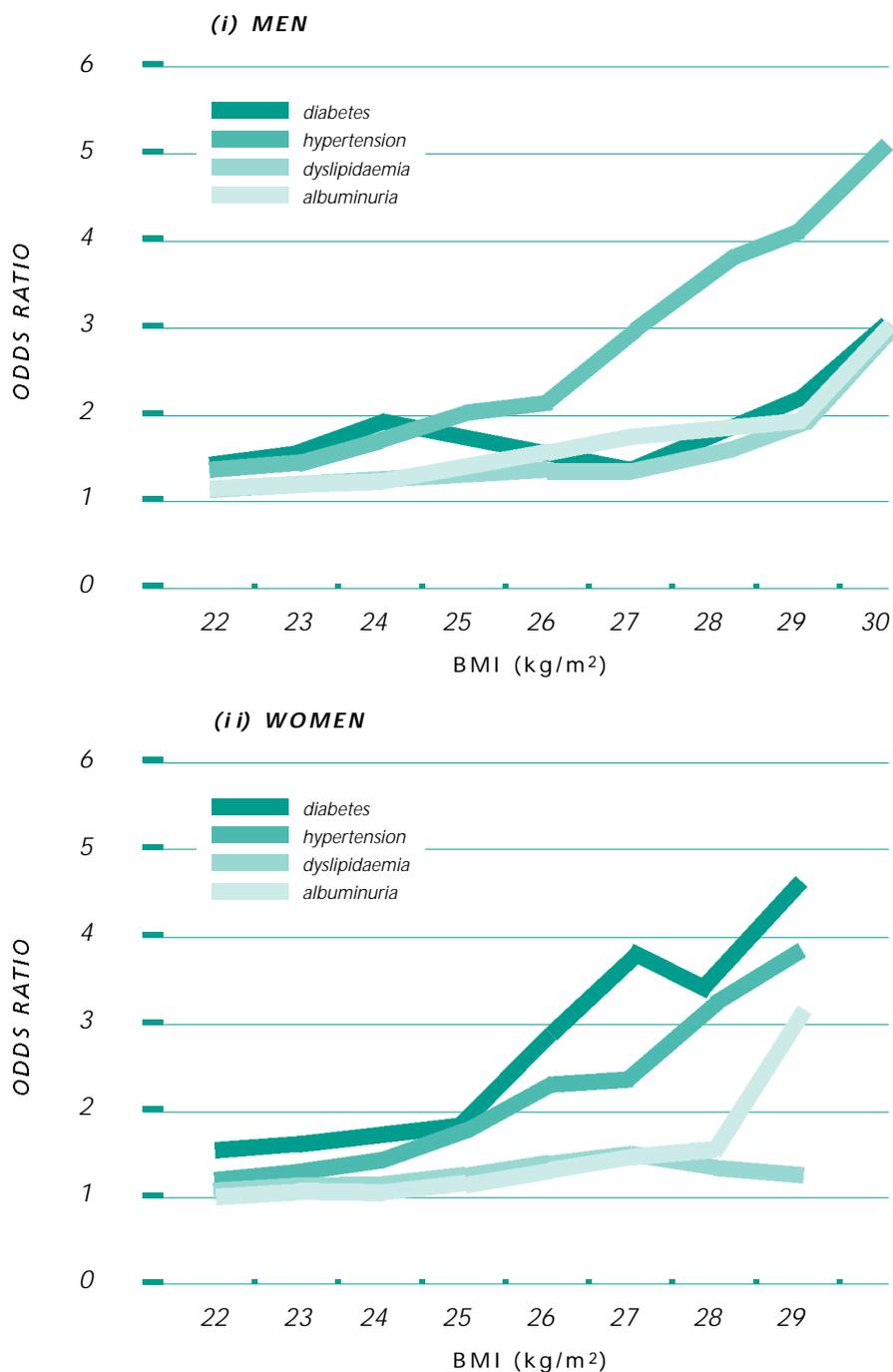
The second approach presents difficulties in children and adolescents for which there is a paucity of longitudinal data on both childhood anthropometry and adult health outcomes. The minimal research that has been conducted so far in this area has shown only weak linear relationships between adolescent overweight and later disease, making it difficult to choose a suitable cut-off. In contrast, obesity is a well-known risk factor in adults for chronic diseases such as hypertension and heart disease, hence cut-offs are easier to set. There was no international standard method used for measurement of body fat until WHO made certain recommendations (WHO 1998). However, these standards based on Europids may not indicate morbidities in certain ethnic groups, especially in this region. Furthermore, there are no widely accepted reference levels with which to compare different measures in the region's diverse national groups and very little data in relation to cut-off points that predict co-morbidities such as cardiovascular disease.

This lack of useful epidemiological data has hindered comparison of the prevalence of obesity among different populations and the study of secular trends. It does pose problems in trying to compare rates of obesity between different ethnic groups eg. Europids and Polynesians. Currently, anthropometric measurements, as discussed in greater detail below, are all that is practicable and economically feasible for epidemiological studies.

Throughout the Asia-Pacific region, there are differences in obesity prevalence and body fat distribution. For example, South Asians (Indians) have a more centralised distribution of body fat, with thick trunk skinfolds and markedly higher mean waist-to-hip

ratios (WHR) for a given level of body mass index (BMI) compared to Europids (McKeigue *et al* 1991). In Asian populations, morbidity and mortality is occurring in people with lower BMIs and smaller waist circumferences. Thus they tend to accumulate intra-abdominal fat without developing generalised obesity. Pacific Islanders tend to be very large, muscular and with high BMIs.

**Figure 2.1. Risk of diabetes, hypertension, dyslipidaemia or albuminuria according to selected BMI cut-offs in Hong Kong Chinese**



[Adapted from Ko *et al* 1999]

In a study of 1513 Hong Kong Chinese, the risk (estimated by the odds ratio) of diabetes, hypertension, dyslipidaemia and albuminuria starts to increase at a BMI of about 23 kg/m<sup>2</sup> (Ko *et al* 1999) (Figure 2.1), which is lower than the current WHO BMI cut-off used to define an increase in morbidity among Europids. Similarly, Deurenberg-Yap *et al* (1999) also demonstrated that the current WHO BMI criteria were inappropriate for Chinese men and women and that lower cut-offs were required to classify overweight and obesity in this population using data from the 1992 National Health Survey in Singapore.

## 2.1 Measurement of obesity

Height and weight are the most simple and commonly used measures. A number of weight-for-height indices have been developed of which the body mass index (BMI) (defined as weight/height (kg/m<sup>2</sup>)) is the most widely used. BMI generally correlates highly with adiposity, although it can sometimes misclassify total body fat content. For example, athletes who are muscular have a high BMI, due to muscle weighing more than fat, and will have BMIs with the overweight range, even though they are not fat. The shortest and tallest subjects also tend to be misclassified as obese (Freeman *et al* 1995). It is possible to measure adipose tissue mass by various means, but most of these require sophisticated apparatus and techniques beyond the scope of most clinical practices. Although, bio-electrical impedance (BIA) is easily applied, this technique requires further evaluation for its reproducibility in clinical and epidemiological population-based studies. Therefore BMI, which is easy to calculate, has been recommended as the measure of obesity for adults to be used in all studies.

**Table 2.1. Classification of weight by BMI in adult Europids (WHO 1998)**

Classification	BMI (kg/m <sup>2</sup> )	Risk of co-morbidities
<i>Underweight</i>	< 18.5	<i>Low (but increased risk of other clinical problems)</i>
<i>Normal range</i>	18.5-24.9	<i>Average</i>
<i>Overweight:</i>	25	
<i>Pre-obese</i>	25-29.9	<i>Increased</i>
<i>Obese I</i>	30-34.9	<i>Moderate</i>
<i>Obese II</i>	35-39.9	<i>Severe</i>
<i>Obese III</i>	40	<i>Very severe</i>

The World Health Organisation (WHO 1998) consultation on obesity proposed a system of classification based on BMI (Table 2.1), similar to classifications used in a number of past studies on Europeans. In these studies the ranges for BMI (underweight, normal, overweight, obese) have been based on mortality outcomes where the confounding influences of cigarette smoking and co-existing chronic disease have been minimised.

Here we recommend different ranges for the Asia-Pacific region based on risk factors and morbidities (Table 2.2). In Asians, the cut-offs for overweight ( $\geq 23.0$  kg/m<sup>2</sup>) and obesity ( $\geq 25.0$  kg/m<sup>2</sup>) are lower than the WHO criteria. These provisional recommendations will need to be revised in the light of further validation of studies and clinical experience. Some support for these cut-offs comes from data on Chinese living in Hong Kong (Ko *et al* 1999) (see Figure 2.1 where the morbidity risk increases with a BMI  $> 23$  kg/m<sup>2</sup>). Similar data have been published from the Chinese in Singapore (Deurenberg-Yap *et al* 1999) and in Indian Asians living in Mauritius, where there is a significantly increased risk of Type 2 diabetes and hypertension among those with a BMI between 23 to 24.9 kg/m<sup>2</sup> compared to those within the normal range (see Appendix 3). Clearly these cut-offs do not apply to Pacific Islanders. In these populations, higher cut-offs are required to define overweight and obesity of BMI  $\geq 26$  kg/m<sup>2</sup> and BMI  $\geq 32$  kg/m<sup>2</sup> respectively (Swinburn 1998) however, sparse data exist at present to make definitive recommendations.

As children are still growing, the adult BMI cut-offs are not considered appropriate for children. As with adults, BMI cut-offs need to be considered for different ethnic groups. Although the risk of problems is mildly increased in those who are overweight, treatment in this group will obtain the greatest benefit because of the numbers of those who are overweight and the ease of reducing risk to normal.

**Table 2.2. Proposed classification of weight by BMI in adult Asians**

Classification	BMI (kg/m <sup>2</sup> )	Risk of co-morbidities
<i>Underweight</i>	$< 18.5$	<i>Low (but increased risk of other clinical problems)</i>
<i>Normal range</i>	18.5-22.9	<i>Average</i>
<i>Overweight:</i>	23	
<i>At risk</i>	23-24.9	<i>Increased</i>
<i>Obese I</i>	25-29.9	<i>Moderate</i>
<i>Obese II</i>	30	<i>Severe</i>

It is not just the amount of fat but also its distribution that determines the risk associated with obesity. Abdominal or visceral fat (android obesity) is associated with the cardiovascular risk factors of the Metabolic Syndrome. These include Type 2 diabetes, impaired glucose tolerance, and hypertension and dyslipidaemia (high triglyceride, low LDL cholesterol). It is the mass of visceral adipose tissue which leads to these abnormalities.

While this can be measured with serial magnetic resonance imaging (MRI), computed tomography (CT), or by dual-energy x-ray absorptiometry (DEXA), it is usually assessed with a single CT or MRI scan performed at L3/L4 level and the visceral fat area calculated. In Caucasians an area  $> 130 \text{ cm}^2$  is associated with metabolic disease whilst areas  $< 110 \text{ cm}^2$  are at low risk. Whilst it is the visceral fat mass that is important, the simple clinical measure is waist circumference. In a study of Dutch men and women the following waist measurements were found to be associated with a substantially increased risk of metabolic complications:  $> 102 \text{ cm}$  in men or  $> 88 \text{ cm}$  in women (Han *et al* 1995).

