Obesity prevention

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Introduction

Many diseases, such as the auto immune disorders, are extremes of normal processes, and obesity is no different in this respect. However, obesity has historic and public health issues that require a new approach by the medical profession. Historically, obesity has been labelled as self-induced, caused by 'greed and laziness' The previous lack of scientific research and an enduring judgmental attitude has caused obese people a great deal of distress. Compounding the issue more recently is the pressure to have a 'model' body shape.

Obesity prevention can be considered in four categories:

1. Primordial prevention requires making environmental changes conducive to developing healthy weight.
2. Primary prevention aims to prevent weight gain, particularly in those at risk.
3. Secondary prevention involves weight reduction and the prevention of further weight gain.
4. Tertiary prevention involves managing the consequences of obesity such as type II diabetes mellitus (TIIDM) and atherosclerotic cardiovascular disease (ASCVD).

The world needs to come to terms with a new view of obesity as it becomes clear that there are huge environmental factors influencing this so called epidemic. Denoting millions of over-fat adults and children as slothful gluttons, ignoring obesity-related disease or failing to try and prevent untimely deaths of the obese (approximately eight to 10 years earlier than the non-obese) is unacceptable.

Obesity is a chronic, damaging disease that often includes significant medical, functional and psychological components, and is certainly overlain by a complex weave of polygenetic influences, and unleashed by extraordinary societal changes in attitudes and physical lifestyle.

New Zealand 1997 data show that more than half of all adults were above a healthy weight, and nearly a fifth obese.[3] The World Health Organization (WHO) states that *'Obesity is a condition of excessive fat accumulation in the body to the extent that health and well-being are adversely affected.'*[4]

New attitudes to obesity

Blaming attitudes to obesity have not cured the disease and other extreme methods of weight gain prevention (food deprivation and/or prolonged intense exercise) rarely work. It is imperative to examine our attitudes in light of our biology.

Surprisingly, very few people have put the issue in an appropriate 'biology in history' context, which I think is vital to initiate changes in both the development of obesity and the management of the established disease. We should expect that humans will have extraordinarily well-developed mechanisms to deal with initiating and finding, keeping, digesting and metabolising food.

Moreover, default desires (making our daily life physically easy, with abundant highly palatable food as convenient as possible, in a life overfull of time pressure and performance stress) that industry advertises and makes available to us, normalises our current lifestyle. In our sedentary lifestyle, we need far less energy than most of us can imagine. We must now tell our patients the truth.

Our history, distant past and recent, tells us that human survival in the face of food deprivation, and hard physical toil, is common. Nearly all humans today are, genetically, famine survivors. The corollary is, however, that we have few genes with which to manage chronic, regular energy overload coupled with underexpenditure of muscular work. Exposure to an 'affluent' energy lifestyle has been relatively rare in evolutionary terms. In the current environment we should marvel that not everyone is massively obese.

Genetics, epigenetics and foetal programming

However, obesity is a complex product of our genetics and the environment. There is now ample evidence that externally imposed stressors in utero and infancy, such as malnutrition (inadequate or excess energy, and unbalanced nutrients) of the mother, or other causes of intra-uterine growth retardation such as altered corticosteroid exposure, alter the gene expression and various metabolic

Table 1.

WHO Waist[30]	Europids / Polynesian			Asian (equivalent values, not finalised)
Male	Healthy <90cm ... Mod risk ... High risk >102cm			<80cm ... >90cm (?)
Female	Healthy <80cm ... Mod risk ... High risk >88cm			<70cm ... >80cm (?)
WHO BMI Classification[4]	**BMI Europids**	**BMI Polynesian**	**BMI Asian**	**Risk of Co-morbidity***
Underweight	Below 18.5	Below 18.5	Below ?17.5	Low
Healthy weight	18.5-24.9	18.5-25.9	17.5?-22.9	Average
Overweight (grade 1 obesity)	25.0-29.9	26-31.9?*	23-24.9	Mild increase
Obese (grade 2 obesity)	30.0-39.0	32.0-39.0	25.0-29.9	Moderate/severe
Morbid/severe obesity (grade 3 obesity)	40.0 and above	40.0 and above	30.0 and above	Very severe

Table adapted from WHO data.

