Overweight and Obesity in Adults

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Summary

In overweight and obese people with impaired glucose tolerance or raised blood pressure, modest weight loss (3–5 kg) achieved using a combination of increased moderate physical activity (e.g. walking) and improved dietary quality will reduce the progression to diabetes by about 50% and reduce blood pressure by about 3 mmHg. Given the high levels of diabetes, cardiovascular disease and renal failure in Aboriginal people, it would be sensible to recommend modest weight loss to all overweight and obese people. The advice should contain both information about moderate physical activity and dietary quality; it should not focus on cutting calories (kilojoules) alone. People should not be discouraged from losing some weight by being told what their 'ideal' weight 'should be' when this is too big a target to be achieved realistically. However, it should be noted that trials showing that progression to diabetes in people with impaired glucose tolerance could be reduced with lifestyle modification when subjects received up to 16 counselling sessions. Achieving the same level of effect in Aboriginal communities would probably require a large commitment in resources.

[Editor: The studies referred to here utilised 150 minutes per week of moderate intensity physical activity (such as walking) as well as resistance training to increase strength and capacity of muscle. Improvements in dietary quality were achieved with intensive specialist support (e.g. seven nutrition consultations in the first year, and four per year thereafter.) While the weight loss achieved was modest, the resources and commitment required to achieve them were not. While any improvements in physical activity and diet should be encouraged, it is important not to assume that a lesser intervention will necessarily achieve an equal effect.

These editorial comments are based on discussions within the editorial committee and specific technical input from Dympna Leonard, Public Health Nutritionist, Tropical Public Health Unit Cairns.]

While individuals can achieve some of the goals by their own actions other important strategies, such as making sure there is a range of foods available for consumption, will require group decisions and action.

Relevance

Overweight and obesity are important predictors of diabetes, hypertension and heart disease and these are major causes of morbidity and early mortality in Aboriginal and Torres Strait Islander Australians. Overweight

and obesity are also becoming more common in Aboriginal, Torres Strait Islander and non-Indigenous Australians.[1] Losing all excess weight is very difficult and has a low success rate. However, randomised controlled trials have shown that some of the important risk factors can be substantially improved after a small amount (3–5 kg) of weight is lost. Therefore, clinic staff can promote the health benefits of losing some weight to overweight and obese people and can justify helping these people lose some weight, and keep it off, even when the 'ideal' weight is never achieved. They should also encourage people in the healthy weight range to stay active to prevent weight gain.[1]

Definitions

Body mass index (BMI, also called the Quetelet Index) is defined as weight (kg)/height(m)^2. This usually yields a value in adults in the range of about 17–35 kg/m^2 (the units of this ratio tend to be omitted in documents). To assist practitioners interpret the BMI values, various national bodies have divided this range up into groups and have given names to the groups such as 'underweight', 'acceptable or healthy weight', 'overweight' etc.

In the 1970s the cut-off points defining each weight group were lower for women than men[2] based on the fact that women have a higher proportion of fat at a given BMI than do men. Other studies showed that fat varies with age, and older people have a higher proportion of fat for a given BMI than younger people.[3] In other words, an old man might have a higher proportion of fat at a given BMI than a young woman. This means that it is not logical to base cut-off points on sex alone if the goal is to have the cut-off points related to percentage of body fat. However, subsequent analyses revealed that the risk of health outcomes was much the same in men and women for the same BMI, despite the differences in proportion of body weight that was fat. So, for the purpose of making guidelines to categorise BMI that are based on mortality, rather than proportion of body weight that was fat, men and women could be grouped together. Like many health phenomena, the relationship between BMI and mortality was a continuous one, and had a J shape, and so to make categories to guide clinical advice:

> The method used to establish BMI cut-off points has been largely arbitrary. In essence, it has been based on visual inspection of the relationship between BMI and mortality: the cut-off of 30 is based on the point of flexion of the curve . . . It may therefore be necessary to revise the classification of overweight in terms of BMI based on health risk.[3]

In 1984, the NHMRC proposed the categories shown in table 1 for use in Australia with adults.[4,5] Note that the decimal places are implied. For example, someone with a BMI of 19.9 is classified as underweight; the decimals are not rounded up to 20.