The ratio of waist to hip circumference (WHR) is also used as a measure of abdominal obesity. In Caucasians, a WHR  $> 1.0$  for men, and WHR  $> 0.85$  for women are used to identify those with abdominal fat accumulation (James 1996): however, waist circumference is the preferred measure of abdominal obesity compared to the WHR (WHO 1998).

**Table 2.3. Co-morbidities risk associated with different levels of BMI and suggested waist circumference in adult Asians**

Classification	BMI (kg/m <sup>2</sup> )	Risk of co-morbidities	
		Waist circumference	
		< 90 cm (men) < 80 cm (women)	90 cm (men) 80 cm (women)
<i>Underweight</i>	< 18.5	<i>Low (but increased risk of other clinical problems)</i>	<i>Average</i>
<i>Normal range</i>	18.5-22.9	<i>Average</i>	<i>Increased</i>
<i>Overweight:</i>	23		
<i>At risk</i>	23-24.9	<i>Increased</i>	<i>Moderate</i>
<i>Obese I</i>	25-29.9	<i>Moderate</i>	<i>Severe</i>
<i>Obese II</i>	30	<i>Severe</i>	<i>Very severe</i>

Populations differ in the level of risk associated with a particular waist circumference. South Asians (Indians) have high levels of abdominal obesity, although they may not be considered obese by conventional BMI criteria (McKeigue *et al* 1991). Although the recent WHO report (WHO 1998) suggests that 94 cm in men and 80 cm in women should be the appropriate measures in Europids, these cut-offs are not suitable for Asian populations. This report suggests that instead 90 cm for men and 80 cm for women be used as interim lower values for Asians. Again these values will require revision when new prospective data is available. However, it is important to include waist measures in any assessment of obesity, since a reduction in waist even with no weight change may result in significant risk reduction. Table 2.3 gives co-morbidities risk in adult Asians for different levels of BMI and waist circumferences.

When using circumference measurements that standard anatomical locations are used. The WHO (WHO 1995) recommended methods are as follows. For waist or abdominal circumference, the subject stands with feet 25-30 cm apart, weight evenly distributed. Measurement is taken midway between the inferior margin of the last rib and the crest of the ilium in a horizontal plane. The measurer sits by the side of the subject and fits the tape snugly but not compressing soft tissues. Circumference is measured to nearest 0.1 cm. For hip circumference the measure is taken around the pelvis at the point of maximal protrusion of the buttocks.

The measurement of overweight and obesity in children and adolescents poses particular problems due to different rates of maturation and growth. Adiposity measures are linked to a child's stage of maturation at the time of measurement and there are two periods when adiposity increases, about the age 5-7 years and in early puberty. Although a fixed cut-off can be used to define obesity in adults, these need to be adjusted for age in childhood (and additionally for maturation in adolescence). Many countries have their own charts for calculating weight, height and obesity for age (eg. Singapore). However recent agreement has been reached on appropriate measures of adiposity which allow classification and comparison. The BMI-for-age chart is recommended. Those greater than the 95th percentile are considered obese, whilst those greater than the 85th percentile of BMI for age are "at risk" (Himes & Dietz 1994, WHO 1995).

Although BMI-for-age charts have been developed for other countries, but many of these are outdated or are only applicable to a narrow age range. However, BMI-for-age charts have recently been developed for Swedish, Italian and British children (Lindgren *et al* 1995, Luciano *et al* 1997, Cole *et al* 1995). The International Obesity Task Force is developing an international standard BMI-for-age chart which should be available in the near future (Dietz *et al* 1999). In this chart the 85th percentile and the 95th percentile roughly correspond to BMIs of 25 kg/m<sup>2</sup> and 30 kg/m<sup>2</sup> respectively in 18 year olds. These are unlikely to be appropriate for Asian and Pacific children; and in any case they artificially confine the prevalence of overweight to 15% and of obesity to 5% of the population.

# Consequences

## 3.1 General mortality and morbidity risk

The obese have an elevated risk from all-cause mortality, with elevated risks of 1.9 being reported among both men and women who were more than 40% of the average weight in a large-scale prospective study of 750 000 individuals (Lew and Garfinkel 1979). Most evidence suggests a J-shaped relationship between BMI and mortality, with the obese having the highest risk. Increased mortality among the obese is evident for several life-threatening diseases including Type 2 diabetes, cardiovascular disease, gallbladder disease, and hormone-sensitive and gastrointestinal cancers. Risks are also higher for some non-fatal conditions such as back pain, arthritis, infertility and, in many westernised countries, poor psychosocial functioning. Approximate relative risks among the obese for several health problems have recently been reported by WHO (Table 3.1) (see Appendix 2 for relative risks in Asian populations).

**Table 3.1. Health risks associated with obesity (WHO 1998)**

Greatly increased (RR <sup>†</sup> >>3)	Moderately increased (RR 2-3)	Mildly increased (RR 1-2)
Type 2 diabetes	Coronary heart disease	Cancer (breast cancer in postmenopausal women, endometrial cancer, colon cancer)
Gallbladder diseases	Hypertension	Reproductive hormone abnormalities
Dyslipidaemia	Osteoarthritis (knees and hips)	Polycystic ovary syndrome
Metabolic Syndrome	Hyperuricaemia and gout	Impaired fertility
Breathlessness		Low back pain
Sleep apnoea		Increased anaesthetic risk
		Foetal defects associated with maternal obesity

<sup>†</sup>Relative risks are approximate

## 3.2 Obesity and cardiovascular risk factors

Several cardiovascular risk factors are associated with obesity including hypertension and dyslipidaemia. Long-term prospective data suggests that obesity may be an independent risk factor for coronary heart disease (CHD) (Willett *et al* 1991). The degree of obesity is related to the rate of development of CHD (Kannel *et al* 1996) and even moderate overweight appears to increase risks of CHD (Willett *et al* 1995). Weight loss accompanies improvements in blood pressure and cholesterol (Kannel *et al* 1996). Many studies investigating the effect of obesity on cardiovascular risk factors are based on Europeans and show increases in risk associated with high BMIs. In Asia however, risks of the same magnitude occur at lower BMIs. In Japan, there is an increase in mortality from CHD for those with a BMI  $\geq 30$  kg/m<sup>2</sup>. If other risk factors such as diabetes, hypertension and hyperlipidaemia are also present, then there is a higher risk of CHD morbidity among those with a BMI between 25 and 29.9 kg/m<sup>2</sup> (Ueshima, personal communication).

### 3.2.1 Hypertension

A positive linear relationship between adiposity and blood pressure is well documented (Stamler *et al* 1978, Dyer & Elliott 1989), with substantial risks of hypertension among the obese being reported. Weight loss is accompanied by a reduction in blood pressure. In Japan, there is a two-fold increase in the risk of hypertension for those with a BMI  $\geq 25$  kg/m<sup>2</sup> compared to those with a BMI of 22 kg/m<sup>2</sup> (Inoue, personal communication). This level of BMI associated with hypertension is much lower than is found in European populations.

It has recently been suggested that the optimal BMI cut-off point to predict hypertension in Hong Kong Chinese is 23.8 kg/m<sup>2</sup> among men and 24.1 kg/m<sup>2</sup> for women (Ko *et al* 1999). Figure 2.1 shows an increased risk of hypertension with increasing BMI in this same sample of Hong Kong Chinese.

### 3.2.2 Dyslipidaemia

Dyslipidaemia is common among the obese and is characterised by raised plasma triglycerides and low density lipoproteins apo B (LDL-apoB) and lower HDL cholesterol concentrations. This metabolic profile is often seen in those with abdominal obesity. An excess of fat in this region is also associated with an increase in small dense low

density lipoprotein (LDL) particles (Despres 1994). In Japan, there is a two-fold increase in the risk of hypertriglyceridaemia, hypercholesterolaemia and of decreased HDL cholesterol in those with a BMI  $\geq 25$  kg/m<sup>2</sup> compared to those with a BMI of 22 kg/m<sup>2</sup> (Inoue, personal communication).

It has recently been suggested that the optimal BMI cut-off point to predict dyslipidaemia in Hong Kong Chinese is 23.0 kg/m<sup>2</sup> among men and 24.1 kg/m<sup>2</sup> for women (Ko *et al* 1999).

### **3.3 Obesity and Type 2 diabetes**

Table 3.2 presents the known modifiable and non-modifiable risk factors or aetiological determinants associated with Type 2 diabetes. The overall risk of Type 2 diabetes must be assessed on the basis of all of these (de Courten *et al* 1997a). Due to the additive effect of different risk factors and determinants, individuals with high levels of non-obesity risk may develop Type 2 diabetes without becoming obese, while in other cases obesity alone may be sufficient to precipitate Type 2 diabetes. Generalised and central obesity are just two of the interrelated risk factors associated with Type 2 diabetes, and of the modifiable lifestyle factors these are probably the most important ones.

In a number of Asia-Pacific populations, Type 2 diabetes is associated with insulin resistance/hyperinsulinaemia, but in Japan and Korea diabetes is more often associated with decreased insulin secretion due to an insulin secretory abnormality (Zimmet 1999). While the cause of most of these cases has not been defined, mutations in the glucokinase and hepatic nuclear factor genes have been demonstrated in maturity-onset diabetes of the young (MODY) families in Japan, Korea and Chinese in Hong Kong. Many of these cases are not obese by the classification suggested in this report. It has recently been suggested that the optimal BMI cut-off point to predict Type 2 diabetes in Hong Kong Chinese is 24.3 kg/m<sup>2</sup> among men and 23.2 kg/m<sup>2</sup> for women (Ko *et al* 1999).

**Table 3.2. Aetiological determinants and risk factors for Type 2 diabetes in Asia**

---

A. Genetic factors
<i>Genetic markers, family history, "Thrifty Genotype" etc</i>
B. Demographic characteristics
<i>Sex, age, ethnicity</i>
C. Modifiable (including behavioural and lifestyle related) risk factors
<i>Obesity (including distribution and duration of obesity)</i>
<i>Physical inactivity</i>
<i>Diet</i>
<i>Urbanisation, modernisation, industrialisation</i>
<i>Intrauterine environment, low birth weight</i>

---

### **3.4 The Metabolic Syndrome**

The common occurrence of Type 2 diabetes or impaired glucose tolerance, with other CVD risk factors such as dyslipidaemia, hypertension and central obesity or with CVD mortality, is well recognised. The various combinations of these metabolic and morphologic risk factors and diseases are generally known as the "Metabolic Syndrome". This is one of the major health problems associated with obesity in the Asia-Pacific region.

While the main focus has been on insulin resistance and/or hyperinsulinaemia as the common aetiological factor/s for the components of the Metabolic Syndrome, there is considerable heterogeneity in these relationships between populations (de Courten *et al* 1997b, Zimmet 1999).

The real question is whether there is a single aetiological factor for the CVD risk factor cluster. Factor analyses suggest that there is no single central aetiological factor. It seems most likely that there are several underlying abnormalities which may have a genetic basis. Insulin resistance and hyperinsulinaemia can be implicated in the aetiology of glucose intolerance, dyslipidaemia and obesity (Zimmet *et al* 1994). The evidence for hyperinsulinaemia playing a role in hypertension is not consistently supported by epidemiological data (Zimmet 1995).