* *Does this BMI mean lower morbidity for Polynesians? This issue is unresolved, but kidney and retinal disease is cause for concern. See Australian Adult and Childhood Obesity Guidelines[31]*

controllers in the growing child. These stressors 'programme' the child's metabolism to expect the worst; protect the brain and heart and lay down as much short-term survival fat as possible, at the expense of lean tissue – skeletal muscle[7] and gut (liver, kidney, pancreas[8]). This programming lasts for the child's life and predisposes the individual to become obese, hypertensive and diabetic – all contributing to ASCVD. Genetic factors may also predispose to both low birth weight and ASCVD, as shown in studies where there tends to be more environmental effect from the mother and more genetic effect from the father. There is also the added factor of *'a well-recognised "intergenerational cycle of growth failure" in the developing world*,'[8] and this may represent intergenerational foetal programming, which can result in the foetus being *'warned in utero of certain malnutritional stress factors to be expected ex-utero'* only to find that unbalanced energy overload is the problem. Evidence suggests that with slower changes this programming may begin to redress the problems of environmental change, such as the adaptation apparent in white Europeans whose food sources have been becoming more fatty and more refined for some centuries. However, for most groups, more recently exposed to western lifestyle transformation change is happening too fast. *'As with most nature versus nurture arguments, the answer is probably a mixture of the two.'*[8]

The development of the human phenotype (for example, obesity) is characterised initially by genetic make-up. There is strong evidence that the genetic expression is, in turn, influenced and programmed at the foetal stage by the maternal and peri-natal environment, with such effects extending back a few generations. Lastly, there are interactions with the immediate environment throughout life.

When discussing obesity prevention, we have to approach obesity as a disease. In the recent past, obesity management was seen as necessary only when obesity was combined with hard to treat ASCVD or cancer co-morbidities. Now we need to treat obesity and co-morbidities as they are, after age, the strongest predictors of ASCVD.[9,10]

Pathophysiology

Interestingly, many of the co-morbidities, risk factors and pharmaceutical treatments associated with obesity have been studied at great length. However, partly due to negative attitudes, research and management directed primarily towards obesity have often been skirted around, and lifestyle contributors (nutrition, physical activity and psychological stress) have almost been ignored.

Historically, the relatively rare autoimmune failure of pancreas islet cells resulting in hyperglycaemia led to the discovery of insulin treatment which was life saving for those affected. The hyperglycaemic microvascular complications of this condition have been well studied. However, even though it was known that

Table 2. Grading of the metabolic syndrome

Metabolic Syndrome MSX*
Variables (cut-off range)
BMI (18.5-25) kg/m2
Metabolic Syndrome Traits
Waist (<102 male, <88 female) cm
{**SBP** (<135) mmHg and/or
{**DBP** (<85) mmHg
FPG (<6.1) mmol/L
HDL-C (>1 male, >1.3 female) mmol/L
TAG (<2) mmol/L
Metabolic Syndrome MSX
Three or more traits from above list.

hypoinsulinaemia was not the main problem in adult onset hyperglycaemia, the two conditions were included in the same model.

Blending the clinical syndromes of the two hyperglycaemic states, TIDM and TIIDM, which stem from different causes, has been problematic.

Drug development proceeded, often empirically, and made a difference to morbidity and mortality. Some nutrition messages were being inferred and disseminated such as to reduce cholesterol, associated with saturated animal fat. Hyperglycaemic patients were told to eat less sugar. By this time obesity rates were rising in Westernised countries and hypertension, dyslipidaemia, hyperglycaemia and atherosclerosis were noticed to be present in the same population. But still, the focus was to treat these four risk factors, as 'treating the obesity was too hard.'

However, researchers started to look for associations between these risk factors, and improved genetic and intervention studies methods spurred research. The (dys)metabolic syndrome [MSX] has been mooted for some decades, but in 2002 the NCEP, ATPIII published a detailed review that rigorously analysed studies on risk factors for ASCVD, and five parameters were deemed to be the most reliable predictors[11] (see Table 2). Note that low density lipoprotein, which is small and dense in the MSX and voluminous and 'fluffy' in healthy slim people and often does not change value after weight loss, has dropped out of the equation.

We now know that fat is not inert energy storage. Its unchecked accumulation in the abdominal region causes functional problems and produces many novel hormones and proteins in an unbalanced way. It is also important to note the independently positive health association of, and ASCVD protection offered by, high levels of hip and thigh fat, and possibly muscle, found in some woman.[12]

High fat feeding is known to down-regulate some of the key appetite controlling hormones[13] – the

Box 1. Macronutrient Metabolism – Notes

Fat

- rapidly and efficiently stored
- the most energy-dense food
- poor satiety, passive over-consumption
- provides energy mainly for thermogenisis, mod intensity PA, when underfed

CHO

- main 24 hr energy supply
- not appreciably turned to fat stores
- limited (1–2kg) glycogen store
- are starch, sugars, fibre i.e. fruit and vegetables
- with fibre, slows glycaemic response
- moderately good satiety
- carries main antioxidants, vitamins

Protein

- not appreciably turned to fat stores
- best satiety esp. fish
- inefficient energy supply
- excess, a problem with renal impairment

NB: Metabolic rate depends on lean tissue and age – prepare to eat less with age, and after menopause.

Box 2. Food Quality Guide

In the following order of importance!