Table 1: 1984 NHMRC cut-off points for weight categories for adults[4,5]

Group	BMI range
Underweight	<20
Acceptable/healthy weight	20> to <25
Overweight	25> to <30
Obese	>30

More recently the WHO has proposed the groupings shown in table 2.[3] The most important difference between the WHO classification and the NHMRC classification is that people in the range 18.5–<20 are described as ‘acceptable’ rather than ‘underweight’. This change was made on the basis that studies from less developed countries indicated that healthy physically active people in this weight range did not have excess mortality and that studies in developed countries had not always corrected for deaths early in the follow-up period or adjusted for smoking. Although the NHMRC has not endorsed the WHO categories for use in Australia, a modified version of them that uses the NHMRC category names is now widely used (table 3) and they were used to analyse the 1995 National Nutrition Survey.[6]

Table 2: Cut-off points for weight categories for adults recommended by the 1995 WHO Expert Committee[3]

Category	**BMI range**
Underweight	
Severe thinness	less than 16
Moderate thinness	16 to <17
Mild thinness	17 to <18.5
Acceptable weight	18.5 to <25
Overweight	
Grade 1	25 to <30
Grade 2	30 to less than 40
Grade 3	40 and over

Table 3: Combined NHMRC/WHO cut-off points in common use in Australia at present [6]

Group	**BMI range**
Underweight	<18.5
Healthy weight	18.5 to <25
Overweight	25 to <30
Obese	30 and over

Changing the lower cut-off point for the acceptable weight category will alter the proportion of the population who fall in the ‘underweight’ and ‘acceptable weight’ categories. Table 4 shows the results for the all-Australian population from the 1995 National Nutrition Survey.[7] The prevalence of underweight and acceptable weight depends on whether the 1984 or 1995 definitions are used. It is important to check which cut-off points have been used in a report and not to make assumptions based on the names of the categories that the authors used.

Table 4: Prevalence of different categories of body size in adults aged 19 years and older in the 1995 National Nutrition Survey[7], recalculated* excluding those not measured

Category and BMI cut-off points		Men	Women
		%	%
Underweight		0.6	2.3
Acceptable	18.5–<20.0	2.3	6.0
	20.0–<25	32.6	42.5
Overweight		45.7	30.2
Obese		18.7	19.1

*The recalculation is necessary as ABS documents tend to include those not measured as part of the percentage calculations.

There are also some small discrepancies between the NHMRC and WHO cut-off points. Specifically, 25.000 . . . 0 is classed as acceptable weight in the NHMRC scheme and as overweight in the WHO scheme; 30.000 . . . 0 has also moved up one category. The consequence at the population level is more annoying than real. For example, when we were analysing the 1994 National Aboriginal and Torres Strait Islander Survey[8], only three out of the 7858 adults had a BMI of exactly 25.000 . . . 0 (these days, computers store an infinite number of decimal places) and would have been affected by this discrepancy.[9] It is more common that the calculation leads to other numbers such as 24.99 or 25.05 which have not changed their classification. Hence these small discrepancies in definition do not affect the population prevalence in an important way.

Table 5: Distribution (%) of Body Mass Index (kg/m^2) in Aboriginal and Torres Strait Islander adults (18 years and older), NATSIS, 1994[10], recalculated excluding those not measured

BMI group	Aboriginal men	Torres Strait Islander men	Aboriginal women	Torres Strait Islander women
Underweight<20	8.2	1.9	13.3	4.8
Acceptable 20 to 25	33.1	25.7	30.9	16.1
Overweight >25 to 30	35.6	30.5	28.8	29.0
Obese >30	23.0	41.8	27.1	50.1

It is worth drawing the reader’s attention to the fact that the category cut-off points described for adults are not appropriate for classifying infants, children or adolescents. Various classifications have been proposed if BMI is calculated for children and adolescents.8,11–13 However, the long-term predictive power of these classifications are unknown.

The categories are part of a continuous distribution

The association between weight and risk of mortality or morbidity is continuous and rising above a BMI in the 20–25 range. Various studies have reported that the lowest risk is found at BMIs as low as 20 or as high as 25 in different Caucasian groups.[3,14] This has lead to different national bodies making different decisions about the cut-off points based on the same information. For example, at the time the NHMRC was setting 25 as the boundary for overweight in Australia, the Americans were setting 27.8 in men and 27.3 in women as the boundary for overweight and the Canadians were setting 27 as the boundary for obesity.[3] The continuous distribution means

that the risk of adverse outcomes is not equal for every point within the overweight range. For example, at the lower end around 25.1, the risk is essentially the same as for someone with a BMI of 24.9 (in the acceptable range) whereas at 29.9, the risk is essentially the same that of someone in the obese group with a BMI of 30.1. From this, it is clear that the choice of the cut-off points to make the categories for guiding clinical practice is somewhat arbitrary. (One suspects that the choice of 25 rather than other cut-off points such as 24.9 or 25.5 was related to digit preference.) However, the consequence of the continuous relationship is that risk reduces even when a small amount of weight is lost. Although the categories are based on observational studies a number of trials confirm the view that small changes in weight lead to reductions in some important intermediate markers even though the subjects did not lose enough weight to reach the healthy/acceptable weight range (see below). Hence it is important not to discourage patients from achieving some level of modest weight reduction by telling them what their weight 'should be'. This 'ideal' weight might be so far below the person's current weight that it is an impossible goal.