The recent report from WHO, *Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications - Part 1: Diagnosis and Classification of Diabetes Mellitus* (WHO 1999) has highlighted the need for a consistent definition of the Metabolic Syndrome and it has suggested parameters. While these criteria may change eventually as new prospective data become available in the Asia-Pacific region, this initiative provides a basis for the development of a standardised definition allowing international comparisons of prevalence, incidence and natural history as previously there was no internationally agreed definition.

The WHO consultation has recommended the following definition (Table 3.3, see Appendix 4 for detailed description) which does not imply causal relationships and is suggested as a working definition to be improved upon in due course.

**Table 3.3. Components of the metabolic syndrome**

---

*Glucose intolerance, impaired glucose tolerance (IGT) or diabetes mellitus and/or insulin resistance together with two or more of the following:*

*Raised arterial pressure*

*Raised plasma triglycerides*

*Central obesity*

*Microalbuminuria*

---

Other components have been suggested for the Metabolic Syndrome eg. hyperuricaemia, raised fibrinogen and plasminogen activator inhibitor-1 (PAI-1), cigarette smoking etc, but the WHO report states they are not necessary for the recognition of the condition (WHO 1999). Clearly in this respect, internationally agreed criteria for central obesity, insulin resistance and hyperinsulinaemia would be very useful.

## **3.5**

### **Stroke**

There is an increase in mortality of cerebrovascular disease in Japan in those with a BMI  $\geq 30$  kg/m<sup>2</sup>. However, an increased risk of stroke morbidity is likely in those with a BMI between 25-29.9 kg/m<sup>2</sup> in the presence of other conditions such as Type 2 diabetes, hypertension and hyperlipidaemia (Matsumura & Tsubono 1998).

### **3.6**

#### **Female reproductive health**

Obesity is related to several gynaecological disorders including polycystic ovary syndrome (PCOS) (Yen 1980, Pettigrew & Hamilton-Fairley 1997), infertility (Zaadstra *et al* 1993, Pettigrew & Hamilton-Fairley 1997) and general menstrual disorders (Rich-Edwards *et al* 1994, Lake *et al* 1997, Hartz *et al* 1979). Poor pregnancy outcome and higher rates of miscarriage have also been found among the obese. Elevated risks may not be confined to those acquiring their overweight in adulthood but may independently be related to fatness in childhood or adolescence (Rich-Edwards *et al* 1994, Hartz *et al* 1979, Lake *et al* 1997).

It has also been suggested that abdominal obesity may be a particularly important risk factor for menstrual disorders (Hartz *et al* 1984) and infertility (Zaadstra *et al* 1993), in that such localised fatness may increase androgenicity (Seidell *et al* 1990). Weight loss may be particularly beneficial for obese women with PCOS, in that it improves the associated hormonal and menstrual abnormalities (Kiddy *et al* 1992).

At the menopause women gain abdominal adiposity (increased waist circumference). Although this may be prevented by Hormone Replacement Therapy (HRT) in some women, others report weight gain with HRT.

### **3.7**

#### **Respiratory function**

It has been proposed that obesity has an adverse effect on respiratory function and increases the risk of respiratory symptoms. Sleep apnoea is a common problem among obese men and women (Millman *et al* 1995, Young *et al* 1993). In particular, abdominal obesity and neck size are related to obstructive sleep apnoea, this may be explained by narrowing of the upper airway in the recumbent position, and in severe cases can result in sudden death.

Other respiratory problems associated with obesity include pulmonary hypertension, Pickwickian Syndrome and increased post-operative risk.

### **3.8**

#### **Musculo-skeletal disorders**

Elevated risk of back pain have been observed in relation to obesity, particularly among women (Deyo & Bass 1989, Han *et al* 1997, Garzillo & Garzillo 1994). Back pain may lead to a reduction in physical activity and hence an increase in adiposity, alternatively,

obesity may increase the mechanical load on the spine, thereby increasing the risk for back pain. This may lead to altered gait, thus reducing efficiency of shock absorbance and increasing stress on the spine. Generally, musculo-skeletal problems are more common in obese and ageing populations.

There is some evidence to suggest that obesity is associated with development of osteoarthritis (OA) and gout. While it seems reasonable to consider obesity contributing to pain of weight-bearing joints, an increased risk of osteoarthritis of the hand among the obese has also been reported (Carman *et al* 1994). The increased risk of gout associated with obesity may be related to accompanying hyperuricaemia, and abdominal obesity may play an important role. These problems are particularly common in Polynesian populations (Zimmet *et al* 1978) and in Japanese Sumo-wrestlers. In the Japanese, it has been demonstrated that serum uric acid increases as BMI increases (Hosoya *et al* 1998).

### **3.9** **Cancer**

In the American Cancer Society prospective study of 750 000 participants, risk of cancer among the obese (> 140% of average weight) was 1.33 and 1.55 for men and women respectively (Lew & Garfinkel 1979). In this study, the principal cancer sites of excess mortality associated with obesity were cancer of the colon and rectum among men, and gall bladder, biliary passages, breast, cervix, endometrium, uterus and ovary among women.

Some studies report higher risk of colon cancer among the obese, but this is not a consistent finding. Lew and Garfinkel 1979 reported a 1.73 risk among obese men but no association was found among women. A recent review provided insufficient evidence to support an increased risk of colon cancer associated with obesity (Shike 1996), however the elevated risk of colon cancer may be related to an increased consumption of energy from fat or a reduction in physical activity.

In the Asia-Pacific region, only weak correlations between obesity and cancer have been observed.

### **3.10** **Gastrointestinal diseases**

Gallbladder disease is the most common form of digestive disease among the obese. The increased cholesterol levels excreted in the bile as obesity develops appear to

be linked to the development of gall stones. Weight loss may also exacerbate gall bladder disease (Stampfer *et al* 1992).

Liver abnormalities, in particular fatty infiltration, are often found in the obese. Associated abnormal liver enzymes may improve with weight loss.

### **3.11 Psychosocial problems**

In Westernised countries obesity, particularly among women and children, is often confronted with social prejudice and discrimination (Staffieri 1967, Gortmaker *et al* 1993). Prejudices have also been experienced in some Asia-Pacific countries. However, in some regions such as the Pacific Islands, obesity is still considered desirable and a symbol of wealth and higher social standing (Dowse *et al* 1995). As the obesity rates in these countries continues to increase, these populations may become increasingly affected by cultural values more common in industrialised countries. A recent study conducted in the Cook Islands demonstrated a substantial westernisation of body size perception (Craig *et al* 1996). Thus these adverse psychosocial problems found in Westernised countries may become more widespread in the Asia-Pacific region.

### **3.12 Consequences of childhood/adolescent obesity**

Childhood obesity can lead to a number of chronic diseases including impaired glucose metabolism, insulin resistance, Type 2 diabetes in adolescence, hypertension, dyslipidaemia, hepatic steatosis, gastrointestinal disturbances, obstructive sleep apnoea and PCOS. In particular, childhood obesity in the Asia-Pacific region is associated with the development of Type 2 diabetes at a younger age. Coronary risk factors measured in children and young adults appear to be associated with early development of calcification of coronary arteries (Mahoney *et al* 1996). Numerous studies have shown a tendency for obese children to remain obese in adulthood (Abraham *et al* 1971, Abraham & Nordsieck 1960, Guo *et al* 1994, Sorensen & Sonne-Holme 1988) thus increasing the risk of obesity-related disorders later in life.

Psychosocial problems can also be a problem in children with obesity as their weight can be seen by them and others as a significant handicap. It has been suggested that adults who have been obese since childhood are more likely to suffer from psychological disturbances and that adolescence may be the period of greatest risk.

# 4 Economic Costs

The costs of obesity to a community and individuals may be divided into the *direct* costs to the health system and the *indirect* or *social* costs to the individual and community (eg sick days, individuals expenditure on weight loss). The direct costs depend in the main part on the diseases caused by obesity and the cost of these diseases. One of the problems in this assessment is the relative risk of the disease in different communities and ethnic groups. Several methods have been used to calculate these costs and there have been a wide range of results on the cost of obesity in different countries. Some diseases which have been included in the calculations are Type 2 diabetes, heart disease, hypertension, endometrial cancer, arthritis and colorectal cancer. Table 4.1 shows the direct costs of obesity for several countries. Indirect costs also vary widely. One of the latest estimates in the United States for indirect costs was US\$47.6 billion per year. Little data is yet available for the Asia-Pacific region.

It is necessary to develop a standard way of calculating the costs of obesity so that various countries health expenditure can be compared and for the benefits of treatment to be calculated. For these reasons, suggested standard formulae are being prepared.

**Table 4.1. Direct costs of obesity**

Country	Year	Population (millions)	Cost (per year)
<i>New Zealand</i>	<i>1996</i>	<i>3.6</i>	<i>NZ \$135 million</i>
<i>Australia</i>	<i>1994</i>	<i>18.4</i>	<i>AUD \$464 million</i>
<i>Netherlands</i>	<i>1995</i>	<i>15.7</i>	<i>NG 1 billion</i>
<i>France</i>	<i>1995</i>	<i>58.0</i>	<i>FF 12 billion</i>
<i>USA</i>	<i>1998</i>	<i>274.0</i>	<i>US \$51.6 billion</i>

# 5 Prevention

There have been efforts by many governments in the Asia-Pacific region to initiate healthy lifestyle and universal prevention programmes. Such programmes, which include strategies targeting children, have the potential to prevent obesity from increasing overall in a population.

Generally, there are three approaches to prevention. Universal prevention is based on a total population approach whereas selective and targeted prevention strategies are directed at high risk groups. The latter two approaches therefore require screening of individuals in appropriate settings such as schools, to identify subjects and subgroups at high risk.

Relevant to the Asia-Pacific region is the recent proposal by Egger and Swinburn (1999) of an ecological model for understanding overweight and obesity. It potentially represents an important new paradigm for understanding obesity as “normal physiology within a pathological environment” and signposts the directions for a wider public health approach to the obesity pandemic. They suggest that the increase in the prevalence of obesity is primarily due to the increasingly obesogenic environment rather than ‘pathology’ in metabolic defects or genetic mutations within individuals. Thus, interventions aimed at creating environments that facilitate and promote behavioural changes in terms of diet and exercise are important in the prevention of obesity.

**Figure 5.1. Different levels of prevention**



## **5.1** Universal prevention

Measures directed at the whole population should aim to stabilise the level of obesity and eventually lower the incidence and hence the prevalence of obesity. A reduction in weight-related disease by lifestyle modification including improved diet and physical activity levels are objectives, as well as a reduction in smoking and alcohol consumption.

## **5.2** Selective prevention

Selective prevention aims to educate sub-groups of the population with a high-risk of obesity (Table 5.1) so that they can deal effectively with the risk factors, which may be genetic and which predispose them to obesity (WHO 1998). Such strategies can be initiated in appropriate settings which allow access to these high-risk groups, including schools, community centres and primary care venues. In Singapore such an approach, targeted at school children, has reduced the prevalence of obesity from 15% to 12.5% over 5 years (Ministry of Education, Singapore, 1996).

## **5.3** Targeted prevention

Targeted prevention aims to prevent weight gain and reduce the number of people with weight-related disorders in those individuals that are already overweight or those with biological markers associated with excess adiposity, who are not yet obese (WHO 1998). This group have a particularly high risk of developing obesity and obesity-related disorders (Table 5.1). Individuals who have some existing weight-related problems and those with a high risk of developing obesity co-morbidities such as cardiovascular disease and Type 2 diabetes should be a key priority in this prevention strategy.

