

National Evidence Based Guidelines for the Management of Type 2 Diabetes Mellitus

Part 2

Primary Prevention of Type 2 Diabetes

Prepared by
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for the Diabetes Australia Guideline Development Consortium

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2.0 Primary Prevention of Type 2 Diabetes

2.1 Introduction

Aim of the guideline

This guideline covers issues related to the primary prevention of Type 2 diabetes. Its aim is to inform and guide health promotion and preventative activities for Type 2 diabetes with evidence based information on what non-pharmacological interventions work and what does not. While this guideline contains information that would be relevant for a range of audiences, the focus of the systematic review is primarily on the efficacy and effectiveness of one to one interventions. The guideline targets clinicians, health promotion practitioners, planners and policy makers.

Since certain risk factors are common to both the Prevention Guideline and the Case Detection and Diagnosis Guideline, where possible overlap has been minimized. For example, family history as a risk factor for the development of Type 2 diabetes is covered in the Case Detection and Diagnosis Guideline but was omitted from the Prevention Guideline as it is a non-modifiable risk and therefore is not subject to preventative interventions.

Methods

In addition to the methods used to identify and critically appraise the evidence to formulate the guideline recommendations which are described in detail in *Part 1* of the document, the Project Management Team reviewed and checked each step of the methods process and:

- repeated a selection of the searches
- double culled the yield from a selection of the database searches
- double reviewed the majority of the articles used as evidence references
- checked all recommendations, evidence statements, evidence tables and search strategy and yield tables

Guideline Format

Issues identified by the EWG and from the literature as critical to the primary prevention of Type 2 diabetes are shown in point 2.2 (next page).

Each of these issues is addressed in a separate section in a format presenting:

- ***Recommendation(s)***
- ***Evidence Statements*** - supporting the recommendations
- ***Background*** - to issues for the guideline
- ***Evidence*** - detailing and interpreting the key findings
- ***Summary*** - of major evidence found
- ***Evidence tables*** - summarising the evidence ratings for the articles reviewed

For all issues combined, supporting material appears at the end of the guideline topic and includes:

- ***Evidence references***
- ***General references***
- ***Other references identified***

2.2 Issues for the Primary Prevention of Type 2 Diabetes

- Does obesity in adult life and/or in childhood increase the risk of Type 2 diabetes?
- Is the pattern of fat distribution an additional contributor to total adiposity in determining the risk of Type 2 diabetes and how does this vary with ethnicity?
- What simple measures can be used for body fat distribution?
- What is the evidence that physical activity can decrease, and sedentary behaviour increase, the risk of Type 2 diabetes?
- What simple measures can be used to determine physical activity and inactivity?
- Do high-fat diets increase the risk of Type 2 diabetes and how is this affected by fat type?
- Does GDM increase the subsequent risk of Type 2 diabetes in mothers and/or infants?
- How does weight at birth affect later risk of Type 2 diabetes?
- Do low glycaemic index (GI) diets decrease risk of Type 2 diabetes?
- Does psychosocial stress increase the risk of Type 2 diabetes?
- What forms of diet and physical activity are recommended to prevent excessive weight gain?

2.3 Summary of Recommendations

| Major Recommendations |
|---|
| <ul style="list-style-type: none">• Regular physical activity is recommended to reduce the risk of Type 2 diabetes |
| <ul style="list-style-type: none">• Since obesity is associated with an increased risk of Type 2 diabetes, interventions to reduce obesity may reduce the risk of Type 2 diabetes |
| <ul style="list-style-type: none">• Abdominal obesity is an important indicator of increased risk of Type 2 diabetes in all ethnic groups and should be a particular focus of weight loss programs |
| <ul style="list-style-type: none">• Individuals at risk of developing Type 2 diabetes should have dietary intake assessed and should receive individualised dietary advice and continued dietetic support• Individuals at risk should consume a diet with <30% energy as fat and <10% energy as saturated fat• Diets of low energy density and containing a wide range of carbohydrate foods rich in dietary fibre and of low glycaemic index (cereals, vegetables, legumes and fruits) are recommended to reduce the risk of Type 2 diabetes |