In addition, it should be realised that BMI:

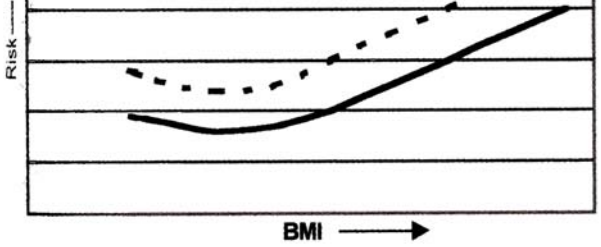
> . . . appears to be a good measure of overweight and obesity in sedentary adult white men and women from Europe and North America. However, since body mass index does not distinguish between weight due to muscle and that due to fat, its relationship with obesity (excess fat) is likely to differ in individuals and populations who differ in body build (e.g. highly muscular individuals) and body proportions (e.g. individuals with unusually long or short legs).[3]

This means that when assessing individuals the BMI category is a good screening tool, but is not a diagnostic tool. Athletes such as footballers often have a BMI in the overweight range and yet they are not over fat. Conversely, someone can be in the healthy weight range and still be over fat. This means that everyone close to the boundary of healthy weight and overweight should be further assessed, regardless of classification. Ultimately, the advice you give someone should be in the light of their other risk factors and lifestyle.

Should cut-off points be different for Aboriginal and Torres Strait Islander people?

A number of studies have been conducted showing that, for a given BMI, Aboriginal people have a greater percentage of body fat than do Caucasians.[15] (The opposite may be true of Torres Strait Islanders, but this is less studied). This has lead to the suggestion that the cut-off points to define categories should be different for Aboriginal people, e.g. that the overweight category might be more like 23–<28 than 25–<30. While this might be true from a theoretical point of view, one needs to consider several other things.

The current BMI categories are based on risk of mortality, not the proportion of body weight that is fat, and are simply cut-off points along a continuous distribution. Different cut-off points could also have been chosen for Caucasian populations. As indicated above, people near the boundary of acceptable and overweight should be assessed individually and their other risk factors taken into account. There are few studies of the association between BMI and mortality in Indigenous people to determine whether the BMI associated with the lowest risk of mortality in that group



is lowest mortality in any other grc

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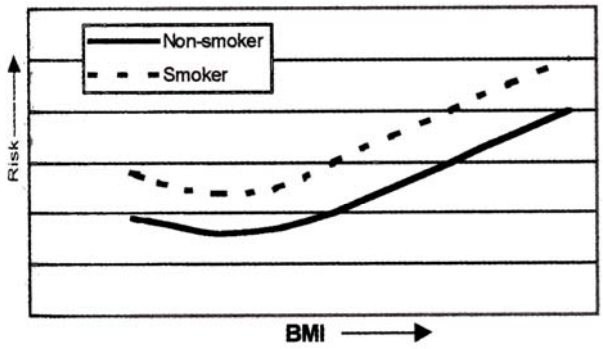

Figure 1: Schematic diagram of risk levels in smokers and non-smokers

The curve for smokers is at a higher level than that for non-smokers and so there are two different ways for an overweight smoker to reduce his/her risk — one is to stop smoking and the other is to lose weight. But does this mean that different cut-off points for healthy weight should be set for smokers and non-smokers? Are we interested in setting cut-off points to give different groups the same absolute risk, or to define relative reductions associated with one risk factor across groups? If it is the first, then we should set different cut-off points for smokers and non-smokers; if it is the second, then the cut-off points would be the same. The figure probably reflects the general pattern for Aboriginals (dotted line) and non-Indigenous Australians (solid line). And so the question arises: what is the cause of the offset? Is it the central obesity (see below)? Is it some other factor, such as low birthweight leading to long-term differences in adult disease? Or perhaps the shape of the two curves are not similar but different and there really should be different cut-off points. More research is needed before we can really start to answer these questions!