**Table 5.1. Sub-groups at high risk from obesity and appropriate prevention strategies**

	Parameter/ measurement	Family history	Other factors
Selective prevention (population groups):	<i>BMI 23 kg/m<sup>2</sup> in Asian populations</i>	<i>Obesity</i>	<i>Smoking cessation</i>
	<i>BMI 25 kg/m<sup>2</sup> in European populations</i>	<i>Type 2 diabetes</i>	<i>Low birth weight</i>
	<i>BMI 26 kg/m<sup>2</sup> in Pacific Islanders*</i>	<i>Hypertension</i>	<i>Sedentary occupation</i>
		<i>Dyslipidaemia</i>	<i>Some ethnic groups eg. Polynesians, Asian Indians, Australian Aborigines</i>
			<i>Minority groups</i>
Targeted prevention (individuals):	<i>BMI 25 kg/m<sup>2</sup> in Asian populations</i>		<i>Type 2 diabetes</i>
	<i>BMI 30 kg/m<sup>2</sup> in European populations</i>		
	<i>BMI 32 kg/m<sup>2</sup> in Pacific Islanders**</i>		
	<i>Waist circumference &gt; 90 cm (men), &gt; 80 cm (women) in Asian populations</i>		
	<i>Waist circumference &gt; 94 cm (men), &gt; 80 cm (women) in European populations</i>		

\* Proposed BMI 26 kg/m<sup>2</sup> in Pacific Island populations (Swinburn 1998)

\*\*Proposed BMI 32 kg/m<sup>2</sup> in Pacific Island populations (Swinburn 1998)

# Treatment Outline

## 6.1 Goals for obesity therapy

The achievement of weight normalisation is often unrealistic and does not have to be the ultimate goal of a weight-reduction strategy. Moderate weight loss can have substantial health benefits. Table 6.1 summarises criteria for the successful treatment of obesity.

**Table 6.1. Long-term goals for obesity therapy**

Criteria	Treatment success
<i>Reduction of excess weight</i>	<i>5-6kg or 10% of initial body weight</i>
<i>Maintenance of BMI</i>	<i>&lt; 23 kg/m<sup>2</sup><sup>†</sup></i>
<i>Blood pressure</i>	<i>any reduction</i>
<i>Blood glucose</i>	<i>any reduction</i>
<i>Glycaemic control</i>	<i>any improvement</i>
<i>Other risk factors</i>	<i>any reduction</i>

<sup>†</sup> For Asian populations. BMI cut-off will be higher in Pacific Islanders.

## 6.2 Lifestyle approaches

Weight management strategies should include modification of diet and physical activity, and of daily habits and thoughts. Specific behaviours conducive to overeating or under-activity need to be identified and corrected. Weight loss is more likely to be achieved and maintained by behaviour modification techniques that focus on lifestyle and attitude. This is discussed in more detail elsewhere (WHO 1998). Many countries will have community lifestyle programs in place for other chronic diseases.

### 6.3

## Overall approach to treatment

**Table 6.2. Treatment options for different levels of BMI and other risk factors in Asian populations**

	Diet	Activity	Drug	VLCD	Surgery
<b>BMI 23-25 kg/m<sup>2</sup>:</b>					
No additional risk	✓	✓	X		
Increased WC <sup>†</sup>	✓	✓	X		
DM/CHD/HT/HL*	✓	✓	✓		
<b>BMI 25-30 kg/m<sup>2</sup>:</b>					
No additional risk	✓	✓	✓ (consider)		
Increased WC	✓	✓	✓ (consider)		
DM/CHD/HT/HL	✓	✓	✓		
<b>BMI &gt; 30 kg/m<sup>2</sup>:</b>					
No additional risk	✓	✓	✓ (consider)	✓ (consider)	✓ (consider)
Increased WC	✓	✓	✓	✓ in severely	✓ in severely
DM/CHD/HT/HL	✓ (intensive)	✓ (intensive)	✓	✓ obese)	✓ obese)

KEY: DM: Type 2 diabetes CHD: coronary heart disease HT: hypertension HL: hyperlipidaemia

✓ = yes X = no

<sup>†</sup>Waist circumference > 90 cm (men), > 80 cm (women)

\*Specific therapies relating to the risk factor or condition (DM, CHD, HT, HL) may be necessary

NOTE: If two or more diseases present eg. hypertension, then an anti-obesity drug may be used.

### 6.4

## Dietary measures

**Table 6.3. Principles of nutrition related to obesity**

Food selection should be guided by available foods which vary from country to country in the Asia-Pacific region.

Distribution of food intake should be as even as possible throughout the day and meals should not be "skipped" as a weight control method.

Meals should be adequately sized so that snacks are not needed between meals.

20-30%<sup>†</sup> or less of the total dietary energy should be from fats and oils.

Carbohydrates should account for 55-65% of total energy.

Protein should not exceed 15% of total energy.

Fresh fruits, vegetables and wholegrain foods should be encouraged.

Alcohol intake should be restricted.

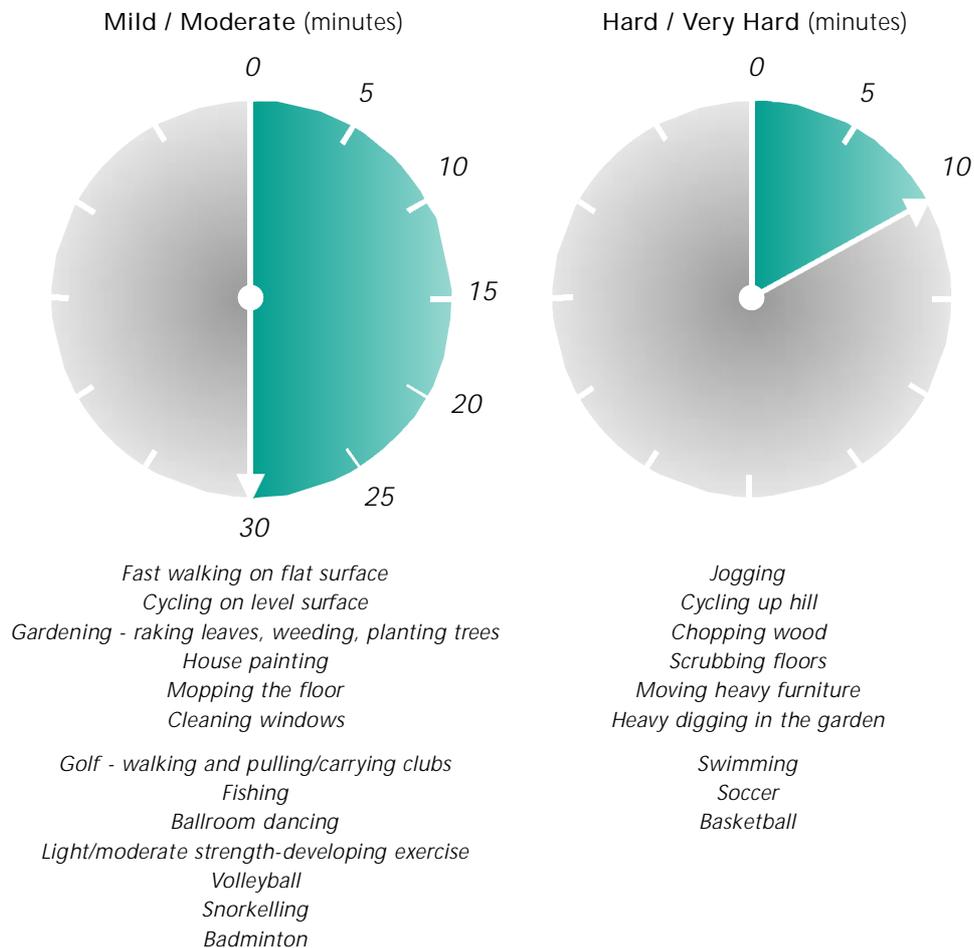
<sup>†</sup> Depends on a country's current fat intake eg. some Asian countries may need to lower fat intake to 20% but in some Pacific Islands, 30% may be sufficient.

Many countries have dietary guidelines based on local experience and data. In some parts of Asia, diets are high in carbohydrates and fat, and low in protein. Modifications need to be made to reduce the total calories by lowering the intake of highly refined carbohydrates and replacing these with complex carbohydrates, and reducing fat intake. The general nutrition principles outlined in Table 6.3 provide a basis for dietary modification approaches in universal prevention strategies.

All weight management strategies need to educate patients about food and healthy eating habits. Dietary therapy essentially involves instructing patients on changing their diet in order to restrict caloric intake. Diets based on healthy eating principles, such as the individualised energy-deficit diet (fixed energy diet) and the *ad libitum* low fat, high carbohydrate diet have better outcomes. Patients should avoid 'fad' diets.

It should be noted that while non-nutritive sweeteners may be used, fat-replacement foods such as Olestra are not generally available in the region and are not advised at present.

**Figure 6.1. Types and levels of physical activity**



## **6.5** Physical activity

Another important factor in a successful weight management programme is increasing daily physical activity. If physical activity or exercise is used alone to treat obesity, only a moderate weight loss of between 4-5 kg over a three month period is likely.

Physical activity advice should be tailored for age and cultural climate and emphasise increased daily activity such as walking and climbing stairs. It is not necessary for the obese patient to participate in strenuous activity, low to medium intensity is sufficient. Figure 6.1 provides examples of different activities and levels of intensity.

## **6.6** Pharmacotherapy

In certain cases pharmacological treatment may need to be considered in addition to diet, exercise and behaviour modification (see Table 6.2). To date, there is little published scientific evidence reporting the long-term safety and efficacy of currently available anti-obesity drugs, thus no particular drug can be recommended for routine use. There is also no available data on the effects of combining two or more types of these anti-obesity agents.

Pharmacotherapy should be considered as part of a long-term management strategy for obesity that is specific to a given patient. A patient may require drug therapy to aid compliance with dietary restriction to augment diet related weight loss, and to achieve weight maintenance after satisfactory weight loss. The risks to a patient from continuing obesity need to be balanced against the risks from therapy, and prescribers need to be aware of possible side-effects. In many patients requiring pharmacotherapy, long-term treatment is necessary. Using the analogy of hypertension, obesity is a lifelong condition and necessitates chronic treatment.

Pharmacotherapy should be used as an adjunct to diet and exercise. Use of drug therapy should be considered when:

- ▶ Hunger or overt hyperphagia are recognised factors contributing to weight gain
- ▶ There are associated co-morbidities including impaired glucose intolerance, dyslipidaemia and hypertension
- ▶ Symptomatic complications of obesity exist such as severe osteoarthritis, obstructive sleep apnoea, reflux oesophagitis, and the compartment syndrome

### 6.6.1 Types of anti-obesity drugs

Anti-obesity drugs can be classified into two broad groups, (i) those acting on the central nervous system to influence appetite and (ii) those acting on the gastrointestinal system to reduce absorption. Drugs currently available or under development, and their side-effects are listed in Appendix 5.

#### (i) Drugs acting on the central nervous system

##### — *Drugs acting via serotonergic (5HT) pathways*

*Fenfluramine* and *Dexfenfluramine* were effective weight reduction drugs but have been withdrawn worldwide because of the drug related complication of primary pulmonary hypertension and hypertrophic cardiac valvular lesions.

*Fluoxetine*, a serotonergic anti-depressant, has modest effects on appetite and weight, and can be used as a surrogate anorectic agent especially in depressed obese patients.