Other Recommendations

- Waist circumference (> 100 cm for European men and > 90 cm for European women) should be used in addition to body weight and body mass index (BMI) to identify individuals who should seek and be offered weight management programs

- Physical activity should be measured in free-living subjects by:
 - movement recorders, particularly the pedometer
 - questionnaires focussing on leisure-time activities
 - heart rate monitoring
- Functional aerobic capacity should be measured from predictive equations based on gender, age, self-reported physical activity and body composition or BMI

Identification of women with GDM would allow:

- Postnatal clinical interventions in those with diabetes persisting after delivery
- The option to use preventive methods to reduce the risk of Type 2 diabetes

- In view of the association between low birth weight and the later development of diabetes, studies are required to evaluate interventions aimed at reducing low birth weight and the impact this has on the development of Type 2 diabetes

- Programs of diet and exercise education in children should include parental involvement and use behavioural techniques to reinforce lifestyle change
- Adults who have achieved weight loss require long-term programs involving diet, exercise and social support to prevent weight regain, however the effectiveness of specific long term programs requires further evaluation

- More research is required to assess the impact of psychosocial stress or a major depressive episode in affecting the risk of Type 2 diabetes

2.4 Recommendations

Section 1: Obesity in childhood and adult life

Issue

Does obesity in adult life and/or childhood increase the risk of Type 2 diabetes?

Recommendation

Since obesity is associated with an increased risk of Type 2 diabetes, interventions to reduce obesity may reduce the risk of Type 2 diabetes

Evidence Statements

Childhood

- Underweight in childhood or adolescence may increase the risk of central adiposity, insulin resistance and diabetes in adult life but more research will be required to substantiate this
Evidence Level III-2
- Rapid weight gain in childhood may increase insulin resistance
Evidence Level III-2
- Intervention programs to increase exercise levels in addition to dietary change can reduce obesity in children
Evidence Level II (see Section 11)
- Programs in children aimed at reducing sedentary behaviour produce more body fat loss after a year than programs aimed at increasing exercise activity or programs that combine both approaches
Evidence Level II (see Section 11)

Adult life

- Obesity in adult life increases the risk of diabetes in both men and women; the greater the magnitude of the obesity, the greater the risk
Evidence Level III-2
- Weight gain in adult life increases the risk of diabetes
Evidence Level III-2
- Risk of diabetes increases with the duration of the obese state
Evidence Level III-2
- In people with impaired glucose tolerance, obesity increases the risk of progression to Type 2 diabetes
Evidence Level III-2
- Weight loss is associated with reduced progression of IGT to Type 2 diabetes
Evidence Level II
- Targeted dietary and exercise programs based on an empowerment model comprising diabetes awareness sessions, exercise groups and cooking demonstrations have prevented weight gain in a high-risk population
Evidence Level III-2 (see Section 11)
- Although body weight can be readily lost during active supervised treatment, it is often gradually regained during the subsequent unsupervised period
Evidence Level II (see Section 11)
- In men taught dietary and exercise strategies, waist circumference may decline during active supervised treatment and remain reduced for up to 2 years
Evidence Level III-2 (see Section 11)
- Men and women who have successfully maintained long-term weight loss report the continued consumption of a low energy, low fat diet
Evidence Level II (see Section 11)
- In women, an ad libitum low fat diet is superior to a low energy diet in maintaining weight after a major weight loss
Evidence Level II (see Section 11)
- A program of combined diet and exercise is more effective in maintaining weight loss than either diet alone or exercise alone
Evidence Level II (see Section 11)
- Overweight men with impaired glucose tolerance who followed a long-term program of diet and exercise exhibited reduced all-cause mortality after a period of 12 years
Evidence Level III-2 (see Section 11)

Background - Obesity

Obesity is a serious public health problem in Australia. Moreover, its prevalence is rising in both men and women in all five-year age groups from 25 to 64 years (Risk Factor Prevalence Study Management Committee, 1984). Overall, 75% of men and 65% of women aged 45 - 64 are overweight or obese (McLennan and Podger, 1997). Obesity is also a growing problem in children in Australia. NHMRC data from 1985 indicated that 4% of boys and 6% of girls between the ages of 9 and 15 were overweight (ie they had body mass index (BMI) values above the 95th percentile) (Moon et al, 1998). Since obesity is an important risk factor for development of Type 2 diabetes (see below), its increasing prevalence in Australia has important implications for diabetes incidence.