As shown below, important improvements in some aspects of heart disease and diabetes risk factors can be achieved by losing 3–5 kg, even though the final weight is still in the overweight or obese range. Presumably larger improvements could be achieved with greater weight loss. This means that we should be careful not to discourage people from losing some weight because we have set goals that are too far away to achieve.

[Editor: The relationship of birth weight and early growth to adult health may be more relevant than ethnicity for explaining differences in health outcomes at different BMI levels in different populations. ‘In general the most unfavorable growth pattern is smallness and thinness at birth, continued slow growth in early childhood, then acceleration of growth so that height and weight approach the population means . . .’ The fetal origins of adult disease: no longer just a hypothesis and may be critically important in south Asia. R. Robinson BMJ 17 Feb 2001.]

Central versus peripheral adiposity

In Caucasians a common pattern is that men tend to put on excess weight on their abdomen (an apple shape) and women on their hips and thighs (a pear shape). Visceral (stomach) fat is more metabolically active than subcutaneous fat and is associated with dyslipidaemias, hypertension and type 2 diabetes[3] and this is one explanation for the observation that Caucasian men have more heart disease than Caucasian women with the same BMI. Among Aboriginal people, both men and women tend to put on excess weight on their stomachs. The different distribution of fat may be one reason why Aboriginal people have higher levels of chronic diseases than Caucasians.

The WHO committee's recommendations for action (see below) class central obesity as an additional risk factor to overweight[3] but only bring in a consideration of central obesity for people who are in the overweight range. Given the comments that the cut-off points are screening guidelines and not diagnostic for excess fat in the body, and the high prevalence of large waists in Aboriginal people, it would probably be sensible to advise people in the 'healthy weight range' who clearly have pot bellies to lose some weight. However, measuring waists is difficult. There are at least three different definitions (at the narrowest point, where the belly button is and the midpoint between two particular bony landmarks) and these do not give the same circumference as they are not in the same place. Hence, it would not seem sensible to make recommendations about numerical cut-off points. Therefore, at present it would seem best to advise staff to use their eyes rather than a tape measure to identify who should be advised to lose weight and for them to ask clients about whether skirts and trousers are getting looser around the waist.

[Editor: WHR is not specifically mentioned in the protocol. Is this appropriate given the high prevalence of high WHR in the A&TSI population, particularly among women? Are the WHO guidelines appropriate for population where poor growth in adult life is prevalent, and where smoking rates are high? Would it be more appropriate to advise loss of belly fat at BMI levels below the 25.0 to 29.9 level?

Waist and WHR are not easy to measure, and ratios and changes in ratio not concepts which are intuitively easy. The editorial committee chose to include a mention of the importance of losing 'belly fat' even if the overall BMI is in the healthy range, but believes routine measurement of WHR may not be well performed or interpreted; guideline threshold waist circumference values are provided.]

Prevalence of overweight and obesity

The available information about overweight and obesity in NT Aboriginal adults is derived from the 1994 National Aboriginal and Torres Strait Islander Survey (NATSIS) and is summarised in 'The health and welfare of Territorians' [16]:

> . . . One problem with interpreting the data is that many of the respondents declined to be measured, so it is not clear whether some of the differences in weight reflect differences in participation rather than size. The available information does not allow an assessment of whether the prevalence of overweight and obesity varies substantially with age.

In the Katherine region, where virtually everyone was measured, the survey found that 46% of Aboriginal adults were overweight or obese (table 6).

This compares with the 1995 National Nutrition Survey figure of 55% for Australians.

The low measurement rate means that the NATSIS results might not be representative in all regions of the NT. Therefore, it is possible that the proportion of obese Aborigines in some parts of the NT is about double the national prevalence, while in other parts the proportion is much lower. Despite the low measurement rate the response rate for interviews was very high, and so we were able to compare the reported characteristics from the questionnaires of those who were measured with those who declined to be measured. From these analyses, we concluded that, at the national level, there was no reason to think that the national figures for weight distribution in Indigenous Australians were affected by the extent of non-measurement[8] but this might not be true for small areas. The prevalence of overweight and obesity is higher in Torres Strait Islanders than in Aboriginal people (table 5). When comparing with the all-Australian prevalence (table 4) the difference in age structure of the two populations should be remembered. Among non-Indigenous Australians, overweight and obesity tends to increase with age, whereas in many locations older Aboriginal people are less overweight than younger adults. This means that the prevalence of overweight and obesity in Aboriginal people and the all-Australian population are more different than appears to be the case at first glance.