##### — *Drugs acting via noradrenergic pathways*

*Ephedrine* and *Caffeine* primarily have anorectic properties although some thermogenic effects have also been demonstrated. Over a one year period, *Astrup et al* (1992) showed that among obese patients on a restricted diet, a significantly greater weight loss was found among those who were also treated with the ephedrine/caffeine combination, compared to those treated with either a placebo or caffeine or ephedrine separately.

*Phentermine* and *Diethylpropion* are amphetamine derivatives that are effective at suppressing appetite and reducing weight loss. Because of their stimulant action on the central nervous system, only short term use, for 3 months or less, is recommended.

##### — *Drugs acting via serotonergic and noradrenergic pathways*

*Sibutramine* is a serotonin and noradrenaline re-uptake inhibitor that enhances post-ingestive satiety and increases resting metabolic rate in animals. In addition, thermogenesis increases leading to greater energy expenditure. Recent 12 month clinical trials have shown maximum weight loss is achieved by 6 months with a significant weight loss being maintained over 12 months (eg. 4.4-6.3 kg weight loss (dose dependent) in

Sibutramine-treated patients compared to a 1.6 kg loss in placebo-treated patients). The weight loss is accompanied by a reduction in WHR and improved blood lipids and glycaemic control (Griffiths *et al* 1995). Although side-effects are generally minimal, there have been reports of increased blood pressure and heart rates in some patients. However, longer term trials show that a reduction in blood pressure accompanies weight loss in patients treated with Sibutramine (Lean 1997).

#### (ii) Drugs acting on the gastrointestinal system

*Metformin* may be useful in managing obesity in those with Type 2 diabetes, PCOS, and potentially in those with impaired glucose tolerance. In some cases it may be used to treat obesity alone. Care should be taken with its use in subjects with cardiac decompensation and renal or hepatic diseases as it may result in lactic acidosis.

*Orlistat* is a pancreatic lipase inhibitor which produces a dose-dependent reduction in dietary fat absorption. Pooled data from five clinical trials indicated that the overall mean weight loss from randomisation to the end of six months and one year of treatment in the intent-to-treat population was 5.6 kgs and 6.5 kgs in the patients treated with Orlistat and 2.8 kgs and 2.6 kgs in the placebo-treated patients, respectively. One possible adverse effect of Orlistat is the malabsorption of fat-soluble vitamins, thus it is recommended that for long-term use patients also eat a diet rich in fruit and vegetables. Currently the manufacturers do not recommend multi-vitamin supplements. An increased risk of breast cancer among Orlistat users has been found in some clinical trials although this has not been substantiated in later studies.

### 6.6.2 Drugs not appropriate for the treatment of obesity

There is no evidence that any alternative therapies/proprietary medicines such as cellulite treatments, herbal preparations or fibre capsules are effective. Adequate clinical trials have not been performed.

Diuretics, laxatives, human chorionic gonadotrophin (HCG) are ineffective and should not be used. Treatment with amphetamine, dexamphetamine and thyroxine may be dangerous and these agents must not be used to achieve weight loss.

The anti-obesity agents fenfluramine and D-fenfluramine were withdrawn from the market in 1997 following evidence of a possible link with cardiac valvular disorders, and are therefore no longer available.

### 6.6.3

## Contra-indications to the use of anti-obesity drugs

Anti-obesity agents are not recommended for certain sub-groups listed in Table 6.6. Monoamine oxidase inhibitors should not be used at all with centrally-acting anti-obesity drugs.

**Table 6.6. Specific groups in which anti-obesity drugs are not recommended**

---

*Children*

*Patients who have previously suffered adverse effects from drugs in this category*

*Pregnant and lactating women*

*Patients who are concurrently taking other selective serotonin re-uptake inhibitors (SSRIs)*

---

### 6.7

## Very low calorie diets (VLCD)

Although severe dietary restriction using “food diets” can achieve weight reduction, the phrase VLCD generally refers to commercially available package diets that provide daily requirements of first grade protein, vitamins and essential minerals, together with sufficient carbohydrate to prevent electrolyte loss (‘Modifast’, ‘Optifast’).

VLCDs usually allow between 400-800 calories/day and can induce an immediate weight loss of 1.0-1.5 kg per week that is predominantly fat loss with minimisation of negative nitrogen balance. However, they do not change eating habits or result in weight loss over the long-term. Although weight loss is rapid on VLCDs, in the short-term, weight gain is common on ceasing the regimen, emphasising the importance of behavioural and lifestyle changes in achieving maintenance of weight loss. Before VLCDs are used individuals should have a trial of healthy eating and increased physical activity.

VLCDs are effective weight loss treatments and may be used in those with severe obesity or who have another medical reason for needing rapid weight loss. In clinical trials, weight loss due to low calorie diets was just as effective as VLCDs after a one year period (Wadden *et al* 1994, Richman *et al* 1992). Although they are effective therapy, they should only be used under medical supervision and with a weight management and weight maintenance program.

## 6.8

### Surgery

Gastric-partition surgery can be an effective therapy to treat those with BMI  $\geq 40$  kg/m<sup>2</sup>. The creation of a small volume proximal stomach pouch of  $\approx 80$  ml results in early satiety via vagus nerve afferents. Exceeding the volume of the proximal pouch results in vomiting. Such surgery is not standard treatment in the Asia-Pacific region. However, there may be individuals who require it and they should be treated in a specialised centre with long term follow-up. Current surgical methods include gastric banding or stapling, laparoscopic placement of an adjustable inflatable band and gastro-jejunal bypass surgery. Interim results from the Swedish Obesity Study indicate weight loss of the order of 35-55 kg, maintained for 4 years, with greatly improved quality of life measures. Liposuction is not a treatment for generalised obesity, but may be used for unsightly local collections of fat or for a specific medical purpose.

## 6.9

### Management of obesity in children

Management of obesity in children differs from that in adults in that the prevention of weight gain is of importance rather than focusing on weight loss. Lean body mass increases as children get older, thus keeping fat mass constant will eventually help to normalise body weight. The best and most effective way to treat children with obesity is to treat the family, and not the child alone, by encouraging increased daily activity and healthy eating habits.

Higher energy expenditure can be achieved more effectively through increased general activity in schools and play rather than competitive or structured sports. In developed countries, the recent increase in the prevalence of childhood obesity appears to be related to the time spent in sedentary behaviour associated with non-active leisure pursuits. Television viewing, computer and video games are principal sources of inactivity among children.

The role of pharmacotherapy in children and adolescents is undefined but may be considered in extreme cases. There has been little or no efficacy or outcomes data on pharmacotherapy in children  $< 18$  years.

In the diets of obese children, only small reductions in energy-intake should be made so that sufficient energy and nutrients are available to ensure normal growth and development. VLCDs are not generally recommended in childhood.

# 7 Risk Assessment

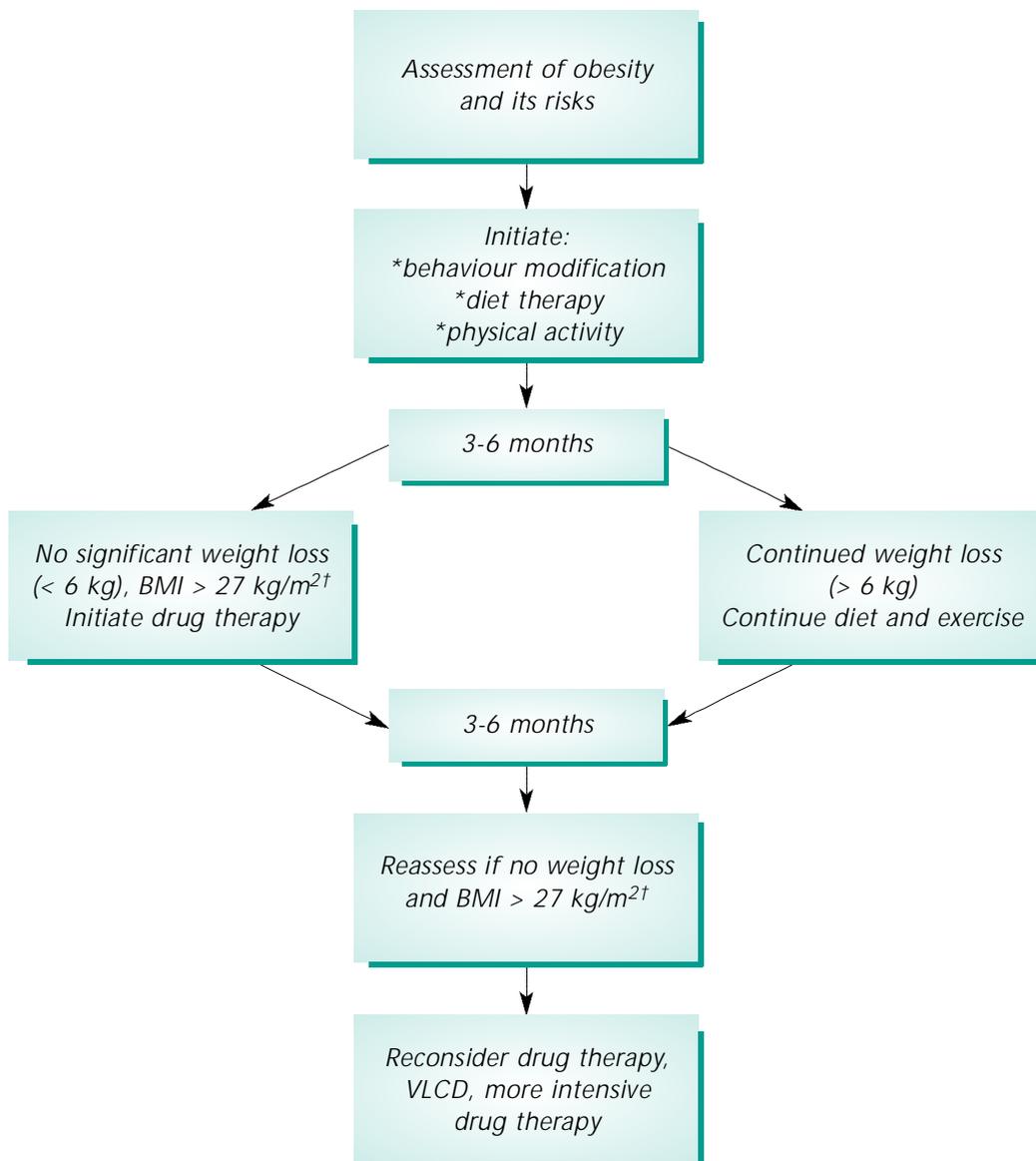
All those who are obese should have a full medical assessment and this should include routine evaluation of co-morbidity including impaired glucose tolerance or diabetes, hypertension and dyslipidaemia. For those who are older than 40 years, or who have a history of heart disease, a cardiovascular examination may be necessary prior to exercise prescription. It is important to check for secondary causes of obesity, which include Cushing's Syndrome, hypogonadism, hypothyroidism and certain genetic disorders. Such causes are rare given the prevalence of obesity. In cases of primary hypogonadism specific therapy with testosterone is of benefit. It should be noted that in obese males, blood testosterone levels may be low secondary to obesity and in this situation treatment with testosterone is not appropriate.

Symptomatic complications of severe obesity should be treated actively regardless of whether the patient is losing weight. These include symptoms of obstructive sleep apnoea, osteoarthritis, reflux oesophagitis, gravitational oedema and other manifestations of the abdominal compartment syndrome.