1. **Reduce dietary fat:** only very small amounts needed - don't add, don't see, beware of hidden, industrialised fats (e.g. baking, biscuits, chips)
2. **Fruit and vegetables:** fresh, frozen, dried. Increase - as much as can be managed, at least '5 a day', no limits, use as snacks, filling, nutrient dense food.

And ...less important for weight control...

3. **Have less refined, processed CHO:** moderate starches, modest sugars.
4. **Eat enough lean protein for hunger control:** pulses, lean meat, fish, poultry.

Box 3. Food frequency guide

- Eat before over-hungry. High-energy foods are overeaten when hungry, tired stressed.
- When hungry, eat. Snack on fruit/vegetables, or low energy dense food.
- Carry own appropriate food snacks.
- Have smaller main meals. OK to snack before or after.
- Have high energy meals earlier in day - more time to compensate if excess energy.

Notes

[A] Large, infrequent meals, especially or constant grazing are linked to obesity.
[B] Large night meals associated with less desire for breakfast
[C] Most do best with eating three to six times day, to fit in with individual patterns.

more fat you eat the more you want, but luckily, with time, the less fat you eat the less you want. In addition, some fatty acids (omega 3 fatty acids) seem to modify inflammation and the tendency to gain fat, whereas others worsen the metabolic reaction, and this can be more pronounced in obesity.[14]

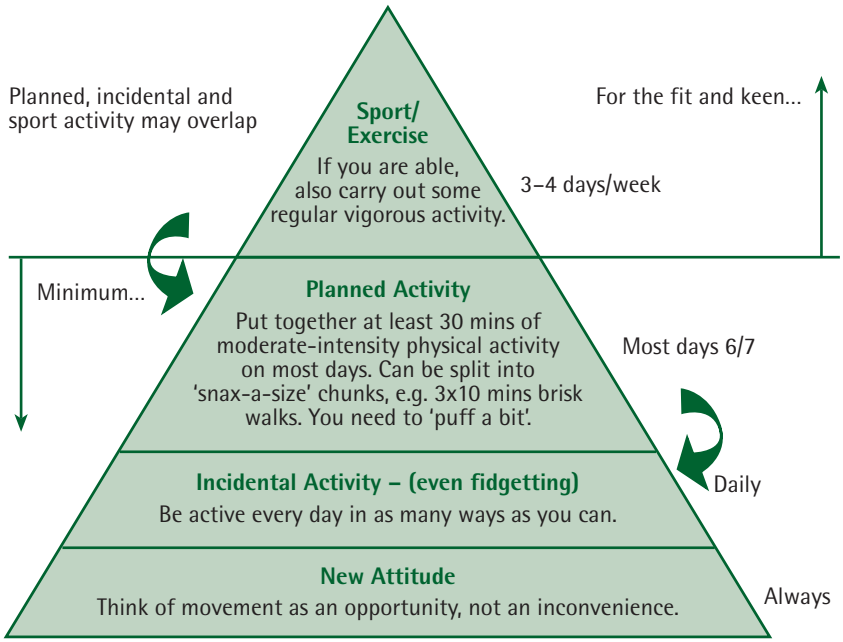
Many of the brain hormones and receptors regulating appetite involve various addiction receptors[15] (presumably encouraging the re-acquisition of high energy food) and other mood-related neurons, which helps to explain why we have a strong drive to eat high energy food, especially when tired, hungry and depressed.

The huge flux of fats and sugars in the body results in many tissue cells such as liver, skeletal and heart muscle being stuffed with toxic fat[16] altering their fat and glucose metabolism and becoming insulin resistant. Organs such as the liver and pancreas, when overfilled with fat, synthesise altered types and quantities of protein, and secrete or leak proteins such as ferritin,[17] transaminases[18] and amylin.[19] Often these proteins have mixed metabolic and anti-pathogen and anti-cancer (i.e. anti-inflammatory) roles, and under the load of lipid, the rather plastic visceral adipose cells and immune leucocytes cells start interchanging functions.[20]

Excess abdominal fat secretes large amounts of inflammatory cytokines that accelerate lipid and inflammatory processes in arterial epithelial and foam cells. Metabolic controls fail and chemicals, such as tissue necrosis factor TNF-a, an inflammatory protein, may activate feed-forward mechanisms that inhibit anti-inflammatory proteins such as insulin[21] and adiponectin[22] and exacerbate all the other imbalances. The high energy trafficking (obese people have more higher-metabolic-rate, lean tissue than is appreciated, though the muscle is often fatty and unfit) has another down side. Oxidative stress in the form of excess production of reactive oxygen species (ROS) or free radicals, from excess fatty acids and glucose levels, requires high levels of antioxidant chemicals found in a variety of fresh fruit and vegetables, the very food that is often lacking in the diet of most people these days, particularly those who are obese.