Table 6: NT Aboriginal adults 18 years and older, 1994[16]

	Number		Per cent			
Group	Surveyed	Measured	Underweight <20	Acceptable 20–25	Overweight >25–30	Obese >30
NT ATSIC Region						
Aputula	4380	570	10.9	50.0	37.0	2.2
Alice Springs	2420	1010	7.9	28.7	32.7	30.7
Tennant Creek	2020	590	10.2	23.7	40.7	25.4
Katherine	3860	3780	14.0	38.4	30.1	17.5
Nhulunbuy	3650	2430	32.2	35.5	15.3	16.9
Jabiru	4790	2600	18.5	39.8	34.0	7.7
Darwin	4400	3220	8.4	28.6	33.5	29.5

Table 7: Interventions according to BMI – WHO Expert Group3

BMI range	Other conditions	Recommendation
18.5 to 24.9		avoid becoming overweight
25.0–29.9	no risk factors	avoid weight gain monitor weight, e.g. yearly intervene if weight gain occurs
25.0– 29.9	and risk factors e.g. high abdomen/hip ratio, hypertension, hyperlipidaemia, glucose intolerance, type 2 diabetes, family history of type 2 diabetes, or premature coronary heart disease	cease smoking, increase physical activity, reduce saturated fat, moderate weight loss
30.00 and over	as for the overweight group, but moderate weight loss is a priority as risk factors and conditions are generally more marked	

Evidence for the benefits of losing 3–5 kg

Although observational studies have shown that being obese increases mortality, the 1995 WHO committee noted that no intervention studies had shown that obese people who lost weight had lower mortality or fewer morbidites such as strokes or heart disease.[3] A Cochrane review to examine whether this situation has changed is currently underway. This lack of information about the benefits of weight loss when no other abnormality existed lead the WHO Expert Committee to make recommendations about weight loss in the overweight and obese that depended on the presence of other factors (table 7).

One of the problems is that there are two sets of studies which investigate different interventions and have different endpoints. Studies investigating how to help people lose weight have tended to report only weight loss as the outcome. Studies with change in chronic disease risk factors as the endpoint have tended to use combined lifestyle and diet interventions rather than weight loss alone, and so it is not clear whether the results are due to weight loss, the other interventions or a mix of the two. However, there is some evidence that modest weight loss can alter some important intermediate risk markers and this supports the recommendation for modest weight loss in those with risk factors.

Mulrow et al.[17] reviewed the effects of weight loss on blood pressure levels. The trials were generally conducted in people with an average weight of 84 kg and blood pressure of at least 190 mmHg systolic and/or 90 mmHg diastolic. Compared to no change in diet, a diet leading to a weight loss of 3–9% initial body weight decreased both systolic and diastolic blood pressure by 3 mmHg, although in neither case was this statistically significant due to the variability in results among the studies. They also compared weight loss to anti-hypertensive therapy and found that stepped care was better than weight loss for reducing blood pressure levels, but that there was an indication that weight loss would lead to a reduction in dose of medication. They concluded that it would be reasonable to delay

anti-hypertensive therapy in someone with mild hypertension who was committed to losing weight but that it is ‘unrealistic to expect a weight-reducing diet alone to achieve blood pressure control in patients with severe hypertension or in patients unmotivated to lose weight’. It is worth noting that many of the people in these trials did not lose enough weight to achieve the acceptable weight range. Although these results are useful, their outcome was change in blood pressure; data on change in stroke incidence or mortality would be more useful, but would require longer and larger studies.

The effect of weight loss alone on cholesterol levels is not so clear cut from randomised controlled trials because the trials have generally involved a diet that had a modified fat and cholesterol content rather than simple energy intake restriction. Hence, the effects on cholesterol levels or outcomes such as heart disease or mortality cannot necessarily be ascribed to weight loss alone. Thus, although multiple risk factor interventions do reduce cholesterol levels[18], their value overall depend on which outcomes are examined. In the primary prevention situation, they have only a very small effect on total mortality[19,20] although there is generally a larger effect when heart disease mortality and non-fatal cardiac events are grouped together.

Until recently, there was no trial examining whether the onset of diabetes could be delayed in those with impaired glucose tolerance (IGT). A trial in Finland compared advice regarding diet+exercise to a pamphlet.[21] The subjects were recruited by screening high risk individuals (such as relatives of diabetics), were aged 40–65 years and had a BMI of 25 or greater. The dietary intervention goals were: weight loss of 5% or more, total fat intake of less than 30% of energy, saturated fat intake of less than 10% energy, increasing fibre to 15g/4.2MJ, and frequent intake of whole grains, fruit, vegetables, low-fat milk and meat, soft margarines and mono-unsaturated oils. Moderate exercise of at least 30 minutes per day was recommended and subjects were advised to do both aerobic exercise and strengthening exercise for the large muscles. Diabetic status was determined in all but 40 of the 523 randomised subjects. The hazard ratio (a type of relative risk) was 0.4 (95% CI: 0.3–0.7) at six years of follow-up (i.e. those in the intervention group were only 40% as likely to have become diabetic as those in the control group).