# Clinical Monitoring Outline

Weight management programmes should include clearly defined procedures for monitoring progress. Once a weight loss target has been achieved new targets should be set. Figure 8.1 presents the general procedure which should be followed for the management of obesity in the Asia-Pacific region.

**Figure 8.1.** Procedure to be followed for managing obesity in the Asia-Pacific region



**NOTE:** Patients on anti-obesity agents should be reviewed regularly to ensure the weight loss benefit continues. With such agents, patients should lose 1.5-2.0 kg in the first month of treatment, otherwise they are likely to be non-responders.

† For Asian populations. BMI cut-off will be higher for Pacific Islanders.

# Future Research

The following research areas are identified as being of particular importance or need in the Asia-Pacific region.

## 9.1      **Assessment of Obesity**

**1**      If possible, countries in the region need to perform standardised studies which will determine the prevalence of obesity, and its co-morbidities such as Type 2 diabetes, hypertension and dyslipidaemia. These prospective epidemiological studies need to be repeated on the same populations to determine the relative risk of developing these co-morbidities with obesity. These will indicate both prevalence and secular trends.

**2**      Studies need to be performed in both Asian and Pacific Island countries to determine the relationship between BMI, waist circumference and risk of development of co-morbidities eg. diabetes, hyperlipidaemia and hypertension. This will allow validated cut-points of BMI to be established.

**3**      Body composition studies need to be performed to determine whether Asian populations have equivalent levels of fatness for body size and BMI, and whether Asians preferentially deposit abdominal fat.

**4**      The above studies will enable the development of waist circumference cut-offs to classify abdominal obesity on the same basis as those for BMI and should include the effectiveness of other simple clinical measures of obesity for determining risk in various populations eg. waist to height, waist to sitting height, waist circumference and waist to hip ratio.

- 5** Studies which investigate the attitudes to obesity in different populations in the Asia-Pacific region are lacking and are needed as the results of such studies will guide planned interventions.
  
- 6** It is important that studies which determine the safety and efficacy of various therapeutic and preventative approaches in these populations be performed. These studies should include the development of effective behavioural strategies which can be utilised in this region.
  
- 7** Together with this, intervention studies on the prevention of obesity-related chronic diseases are needed. These might be either population interventions or targeted interventions.

## References

- Abraham S, Collins G, Nordsieck M. Relationship of childhood weight status to morbidity in adults. *HSMHA Health Report* 1971; 86: 273-284.
- Abraham S, Nordsieck M. Relationship of excess weight in children and adults. *Public Health Reports US* 1960; 75(3): 263-273.
- Amos A, McCarty D, Zimmet P. The rising global burden of diabetes and its complications: Estimates and projections to the year 2010. *Diabetic Med* 1997;14 (Suppl 5): S1-S85.
- Astrup A, Breum L, Toubro S, Hein P, Quaade F. The effect and safety of an ephedrine/caffeine compound compared to ephedrine, caffeine and placebo in obese subjects on an energy-restricted diet. *Int J Obes* 1992; 16 (4): 269-77.
- Carman WJ, Sowers M, Hawthorne VM, Weissfeld LA. Obesity as a risk factor for osteoarthritis of the hand and wrist: a prospective study. *Am J Epidemiol* 1994;139(2):119-29.
- Chen J. Institute of Sports Medicine, Beijing Medical College, Haidian District, Beijing, China.
- Cole TJ, Freeman JV, Preece MA. Body mass index reference curves for the UK, 1990. *Arch Dis Child* 1995 1995, 73: 25-29.
- Craig PL, Swinburn BA, Matenga-Smith T, Matangi H, Vaughn G. Do Polynesians still believe that big is beautiful? Comparison of body size perceptions and preferences of Cook Islands, Maori and Australians. *NZ Med J* 1996; 109 (1023): 200-3.
- de Courten M, Bennett PH, Tuomilehto, Zimmet P. Epidemiology of NIDDM in non-Europids. In: Alberti KGMM, Zimmet P, DeFronzo RA, Keen H (eds) In: *Textbook of Diabetes Mellitus*: Second Edition. John Wiley & Sons 1997a: 143-170.
- de Courten M, Zimmet P, Hodge A, Collins V, Nicholson M, Staten M, Dowse G, Alberti KGMM. Hyperleptinaemia: the missing link in the Metabolic Syndrome? *Diabetic Med* 1997b; 14: 200-108.
- Despres J-P. Dyslipidaemia and obesity. *Clinical Endocrinology and Metabolism* 1994; 8: 629-60.
- Deurenberg-Yap M, Yian TB, Kai CS, Deurenberg P, van Staveren WA. Manifestation of cardiovascular risk factors at low levels of body mass index and waist-to-hip ratio in Singaporean Chinese. *Asia Pacific J Clin Nutr* 1999; 8(3): 177-183.
- Deyo RA, Bass JE. Lifestyle and low back pain: the influence of smoking and obesity. *Spine* 1989; 14: 501-506.
- Dietz WH, Bellizzi MC. Introduction: the use of body mass index to assess obesity in children. *Am J Clin Nutr* 1999; 70(1): 123S-5S.
- Dowse GK, Hodge AM, Zimmet PZ (1995). Paradise lost: obesity and diabetes in Pacific and Indian Ocean populations. In: *Progress in Obesity Research* 1994, eds. A Angel, et al, London: John Libbey & Co.
- Dyer AP, Elliot P. The INTERSALT study: relations of body mass index to blood pressure. INTERSALT Co-operative Research Group. *J Hum Hypertens* 1989; 3:299-308.
- Egger G, Swinburn B. An 'ecological' approach to the obesity pandemic. *Br Med J* 1997; 315: 477-80.
- Freeman J, Power C, Rodgers B. Weight for height indices of adiposity in childhood and early adult life. *Int J Epidemiol* 1995; 24: 970-976.
- Garzillo MJD, Garzillo TAF. Does obesity cause low back pain? *Journal of Manipulative and Physiological Therapeutics* 1994; 17: 601-104.
- Ge L. Body mass index in young Chinese adults. *Asia Pacific J Clin Nutr* 1997, 6(3): 175-179.

- Griffiths J, Brynes AE, Frist G, Bloom SR, Finer N, Jones SP, Romanec FM. Sibutramine in the treatment of overweight non-insulin dependent diabetics. *Int J Obes* 1995; 19 (Suppl 2): 41.
- 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: 1008-12.
- Guo SS, Roche AF, Chumlea WC, Gardner JD, Siervogel RM. The predictive value of childhood body mass index values for overweight at age 35y. *Am J Clin Nutr* 1994; 59: 810-19.
- Hales CN, Barker DJP. Type 2 (non-insulin dependent) diabetes mellitus: the thrifty phenotype hypothesis. *Diabetologia* 1992, 35: 595-601.
- Han TS, van Leer EM, Seidell JC, Lean MEJ. Waist circumference action levels in the identification of cardiovascular risk factors: prevalence study in a random sample. *Br Med J* 1995, 311: 1401- 1405.
- Han TS, Schouten JSAG, Lean MEJ, Seidell JC. The prevalence of low back pain and associations with body fatness, fat distribution and height. *Int J Obes* 1997; 21: 600-607.
- Hartz AJ, Barboriack PN, Wong A, Katayama KP, Rimm AA. The association of obesity and infertility and related menstrual abnormalities in women. *Proc Nutr Soc* 1979; 3: 57-73.
- Hartz A, Rupley D, Rimm A. The association of girth measurements with disease in 32,856 women. *Am J Epidemiol* 1984; 119:71-80.
- Himes J, Dietz W. Guidelines for overweight in adolescent preventative services: recommendations from an expert committee. *Am J Clin Nutr* 1994; 59: 307-16.
- Hodge AM, Dowse GK, Toelupe P, Collins VR, Imo T, Zimmet, PZ. Dramatic increase in the prevalence of obesity in Western Samoa over the 13 year period 1978-1991. *Int J Obes* 1994, 18: 419-428.
- Hodge AM, Dowse GK, Gareeboo H, Tuomilehto J, Alberti KGMM, Zimmet PZ. Incidence, increasing prevalence and predictors of change in obesity and fat distribution over 5 years in the rapidly developing population of Mauritius. *Int J Obes* 1996, 20: 137-146.
- Hosoya T *et al.* Obesity and hyperuricaemia. *Journal of the Japanese Society for the Study of Obesity* [in Japanese] 1998; 14 (1): 79-85.
- Inoue S. Division of Geriatric Health and Nutrition, National Institute of Health and Nutrition, St Marianna University, School of Medicine, Tokyo, Japan.
- Ismail MN, Zawiah H, Chee SS, Ng KK. Prevalence of obesity and chronic energy deficiency (CED) in adult Malaysians. *Mal J Nutr* 1995; 1: 1-9.
- Ismail MN, Zulkifli MAH. A study on obesity amongst male adolescents. In: 20th. Malaysian Society of Health National Conference on "Adolescent health - Challenges of the 21st. Century", Oct.1996, Kuala Lumpur.
- Ismail MN, Tan CL. Prevalence of obesity in Malaysia. Country report at the Regional Advisory Board meeting on obesity, August 1998, Manila, Philippines.
- Ismail MN, Vickneswary EN. Prevalence of obesity in Malaysia: Data from three ethnic populations. Country report at the Asian BMI/Obesity Workshop, June 1999, Milan, Italy.
- Ito K, Murata M. Diagnostic criteria of childhood obesity. *Japanese Journal of Pediatrics* 1999; 52 (Suppl):1182-96.
- James WPT. The epidemiology of obesity. In: Chadwick DJ, Cardew GC, eds. *The origins and consequences of obesity*. Chichester, Wiley, 1996: 1-16 (Ciba Foundation Symposium 201).
- Kannel WB, D'Agostino RB, Cobb JL. Effect of weight on cardiovascular disease. *Am J Clin Nutr* 1996, 63 (Suppl 4): 19S-22S.