Both within and between populations there is a strong association between body weight and diabetes prevalence. A strong association ($r = 0.89$) has been reported between the prevalence of diabetes and population mean body weight based on data from 11 countries of widely ranging socioeconomic development (West and Kalbfleisch, 1971). Himsworth in 1949 observed that diabetes-related mortality fell markedly in the UK with food rationing during the first and second World War, a probable consequence of reduced population body weight in older people (Himsworth, 1949). A much more frequent occurrence worldwide, is an increase in diabetes prevalence with increased prevalence of overweight and obesity, as has happened in Australia (Welborn et al, 1968; Glatthar et al, 1985; Risk Factor Prevalence Study Management Committee, 1990). The relationship between increased obesity and development of diabetes is particularly evident in populations that have rapidly made a transition from a traditional to an urbanised way of life. Examples are the Pima Indians (Knowler et al, 1990), Nauruans (Zimmet et al, 1990) and in Australian Aboriginal people (Rowley et al, 1997).

Determination of childhood and adolescent obesity

In children between the ages of 2 to 18 years the presence of obesity should be determined by measurement of height and weight and reference to standard gender-specific growth charts.

Determination of adult obesity

A simple measure to determine obesity in adults is the body mass index (BMI). This is calculated as weight (kg)/ height ² (m) and is significantly correlated with body fat content (National Institutes of Health Expert Committee, 1998). The health risks associated with various BMI categories are shown in Table 1.

Table 1: Body Mass Index criteria

| <i>BMI</i> | <i>Category</i> | <i>Health risk</i> |
|------------|-------------------|----------------------------|
| <18.5 | Underweight | Long term hazard to health |
| 18.5-24.9 | Normal | Healthy range |
| 25.0-29.9 | Overweight | Some risk to health |
| 30.0-34.9 | Obesity class I | High risk to health |
| 35-39.9 | Obesity class II | Severe risk to health |
| >40 | Extreme obesity | Very severe risk to health |
| | Obesity class III | |

It must be noted however, that although BMI is simple to determine, it is an imperfect measure of total adiposity. When BMI is compared with the more accurate standard of densitometry, correlation coefficients are found to lie between 0.6 and 0.8 (Spiegelman et al,

1992). The relationship between BMI and total adiposity moreover may differ between Europeans and other ethnic groups such as Asian Indians and Australian Aborigines. For example, for any given BMI, Aboriginal people have more body fat than Europeans (Rutishauser and McKay, 1986).

Evidence – Childhood Weight and Obesity in Adult Life

Underweight in childhood and the risk of diabetes in adult life

In childhood the presence of Type 2 diabetes is uncommon although it may be found in some sedentary, severely obese adolescents and its prevalence is increasing (Glaser, 1997; ADA, 2000). Nevertheless, body weight in childhood may influence the risk of diabetes in later adult life. A study of 2,000 Americans aged 50 years or more, has indicated that self-reported underweight as a child or as a teenager was associated with a slightly increased risk of Type 2 diabetes in adult life [Relative Risk (RR) 1.3 and 1.4, respectively] compared to those who had maintained an appropriate childhood or adolescent weight (Holbrook et al, 1989). Moreover, the relative risk was further increased in those who reported underweight in childhood and overweight (BMI>26) in middle-age (RR 1.7, $p < 0.01$). Prospective studies, based on measured rather than self-reported body weight are necessary to fully delineate the effects of childhood and adolescent underweight on diabetes risk. Evidence relating to the question of whether the low weight in childhood and adolescence leading to increased risk of diabetes in adult life reflects low birth weight or intrauterine growth retardation will be discussed separately in Prevention Issue 7.