By the end of the first year the intervention group had lost 4.2 kg compared to 0.8 kg in the control group and a greater proportion had changed various aspects of diet (except alcohol consumption) than the control group. However, success in achieving specific aspects of the intervention ranged from 25% for fibre intake to 86% for exercise. In addition to this intention-to-treat analysis, other analyses were done according to compliance. In the intervention group, those who lost 5% or more of body weight had a hazard ratio of 0.3 compared to those with less weight loss. Among those who did not achieve the weight loss goal, those who did achieve the activity goal of four hours per week had a hazard ratio of 0.2 compared to those who did not achieve the activity goal.

Other benefits were reported from this trial. The reduction in blood pressure level is consistent with that reported in Mulrow et al.’s meta-analysis.[17] Triglycerides, which are commonly raised in Aboriginal people, also fell although there was no change in cholesterol level. As other work has shown that controlling hypertension in diabetics reduces the incidence of important outcomes such as renal failure, these findings are presumably very beneficial for this group of people.

In 2002, similar results were reported from an American study.[22] This study is particularly interesting because 45% of the study population were from minority populations — African, Hispanic and Native Americans — who have very high rates of diabetes. All of these sub-groups had the same benefit as the larger group. From this it is reasonable to conclude that the studies conducted overseas can be extrapolated to Aboriginal people in Australia. The US study also tested metformin in another arm and found that the lifestyle intervention was more effective in delaying progression to diabetes than the metformin.[22]

The BMIs seen in the participants in the trials are quite commonly seen in Aboriginal groups. It is worth noting that an average weight loss of 4.2 kg in people with a average baseline BMI of 31.3 means that about half the people in the Finnish trial were still in the obese category at the end of the first year. In other words, these benefits can be achieved without people having to lose enough weight to get down to the healthy weight range. Presumably, if people manage to lose more weight, they will have additional benefit. In addition, the physical activity was moderate, it was not strenuous sport-type 'exercise'.

Table 8: Selected baseline characteristics and results from the Finnish trial[20]

	Intervention group n=256	Control group n=250
Baseline characteristics		
BMI	31.3	31.0
Total cholesterol (mg/dL)	215	215
Triglycerides (mg/dL)	154	158
Systolic blood pressure (mmHg)	140	136
Diastolic blood pressure (mmHg)	86	86
Change at 1 year		
Weight (kg)	-4.2	-0.8
Total cholesterol	-5	-4
Triglycerides	-18	-1
Systolic blood pressure (mmHg)	-5	-1
Diastolic blood pressure (mmHg)	-5	-3
Diabetes at six years (hazard ratio)	0.4	1.0

All changes at one year are significantly different between the intervention and control groups except that for total cholesterol

The role of physical activity in prevention

Decreasing physical activity rather than overeating seems to be responsible for the epidemic of overweight in the Western world. In fact, contrary to popular perception, we now consume less energy, on average, than our grandparents, although there are undoubtedly exceptions to this. Research in the UK has documented a greater decline in physical activity levels than energy intake over the past few decades, although this may not be the case in all groups.[23] Encouraging enjoyable physical activity in children is an important strategy for long-term prevention of overweight and obesity.[23]

It is certainly true that most people are unable to exercise for the amount of time that would be needed to achieve a large weight loss without dieting. As the Finnish study above showed, weight loss with mild physical activity and mild diet occurred at a rate of about 1 kg per three months. Despite this, the impact on health was notable. As far as weight control itself is concerned, the role of physical activity is to prevent the slow weight gain that tends to occur throughout life and so this should be encouraged in all age groups. It is also important to ensure that people understand that activities like walking, dancing or housework are being encouraged as there is a tendency for people to think of exercise only in terms of what is aerobic and very energetic. The secondary analysis of the Finnish study — which found that people who did the exercise but did not lose weight had a slightly larger benefit than the intervention group as a whole — is very intriguing. Does this suggest that physical activity might be an independent protective factor in its own right and not simply a method to help people to lose weight? (That is, in the same way that an overweight smoker who quits smoking but does not lose weight will reduce his/her risk of heart disease.) If so, then people should be told to increase their activity for its own benefit and not simply because activity helps to control weight (and, therefore, not to give activity up if weight loss does not occur). However, a trial of activity in IGT without concurrent dietary interventions to reduce the subjects' weight has not yet been done to answer this question.