The traditional risk factors for TIIDM and ASCVD – hypertension, dyslipidaemia, hyperglycaemia are now interwoven with 'atypical risk factors' – abdominal obesity, insulin resistance, coagulopathies, steatohepatitis, eccentric cardiac hypertrophy, cardiomyopathy and/or CHF, hyperuricaemia, hyperhomocysteinaemia, inflammation,

Figure 1. Physical Activity Guidelines



Adapted from Garry Egger, 1999, Weight Management Workshop Series (unpublished).

Box 4. Current drugs used for weight loss

Orlistat: Lipase blocker, can take with psychotropics, may double the rate of fat loss compared with placebo.
Side effects: cramping, oily anal loss.

Sibutramine: Designer serotonin and dopaminergic effects, may double weight loss, now thought to reduce blood pressure in very overweight due to central effects (may increase in normal weight people), can be used long-term.
Side effects: dry mouth, sleeplessness, constipation which usually resolve early.

Phentermine: An older drug, less studied, adrenergic and dopaminergic effects but no addiction or dependence, possible tachyphylaxis, still has six-month restriction.
Side effects: dry mouth, sleeplessness, constipation, palpitations and agitation, which usually resolve early.

Fluoxetine, an SSRI, (alone or with Phentermine) can have anti-obsessive, anti-binge and antidepressant effects as well as promoting weight loss.[2]

Upcoming drugs (about 100 are being researched)
Cannabinoid receptor blockers may aid in both weight loss and smoking cessation.[5,6]

For a recent review of current and new drugs see Korner.[5]

activation of the hypothalamic-pituitary-adrenal (HPA) axis and suppression of the hypothalamic-pituitary-thyroid and hypothalamic-pituitary-gonadal axes.[23]

Somewhat serendipitously, most of the hypotensive, hypoglycaemic, normocholestrolaemic and obesity reduction medications have anti-inflammatory, corticosteroid, angiotensin and adreno-sympathetic modifying effects.

Prevalence and epidemiology

In spite of a few recent articles intimating that some of us are over-estimating the 'weight' or seriousness of obesity, and causing a degree of panic[24] there is good reason to believe that clinically we are underestimating the importance of new research described above.[16] The increase in prevalence of obesity is well described, as is the incidence of TIIDM, in adults, children and different ethnic groups.[25,26]

The epidemiology literature should be spurring us on as it is replete with examples of the epidemiologic transition of less wealthy nations developing 'hypertriglyceridaemic waist'[27] obesity and the MSX, alongside poverty and under-nutrition. Non-white people whose ethnic groups have had long histories of low fat and low processed food have even greater health problems than white populations, which have been relatively protected.[28,29]

Classification

BMI has been used to classify obesity, as it is easy to measure and better than using weight alone. However, we now have techniques that measure body fat proportions and distributions, and the science to tell us where the problems lie. It is inaccurate to judge an individual's ASCVD/TIIDM risk by BMI alone, but it is useful to record due to its widespread use and ease of measurement.

A simple girth circumference, taken horizontally as near as possible to where the lower ribs end above the iliac crest, remains a good surrogate for abdominal fat, especially in men. In women, particularly, we have gone back to recording hip circumference, as a large diameter looking horizontally from the side at the widest point of the buttocks, as this is protective for ASCVD, irrespective of waist, even in slim women.[1] For measurements, use a large or thigh blood pressure cuff, tape measure to 200cm and scales that weigh to 200kg.

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Management

As a clinician it is important to find out where individuals 'are at', and what they think about their weight and their efforts to control their weight. Brief intervention methods are important and often if people are ready to start changing, it helps to ask:

1. What is the most important thing about their weight to them; and
2. What is the one thing they feel they could change.

Weight loss is generally going to be more sustained if it is done slowly, over time and with any blame directed to the environment. We also need to tell people the truth about how little energy we need, and how physically efficient the human body can be and redress some of the common myths.

Lifestyle

Although it is important to ask patients to report their eating patterns, these are often more revealing for what is not said.[32] Almost everyone who is overweight will misreport. Likewise, physical activity is difficult for the obese, and is often over reported. Rather than correct their accounts it is useful to keep on listening, over time, as snippets of what really happens generally appear.

Drugs and obesity

Useful drugs for the metabolic syndrome are those that target each individual risk factor. When to start them depends on either a very high level of a single risk factor or a combination of risk factors. Drugs may even be useful for the prevention of weight gain and regain.

Summary

Until obesity is treated as a public health crisis and major societal and industrial changes are made, primary health workers need to enable individual patients to navigate the insidious, hostile environment. Researched information on the biological response to the current situation can be used to provide new practical, helpful management strategies to prevent unhealthy fat mass gain. Such help needs to be given in a continuous, long-term manner, as and when patients perceive the need for aid.

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