Overweight in childhood is related to adult obesity

As yet there is little direct evidence linking obesity in childhood to increased risk of diabetes in adult life and more studies are needed in this area. Nevertheless, overweight or obesity in childhood appears strongly related to the presence of obesity in adulthood. For example, in the Minneapolis Childrens' Blood Pressure Study, weight and BMI at age 7 years were closely related to young adult weight and BMI (Sinaiko et al, 1999). In addition, the Bogalusa Heart Study, indicated that in a biracial cohort of 783 children, followed from age 13-17 until age 27-31 years, excess weight in adolescence (ie BMI above the 75th percentile) persisted into young adulthood (Srinivasan et al, 1996). Once overweight children have become overweight adults, they are then at an increased risk of developing diabetes (evidence outlined above).

Evidence relating obesity in childhood to diabetes risk

A prospective study followed 137 Afro-American children from the Philadelphia Perinatal Collaborative Project for 35 years (Hulman et al, 1998). In this study, BMI at age seven years correlated with adult weight, and body weight at age 14 and was negatively correlated with measures of insulin-stimulated glucose utilisation indicating that obese adolescents are at greater risk for developing insulin resistance (Hulman et al, 1998). Similarly, a study in 1,258 Pima Indian children aged 5-19 years found that increased weight in childhood and adolescence was predictive for later development of diabetes (McCance et al, 1994). The rate of weight gain in childhood may also be of importance. In a longitudinal study in 152 black South African children examined until age 7 years, rapid weight gain during childhood was weakly related to insulin resistance as measured by homeostasis model assessment (HOMA) ($r = 0.18$, $p = 0.04$) (Crowther et al, 1998). While people who become obese in adult life are

at increased risk of developing diabetes, the risk is not greater if they were obese from childhood. In the study by Holbrook and coworkers (Holbrook et al, 1989), overweight adults who reported that they were overweight as children or teenagers were at a similar risk of diabetes to overweight adults who did not have a history of obesity in childhood or adolescence.

The effect of BMI at age 21 on later risk of diabetes was examined in 27,983 American men from the Men's Health Professionals Study. After controlling for age, family history, smoking status and subsequent weight change, men with a BMI above 27 kg.m⁻² at age 21 had 6.4 times the risk of developing diabetes in adult life than men who had a BMI < 21 kg.m⁻² at the same age (Chan et al, 1994). In a study of 895 Japanese men, it was also found that weight gain in early adult life (ie between the ages of 21 and 25 years) was significantly related to risk of Type 2 diabetes. If 10 kg or more was gained in this period, the Odds Ratio (OR) for risk of Type 2 diabetes was 3.9 with a confidence interval (CI) 1.5-10.0 (Sakurai et al, 1997).

Evidence – Obesity in Adult Life

Obesity in adult life and the risk of Type 2 diabetes

Many studies have demonstrated the importance of obesity in adult life as a major independent risk factor for the development of Type 2 diabetes. Of 27,983 American men aged 40-75 years, selected from the Men's Health Professionals Study and followed over a period of five years, 272 developed diabetes (Chan et al, 1994). The risk of Type 2 diabetes was strongly associated with the presence of obesity as measured by the BMI (weight in kg divided by height in metres, squared). Moreover, after adjustment for age, family history of diabetes and smoking, the relative risk of diabetes was found to increase continuously with increasing BMI. Men who were modestly overweight (BMI 25.0-26.9 kg.m⁻²) had a relative risk (RR) of diabetes only 2.2 times that of healthy men (BMI < 23 kg.m⁻²) but men with severe obesity (BMI > 35 kg.m⁻²) had a relative risk 42 times greater than that of non-obese men.