- Kiddy DS, Hamilton-Fairley D, Bush A, Short F, Anyaoku V, Reed MJ, Franks S. Improvement in endocrine and ovarian function during dietary treatment of obese women with polycystic ovary syndrome. *Clin Endocrin* 1992; 36: 105-111.
- King H, Taylor R, Zimmet P, Pargeter K, Raper LR, Beriki T, Tekanene J. Non-insulin dependent diabetes (NIDDM) in a newly developed Pacific Nation: The Republic of Kiribati. *Diabetes Care* 1984; 7: 409-415.
- Ko GTC, Chan JCN, Cockram CS, Woo J. Prediction of hypertension, diabetes, dyslipidaemia or albuminuria using simple anthropometric indexes in Hong Kong Chinese. *Int J Obes* 1999; 23(ii): 1136-42.
- Lake JK, Power C, Cole TJ. Women's reproductive health: the role of body mass index in early and adult life. *Int J Obes* 1997; 12: 432-438.
- Lean ME. Sibutramine - a review of clinical efficacy. *Int J Obes* 1997; 21(Suppl 1): S30-S36.
- Lew EA, Garfinkel L. Variations in mortality by weight among 750 000 men and women. *J Chron Dis* 1979, 32:563-576.
- Lindgren *et al.* Swedish population reference standards for height, weight and body mass index attained at 6 to 16 years (girls) or 19 years (boys). *Acta Paediatrica* 1995, 84(9): 1019-1028.
- Luciano A, Bressan F, Zoppi G. Body mass index reference curves for children aged 3-19 years from Verona, Italy. *European J Clin Nutr* 1997, 51(1):6-10.
- Mahoney LT, Burns TL, Stanford W, Thompson BH, Witt JD, Rost CA, Lauer RM. Coronary risk factors measured in childhood and young adult life are associated with coronary artery calcification in young adults: the Muscatine Study. *J Am Coll Cardiol* 1996; 27: 277-284.
- Matsumura Y, Tsubono Y. Optimal BMI in respect of mortality. Research report of the Foundation of Health Promotion and Fitness. Tokyo, Japan 1998 (in Japanese).
- McKeigue PM, Shah B, Marmot MG. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet* 1991; 337: 382-386.
- Millman RP, Carlisle CC, McGarvey ST, Eveloff SE, Levinson PD. Body fat distribution and sleep apnea severity in women. *Chest* 1995; 107: 362-366.
- Ministry of Education. *Update of Trim and Fit Programme*. Singapore 1996.
- Pettigrew R, Hamilton-Fairley D. Obesity and female reproductive function. *Br Med Bull* 1997;53: 341-358.
- Rich-Edwards JW, Goldman GB, Willett WC, Hunter DJ, Stampfer MJ, Colditz EA, Manson JE. Adolescent body mass index and infertility caused by ovulatory disorder. *Am J Obstet Gynecol* 1994; 171: 171-177.
- Richman R, Steinbeck KS and Caterson ID. Severe obesity: the use of very low energy diets or standard kilojoule restriction diets. *Med J Aust* 1992, 156: 768-770.
- Seidell JC, Cigolini M, Charzewska J, Ellsinger BM, Di Biase G, Björntorp P, Hautvast JG, Contaldo F, Szostak V, Scuro LA. Androgenicity in relation to body fat distribution and metabolism in 38 year-old women - the European Fat Distribution Study. *J Clin Epidemiol* 1990; 43(1): 21-34.
- Shike M. Body weight and colon cancer. *Am J Clin Nutr* 1996, 63(3 Suppl):442S-444S.
- Sorensen TIA, Sonne-Holme S. Risk in childhood of development of severe adult obesity: retrospective, population-based case-cohort study. *Am J Epidemiol* 1988; 127: 104-113.
- Staffieri JR. A study of social stereotype of body image in children. *J Person Social Psychol* 1967; 7:101-4.
- Stamler R, Stamler J, Riedlinger WF, Algera G, Roberts RH. Weight and blood pressure. Findings in hypertension screening of 1 million Americans. *JAMA* 1978; 240: 1607-1610.

- Stampfer MJ; Maclure KM; Colditz GA; Manson JE; Willett WC. Risk of symptomatic gallstones in women with severe obesity. *Am J Clin Nutr* 1992; 55: 652-8.
- Swinburn B. Using the body mass index: weigh then weigh up (editorial). *NJ Med J* 1998, 111 (1075): 377-379.
- Tan C-E, Emmanuel SC, Tan B-Y, Jacob E. Prevalence of diabetes and ethnic differences in cardiovascular risk factors. The 1992 Singapore National Health Survey. *Diabetes Care*. 1999;22:241-247.
- Taylor R, Badcock J, King H, *et al*. Dietary intake, exercise, obesity and noncommunicable disease in rural and urban populations of three Pacific Island countries. *J Am Coll Nutr* 1992; 11: 283-293.
- Wadden TA, Foster GD, Letzia KA. One-year behavioural treatment of obesity: comparison of moderate and severe caloric restriction and the effects of weight maintenance therapy. *J Consult Clin Psychol* 1994; 62: 165-171.
- Willett WC, Stampfer M, Manson J, Vanltallie T. New weight guidelines for Americans: justified or injudicious. *Am J Clin Nutr* 1991, 53(5): 1102-3.
- Willett *et al* 1995. Weight, weight change and coronary heart disease in women. Risk within the 'normal' weight range. *JAMA* 1995, 273(6):461-465.
- Wolf AM, Colditz GA. The costs of obesity: the U.S. perspective. *Pharmacoeconomics* 1994, 5: 34-37.
- World Health Organisation. *Diet, Nutrition, and the Prevention of Chronic Diseases*. Geneva: WHO, 1990.
- World Health Organisation. *Physical Status: the Use and Interpretation of Anthropometry*. Geneva. WHO, 1995.
- World Health Organisation. *Obesity: Preventing and Managing the Global Epidemic*. Geneva: WHO, 1998.
- World Health Organisation. *Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications-Part 1: Diagnosis and Classification of Diabetes Mellitus*. Geneva: WHO, 1999.
- Yen SSC. The polycystic ovary syndrome. *Clin Endocrinol* 1980; 12: 177-208.
- Yoshiike N, Matsumura Y, Zaman MM, Yamaguchi M. Descriptive epidemiology of body mass index in Japanese adults in a representative sample from the National Nutrition Survey 1990-94. *Int J Obes* 1998; 22: 684-87.
- Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; 328: 1230-35.
- Zaadstra BM, Seidell JC, Van Noord PAH, Tevelde ER, Habema JDF, Vrieswijk B, Karbaat J. Fat and female fecundity: prospective study of body fat distribution on conception rates. *Br Med J* 1993; 306: 484-487.
- Zimmet PZ, Whitehouse S, Jackson L, Thoma K. High prevalence of hyperuricaemia and gout in an urbanised Micronesian population. *Br Med J* 1978; 1(6122): 1237-9.
- Zimmet PZ, Taylor R, Ram P, *et al*. Prevalence of diabetes and impaired glucose tolerance in the biracial (Melanesian and Indian) population of Fiji: A rural-urban comparison. *Am J Epidemiol* 1983; 118: 673-678.
- Zimmet P, Dowse G, Serjeantson S, King H. The epidemiology and natural history of NIDDM - lessons from the South Pacific. *Diabetes Metab Rev* 1990, 6: 91-124.
- Zimmet PZ, Collins VR, Dowse GK, Alberti KGMM, Tuomilehto J, Knight LT, Gareeboo H, Chitson P, Fareed D. Is hyperinsulinaemia a central characteristic of a chronic cardiovascular risk factor clustering syndrome? Mixed findings in Asian Indian, Creole and Chinese Mauritian. *Diabetic Med* 1994; 11: 388-96.
- Zimmet P. The pathogenesis and prevention of diabetes in adults: genes autoimmunity and demography. *Diabetes Care* 1995; 18(7): 1050-64.
- Zimmet P. Diabetes epidemiology as a trigger to diabetes research. *Diabetologia* 1999; 42:499-518.

## Appendix 1

### Prevalence of overweight/obesity in the Asia-Pacific region

Country	Year	Age	BMI cut-off	Prevalence (%)
<b>China</b> (Ge 1997 <sup>1</sup> & Chen - personal communication)	1992	20-45 years	BMI 25-30	URBAN: 12.3% (m), 14.4% (f) RURAL: 5.3% (m), 9.8% (f)
	Chinese National Nutrition Survey		BMI > 30	URBAN: 1.0% (m), 1.7% (f) RURAL: 0.5% (m), 0.7% (f)
			BMI 25	URBAN: 17.2% (Shanghai) 32.8% (Beijing) 26.5% (Tianjin) URBAN: (all of China) 1992: 14.9% 1989: 12.0% 1982: 9.7%  RURAL: (all of China) 1992: 8.4% 1989: 7.5% 1982: 6.0%
1992	Children (age not specified)	Obesity (not defined)	Approximately 10%	
<b>Hong Kong Chinese<sup>2</sup></b>				
	1990	18-65 yrs n= 1513	BMI 30 BMI 27 (m), 25 (f)  WC 94 cm (m), 80 cm (f)	2.2% (m), 4.8% (f) 10.0% (m), 27.9% (f)  4.5% (m), 22.5% (f)
<b>Japan</b>				
National Nutrition Survey <sup>3</sup>	1990-94	35-64 yrs n= 12 926	BMI 25.0-29.9 BMI 30	24.3% (m), 20.2% (f) 1.9% (m), 2.9% (f)
National Survey of Primary & Middle Schools <sup>4</sup>	1970 & 1997	9 yrs	Obesity (not defined)	1970: 2.9% (m), 3.4% (f) 1997: 9.7% (m), 8.0% (f)
<b>Korea</b>				
	1994-97	20-60+ yrs (n= 15 145)	BMI > 28 BMI > 30	5.9% (6.1% (m), 5.7% (f)) 1.7% (1.4% (m), 1.9% (f))

Country	Year	Age	BMI cut-off	Prevalence (%)
<b>Malaysia</b>				
<i>Ismail et al 1995<sup>5</sup></i>	1991-94	18-60 yrs (n= 4747)	BMI 25-30 BMI > 30	URBAN (incl. Malays, Chinese & Indians) 24.0% (m), 18.1% (f) 4.7% (m), 7.7% (f)
			BMI 25-30 BMI > 30	URBAN (Malays only) 23.9% (m), 19.6% (f) 5.6% (m), 8.8% (f)
			BMI 25-30 BMI > 30	RURAL (Malays only) 12.9% (m), 17.5% (f) 1.8% (m), 2.6% (f)
<i>2<sup>nd</sup> National Health &amp; Morbidity Survey (Malaysia)<sup>6</sup></i>	1996	18 yrs (n= 31 097)	BMI 25-30 BMI > 30	16.6%, 95% CI (6.1%, 17.1%) 4.4%, 95% CI (4.1%, 4.7%)
<i>Ismail &amp; Zulkifli 1996<sup>7</sup></i>	1990	13-17 yrs (n= 4754)	BMI 85 <sup>th</sup> pc	1990: 1%
<i>Ismail &amp; Vickeneswary 1999<sup>8</sup></i>	1997	13-17 yrs (n= 4018)	BMI 85 <sup>th</sup> pc	1997: 6%
<i>Ismail &amp; Tan 1998<sup>9</sup></i>	1998	7 yrs 8 yrs Total (n= 2292)	wt/ht > +2 SD	6.6% 9.2% 9.9% 13.8%
		10 yrs 7-10 yrs 7-10 yrs n= 1266 (boys) n= 1026 (girls)	wt/ht > +2 SD	9.1% Boys: Malays 16.8% Chinese 11.2% Indians 11.3% Total 12.5%
				Girls: Malays 8.0% Chinese 4.1% Indians 7.1% Total 5.0%
<b>Philippines</b>				
<i>Food &amp; Nutrition Research Institute</i>	1987	20 <sup>+</sup> yrs n= 3689 (m) n= 4466 (f)	BMI 25-29.9 BMI > 30	9.9% (m), 13.4% (f) 0.7% (m), 2.6% (f)
	1993	n= 4588 (m) n= 4997 (f)	BMI 25-29.9 BMI > 30	12.7% (m), 15.2% (f) 1.7% (m), 3.4% (f)
<i>Florentino et al 1999<sup>10</sup></i>	1997	8-10 yrs n= 535 (m) n= 610 (f)	BMI > 85 <sup>th</sup> pc BMI > 95 <sup>th</sup> pc	12.8% 12.0%

Country	Year	Age	BMI cut-off	Prevalence (%)
<b>Singapore</b>				
(World Health Day, - Singapore Health Policy 1999)	1992 & 1998	18-69 yrs (n= 4700)	Obesity (not defined)	6% in 1992 & 1998 (all of Singapore) 11.1% to 16.2% (Malay women) 12.5% to 17.5% (Indian women)
<b>Taiwan</b>				
		> 19 yrs	BMI 24.2-26.4 BMI 26.4-28.6 BMI > 28.6	17.5% (m), 15.9% (f) 9.6% (m), 7.9% (f) 5.0% (m), 7.9% (f)
<b>Thailand</b>				
National Health Examination Survey <sup>11</sup>	1991	20 yrs (n= 13 300)	BMI 25-30 BMI > 30	16.7% (12% (m), 19.5% (f)) 4.0% (1.7% (m), 5.6% (f))

KEY: BMI: body mass Index CI: confidence interval f: females m: males pc: percentile  
SD: standard deviation WC: waist circumference wt/ht: weight-for-height.