One difference between the Finnish and US trials is that the US trial did not include the large muscle strengthening exercise in its program, but had only encouraged 'physical activity of moderate intensity, such as brisk walking'. However, it should be noted that the subjects in the intervention groups of both trials received numerous counselling and encouragement sessions. Hence the same level of result could not be expected from a single five minute talk.

[Editor: The diet changes and physical activity achieved during the Finnish and US studies were not 'mild'. They were significant and sustained. While it is important to emphasis that small changes in weight are achievable and can provide great benefits in health, the behavioral changes in terms of diet and PA need commitment and support to be achieved and sustained.]

Infant birth weight and maternal weight

As noted above, the cut-off points describing weight categories are based on risk of mortality. However, the risk of low birth weight babies increases as maternal pre-pregnant weight decreases. This outcome does not appear to have been considered by the Expert Committee when they lowered the cut-off for healthy weight.[3] Similarly, the literature discussing the relationship between maternal weight and infant outcomes does not usually consider effects on maternal health in the long-term. Hence, it is not immediately obvious how to weigh up the risks and benefits identified in the two distinct sets of literature for a population such as Aboriginal people, who have a high rate of low birth weight and also high levels of diabetes and other adult chronic disease.

There are two distinct reasons why an infant may be small when it is born. Firstly, it may be born early (preterm) but be within the expected size range for its gestational age. Secondly, it may be small for its gestational age. This is variously called small-for-dates, small-for-

gestational age or intra-uterine growth retardation (IUGR). An infant may be both preterm and growth retarded. Although the relative contribution of preterm delivery and IUGR to low-birth weight in the Aboriginal population is the subject of intense argument at present[24–26] both are related to maternal pre-pregnant weight and/or gestational weight gain.[27] Table 9 shows the factors associated with the two types of low birth weight that were identified by Kramer[27] in a review that considered only deliveries in women with no underlying chronic illness. Studies published in the last decade indicate that there are some other important factors for preterm delivery, including those for the underlying chronic illnesses not considered by Kramer.[27] In particular, urogenital infections, including bacterial vaginosis, and diabetes[28,29] may be important in Aboriginal women. Studies on Aborigines in WA [30–32] and the NT agree that the factors described elsewhere in the world are important influences on birth weight.

Rae[33] examined the associations between birth weight and various antecedents using data routinely recorded in antenatal charts in two Top End communities between 1983–86. She found that the predictors for having an IUGR infant were maternal pre-pregnant weight of less than 43 kg, gestational weight gain of less than 8 kg, attending for ante-natal care after 20 weeks, maternal age less than 19 years and maternal parity less than four. The effect of pre-pregnant weight was stronger than the effect of gestational weight gain (39–62 g versus 36–40 g in birth weight for a 1 kg difference in maternal weight respectively). Cigarette smoking was not well documented in the antenatal records.

Sayers and Powers[32] examined the antecedents of 503 Aboriginal infants born at Royal Darwin Hospital between 1987–90. They found that 15% of the IUGR was attributable to low maternal weight and 10% to cigarette smoking. Only diabetes predicted preterm delivery and they estimated that 5% of the preterm birth could be attributed to it. Overall, they estimated that 28% of the low birth weight could be attributed to the mothers having a body mass index of less than 18.5 kg/m^2 at delivery, and 18% could be attributed to mothers smoking more than half a pack of cigarettes per day.

It is worth noting the low weights described by the two authors. The 43 kg of Rae[33] is equivalent to a BMI of 16.8 in a woman who is 160 cm tall. The 18.5 BMI of Sayers and Power[32] was a post-partum BMI and therefore presumably somewhat higher than the women’s pre-pregnant weight. Diabetes and gestational diabetes are becoming increasingly common in Aboriginal people at young ages. Given the association with obesity, one would presume that gestational diabetes is occurring in the heavier women rather than the thin women, but as the Midwives Collection does not collect maternal weight it is not possible to confirm this.

Table A.3: Predictors of intra-uterine growth retardation and preterm birth[27]

	Intra-uterine growth retardation	Pre-term birth
Established determinants (Kramer 1987)		
Direct	infant sex race/ethnic origin maternal height maternal pre-pregnancy weight paternal height and weight maternal birthweight parity prior low birthweight infant gestational weight gain energy intake general morbidity malaria maternal cigarette smoking alcohol consumption tobacco chewing	Direct maternal pre-pregnancy weight prior preterm birth prior spontaneous abortion maternal cigarette smoking in utero diethy-stilboestrol exposure
Indirect	very young maternal age socio-economic status (including maternal education)	
		Other proposed predictors of preterm: maternal diabetes, urogenital infections, bacterial vaginosis, placental, cervical or uterine abnormalities

Can low birth weight be improved by altering maternal weight?

The studies described in the previous section are all observational. Although they describe associations that allow identification of potentially useful interventions, they do not test whether the proposed interventions are efficacious. Without proper testing of a proposed intervention, it is impossible to distinguish between the predictive factors that are reversible causes and the predictive factors that are only indicators to identify women at higher risk.

Perhaps the most supportive evidence that altering a woman's weight during pregnancy could alter the weight of the infant comes from the unintended experiment that occurred in Holland during the Second World War. The amount of food available was drastically reduced from about 1800 kcal (7.5MJ) per day to 580 kcal (2.5MJ) per day at the height of a siege.[34] As hospital records in the affected towns were kept carefully, the effect of food restriction at different points during pregnancy could be examined. The birth weight of those born during the famine was more than 300 g lower, but the birth weight of those conceived during the famine was not different from that of non-famine infants.

It would not be ethical to reproduce this study by starving a group of pregnant women. However, the opposite approach — comparing the effect of a supplement versus no supplement or placebo — has been tried in a number of countries. Some trials have tested a combination diet that increased energy and protein, while others tested the effect of increasing just the protein content of the diet. Considering only randomised controlled trials, Kramer[35] concluded that a balanced supplement of protein and an extra 1 MJ/day of energy caused an increase of 21 g/week (95%CI: 9 g; 33 g) in maternal weight gain, an increase of 30g (95%CI: 1 g; 58 g) in birth weight, a non-significant increase of 0.2 cm (95%CI: 0.0 cm; 0.4 cm) in birth length and no difference in head circumference. There was a reduction in both IUGR and preterm birth, both having odds ratios of about 0.8 (95%CI for preterm birth: 0.6; 1.1) but neither were statistically significant. Thus, the effect on birth weight was only about half that predicted from the Dutch results. Kramer (1993) also noted that the effect of supplementation on birth weight was not larger in women with lower pre-pregnant weight, as had been predicted from large observational studies (e.g. Naeye et al[36]).

By contrast, the infants of women randomised to a high protein supplement but no additional energy intake were significantly lighter than control infants (-64 g, 95%CI: -124 g; -3 g) and their mothers gained less weight than the control mothers.[35]

The results of Naeye et al[36] had also predicted that birth outcomes in obese women would be improved if gestational weight gain were limited. In another review, Kramer[37] examined trials of restricting the intake of obese pregnant women to between 1200–2000 kcal/day. Contrary to prediction, there was a statistically significant reduction in birth weight of 153 g (95%CI: -256 g; -30 g) in the women who were dieted. There was no reduction in the incidence of pregnancy-induced hypertension or pre-eclampsia and a non-significant reduction in the preterm delivery rate (OR = 0.5, 95% CI: 0.1; 2.6).

To summarise, increasing maternal intake by about 1 MJ (400 kcal) using a balanced diet containing a range of nutrients (table 10) will cause a small increase in infant birth weight, probably in the vicinity of 30 g. At

present, there is no evidence that increasing intake in underweight women will have a greater effect on increasing birth weight than for normal weight women. Therefore, it would seem reasonable to advise women who have a BMI of less than about 18.5 and have a poor quality diet to improve their dietary quality and put on a small amount of weight before becoming pregnant. But there is no reason to advise women in the healthy weight range to 'fatten up' during pregnancy beyond the usual recommendations for gestational weight gain. Advising obese women to restrict their intake will decrease the birth weight of their infants.

In addition it should be remembered that there are a number of other factors which lead to low birth weight — such as cigarette smoking[38] — that are common in Aboriginal women. These factors may have at least the same magnitude of effect on birth outcomes as maternal weight, and advice about them should also be given. The information available to date would suggest that smoking rates in pregnant women did not change in the first set of communities in the Strong Women program.[39] Hence this would seem to be a useful additional focus of intervention for pregnant women.

Table 10: Energy content of typical foods recommended for increasing the dietary intake of pregnant women

Food	kJ	kcal
1 slice toasting bread and 1 tsp margarine	611	145
1 medium orange or large apple	255	61
300 ml reduced fat milk	624	149
30g meat (about size of 1 matchbox)	360	86

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