## References

- 1 Ge L. Body mass index in young Chinese adults. *Asia Pacific J Clin Nutr* 1997; 6(3): 175-179.
- 2 Ko GTC, Chan JCN, Woo J, Lau E, Yeung VTF, Chow C-C, Wai HPS, Li JKY, So W-Y, Cockram CS. Simple anthropometric indexes and cardiovascular risk factors in Chinese. *Int J Obes* 1997; 21: 995-1001.
- 3 Yoshiike N, Matsumura Y, Zaman MM, Yamaguchi M. Descriptive epidemiology of body mass index in Japanese adults in a representative sample from the National Nutrition Survey 1990-94. *Int J Obes* 1998; 22: 684-87.
- 4 Ito K, Murato M. Diagnostic criteria of childhood obesity. *Japanese Journal of Pediatrics* 1999; 52 (Suppl): 1182-96.
- 5 Ismail MN, Zawiah H, Chee SS, Ng KK. Prevalence of obesity and chronic energy deficiency (CED) in adult Malaysians. *Mal J Nutr* 1995; 1: 1-9.
- 6 Fatimah S, Tahir A, Siti Sa'adiah HN, Maimunah H. *Nutritional status of adults aged 18 years and above. National Health and Morbidity Survey (NHMS). Vol 14, 1997, Kuala Lumpur.*
- 7 Ismail MN, Zulkifli MAH. *A study on obesity amongst male adolescents. In: 20<sup>th</sup> Malaysian Society of Health National Conference on "Adolescent health - Challenges of the 21st Century". Oct 1996, Kuala Lumpur.*
- 8 Ismail MN, Vickneswary EN. *Prevalence of obesity in Malaysia: Data from three ethnic populations. Country report at the Asian BMI/Obesity Workshop. June 1999. Milan, Italy.*
- 9 Ismail MN, Tan CL. *Prevalence of obesity in Malaysia. Country report at the Regional Advisory Board meeting on obesity. August 1998. Manila, Philippines.*
- 10 Florentino R, Villavieja GM *et al. Nutritional status, dietary and physical activity patterns of schoolchildren in Manila. Food and Nutrition Research Institute, Department of Science and Technology, Republic of the Philippines, 21 Jan 1999.*
- 11 *First report on survey of health status in Thai populations by questionnaires and physical examination, 1991-92. Churapavarn J eds. Bangkok: Institute of Public Health, 1996 (in Thai).*

## Appendix 2

### Morbidity/mortality risk associated with overweight/obesity

Country	Study sample adjustments etc	BMI cut-off/waist circumference cut-off	Disease/outcome	Risk/conclusions
<b>Hong Kong Chinese<sup>1</sup></b>				
	<i>n</i> = 910 (m) <i>n</i> = 603 (f)	1kg/m <sup>2</sup> increase in BMI	Dyslipidaemia	OR= 0.80 (0.72, 0.88) (m)
		1cm increase in WC	Albuminuria	OR= 1.05 (1.02, 1.09) (f)
		Unit increase in WHR	Diabetes	OR= 1.16 (1.10, 1.23) (m)
			Albuminuria	OR= 1.10 (1.05, 1.15) (m)
			Dyslipidaemia	OR= 1.09 (1.06, 1.13) (f)
			Hypertension	OR= 1.26 (1.18, 1.35) (f)
<b>Japan</b>				
		BMI > 25	Diabetes	OR= 2
			Hypertension	OR= 2
			Hypertriglyceridaemia	OR= 2
			Hypercholesterolaemia	OR= 2
			Decreased HDL-cholesterol	OR= 2
<b>Korea</b>				
	Annual Health Examination Survey (1994-97)	BMI > 28	Hypertension	OR= 4.1 (3.4, 5.0)
			Diabetes mellitus	OR= 2.2 (1.5, 3.2)
			Dyslipidaemia	OR= 3.7 (3.0, 4.4)
			Gallstones	OR= 1.4 (1.1, 2.6)
	Adj for age, sex		Fatty liver	OR= 2.2 (1.4, 3.6)
			Hyperuricaemia	OR= 2.4 (1.7, 3.2)
Kim <i>et al</i> 1997 <sup>2</sup>	20-60+ years <i>n</i> = 15 145,	BMI > 26 (m) BMI > 25 (f)	Diabetes mellitus	OR= 3.2 (2.3, 4.4)
			Hypercholesterolaemia	OR= 1.2 (0.9, 1.4)
			Hypertriglyceridaemia	OR= 2.1 (1.7, 2.8)
	Annual Health Examination, 1994-1997		Gall stones	OR= 1.4 (1.1, 2.6)
			Fatty liver	OR= 1.3 (0.8, 2.3)
	Adj for age & sex			
Kim <i>et al</i> 1998 <sup>3</sup>	<i>N</i> = 410 women 71 men, hospital obesity clinic	Waist circumference > 91.3cm (m) > 78cm (f)	Waist circumference greater than these suggested cut-offs are likely to be associated with metabolic disorders	

KEY: BMI: body mass index f: females m: males OR: odds ratio pc: percentile SD: standard deviation  
WC: waist circumference WHR: waist-to-hip ratio

#### References

- 1 Ko GTC, Chan JCN, Cockram CS, Woo J. Prediction of hypertension, diabetes or albuminuria using simple anthropometric indexes in Hong Kong Chinese. *Int J Obes* 1999 (in press).
- 2 SM Kim, DJ Lee *et al*: A Study on the Obesity Index of Health Examination Center Data. *J of Korean Society for the Study of Obesity* 1997; 6(2): 137-142.
- 3 SM Kim, DJ Lee *et al*: What is the Best Simple Anthropometric Indexes of Abdominal Visceral Fat in Obese Patients? *J of Korean Society for the Study of Obesity* 1998; 7(2) 157-168.

## Appendix 3

Age-adjusted risk of hypertension and Type 2 diabetes for Indian Asians and Chinese associated with different levels of BMI (new cut-offs for Asians)

BMI group <sup>1</sup>	INDIAN ASIANS				CHINESE				
	Hypertension		Type 2 diabetes		Hypertension		Type 2 diabetes		
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	
<b>Men</b>									
<b>18.5-22.9</b>	<b>Normal range</b>	<b>1.00</b>	<b>-</b>	<b>1.00</b>	<b>-</b>	<b>1.00</b>	<b>-</b>	<b>1.00</b>	<b>-</b>
< 18.5	Underweight	0.98	(0.62, 1.57)	0.43*	(0.24, 0.79)	-(n=0)	-	1.11	(0.11, 11.64)
23.0-24.9	At risk	1.40*	(1.02, 1.93)	1.42*	(1.02, 1.98)	1.24	(0.53, 2.89)	4.46*	(1.64, 12.08)
25	Obese	2.16*	(1.64, 2.85)	2.33*	(1.76, 3.09)	1.49	(0.68, 3.28)	3.44*	(1.31, 9.00)
<b>Women</b>									
<b>18.5-22.9</b>	<b>Normal range</b>	<b>1.00</b>	<b>-</b>	<b>1.00</b>	<b>-</b>	<b>1.00</b>	<b>-</b>	<b>1.00</b>	<b>-</b>
< 18.5	Underweight	0.56	(0.29, 1.10)	0.68	(0.34, 1.37)	1.47	(0.23, 9.35)	-(n=0)	-
23.0-24.9	At risk	1.41	(0.96, 2.08)	1.49*	(1.00, 2.22)	1.74	(0.64, 4.74)	2.63	(0.81, 8.55)
25	Obese	2.42*	(1.81, 3.24)	2.72*	(2.02, 3.67)	3.57*	(1.53, 8.31)	5.37*	(1.95, 14.74)

<sup>1</sup> ORs (odds ratios) relative to those within the normal range (BMI 18.5-22.9 kg/m<sup>2</sup>)

## Appendix 4

### Defining the Metabolic Syndrome

Component	Definition
Glucose intolerance, IGT or diabetes mellitus and/or insulin resistance together with 2 or more of the following:	Defined as - under hyperinsulinaemic euglycaemic conditions, glucose uptake below lowest quartile for background population under investigation
Raised arterial pressure	160/90 mmHg
Raised plasma triglycerides and/or low HDL-cholesterol	1.7 mmol/l; 150mg % (< 0.9 mmol/l, 35mg % men; <1.0 mmol/l, 39mg % women)
Central obesity	Males: waist to hip ratio > 0.90; Females: waist to hip ratio > 0.85 and/or BMI > 30kg/m <sup>2</sup>
Microalbuminuria	Urinary albumin excretion rate (20 µg min <sup>-1</sup> or albumin:creatinine ratio (20mg/g-1).

NOTE: These values are not applicable in Asian populations, need data on Asian communities.

## Appendix 5

### Weight loss drugs and their side effects

Drug	Action	Adverse effects	Stage of development
Sibutramine	<i>centrally acting via serotonergic and nonadrenergic pathways, not recommended for those with severe hepatic disease</i>	<i>increase in blood pressure and heart rate, nausea, insomnia, dry mouth, rhinitis, constipation</i>	<i>approved by the FDA (November 1997)</i>
Phentermine	<i>centrally acting via nonadrenergic pathways</i>	<i>increase in blood pressure, insomnia, nervousness</i>	<i>approved by the FDA &amp; licensed in the UK, but recommended only for short-term use (12 weeks)</i>
Mazindol	<i>appetite suppressant and inhibits re-uptake of nonadrenaline</i>	<i>increase in blood pressure, insomnia, constipation, dry mouth</i>	<i>approved by the FDA and the Japanese government, available in Australia, but recommended only for short-term use (12 weeks)</i>
Amfepromone (diethylpropion)	<i>appetite suppressant</i>	<i>increase in blood pressure, insomnia, nervousness</i>	
Fluoxetine (Prozac)	<i>anti-depressant, appetite suppressant and a selective serotonin re-uptake inhibitor</i>	<i>anxiety, drowsiness, insomnia, nervousness</i>	<i>approved by the FDA as a treatment for depression, bulimia and obsessive-compulsive disorder, not specifically approved for weight loss</i>
Metformin	<i>this may be useful in managing obesity in the Type 2 diabetic patient, although efficacy is not proved or licensed for obesity</i>		
Ephedrine/caffeine	<i>has anorectic properties and some thermogenic effects</i>	<i>hypertension wakefulness tachycardia</i>	<i>available in Denmark</i>
Orlistat	<i>peripherally acting pancreatic lipase inhibitor, decreases fat absorption</i>	<i>loose stools, malabsorption of fat-soluble vitamins, possible link with breast cancer</i>	<i>approved by the FDA</i>

## Notes

---