Clear positive associations between obesity, as measured by BMI, and increased risk of diabetes have also been demonstrated in American men enrolled in the Usual Care Group of the Multiple Risk Factor Intervention Trial (MRFIT) (Shaten et al, 1993). Associations were also evident in British men participating in the British Regional Heart Study (Perry et al, 1995; Shaper et al, 1997) or in the 20 year follow-up to the Wickham Study (Vanderpump et al, 1996); as well as in Norwegian men participating in the Finmark Study (Njolstad et al, 1998); in men from the Kinman Islands (Chou et al, 1998); in African American and European men participating in the Atherosclerosis Risk in Communities Trial (Liese et al, 1997); in Japanese American men (Kahn et al, 1996), and in Pima Indian men (Hanson et al, 1995).

There is also evidence that obesity increases the risk of diabetes in women. In a cohort of 113,861 women aged 30-55 years and initially free of known diabetes, BMI was the most striking predictor of diabetes eight years later. In comparison with women of BMI < 22 kg.m⁻² (relative risk =1.0), women at the upper end of the normal weight range (BMI, 25 kg.m⁻²) had a relative risk of 3.1. For women with a BMI of 30 kg.m⁻² the relative risk was 20 and for those with a BMI of 35 kg.m⁻² the relative risk was 60 (Colditz et al, 1990). A later study was based on a sub-cohort of 43,581 of these same American women derived after application of tighter exclusion criteria. In this study, the relative risk of Type 2 diabetes was 11.2 (CI 7.9-

15.9) among women with self-reported BMI in the 90th percentile (29.9 kg.m⁻²) relative to women whose BMI was in the 10th percentile (20.1 kg.m⁻²) (Carey et al, 1997). In one Norwegian study, the association between BMI and diabetes seen in women was attenuated after multiple adjustment for other risk factors whereas in men the association remained marked (Njølstad et al, 1998).

Weight gain in adult life and the risk of diabetes

Absolute weight gain throughout adulthood is also a significant independent risk factor for diabetes. For example, in men from the Mens' Health Professionals Study, it was found that men who had gained 5 kg since age 21 had no increased risk of diabetes in middle age [RR 0.9 (CI 0.5-1.8)]. In contrast, in men who had gained 15 kg the relative risk increased to 8.9 (CI 5.5-14.7) (Chan et al, 1994). Similarly, a study of 2,272 men from the Kuopio Ischemic Heart Disease Risk Factor Study in Finland indicated that men who had gained more than 30% in body weight since the age of 20 were 10.6 times as likely to develop insulin resistance than men who had remained within 10% of their earlier body weight (Everson et al, 1998). A study of 2,000 American men and women found that a weight gain of more than 4.5 kg between the ages of 40 and 60 years was associated with an increased risk of diabetes (RR 1.4, P < 0.05) (Holbrook et al, 1989). A weight fluctuation of 4.5 kg between the ages of 40 and 60 years also increased risk (RR 1.7, p< 0.01) (Holbrook et al, 1989).

Duration of obesity in adult life and the risk of diabetes

Risk of diabetes also increases with the duration of obesity. A study of 1,598 Japanese men employed by a railway company in Yokohama has shown that men who had a BMI greater than 27.8 kg.m⁻² for a period of up to ten years had 7.6 times the risk of developing diabetes compared with non-obese men. If obesity continued for up to 20 years the relative risk of diabetes climbed to 58.6 (CI 10.8-317.3) (Sakurai et al, 1999). In American Pima Indians, a population group at very high risk of diabetes, a highly significant association (p< 0.001) was evident between the duration of obesity and the incidence of obesity after adjustment for age, sex and current BMI. The adjusted incidence of diabetes was 24.8 cases per 1,000 person years in people with less than five years of obesity and 59.8 cases per 1,000 person years in people with at least ten years of obesity (Everhart et al, 1992). The relative risk however, in this population has to be seen within the background of known high incidence. There is also a need for prospective studies on this question in other populations including those of European origin.

Obesity and the risk of progression from IGT to Type 2 diabetes

The effect of obesity on the risk of progression from impaired glucose tolerance (IGT) to Type 2 diabetes has been examined in a study on 183 middle-aged Finnish men and women. These subjects were classified as having IGT by WHO criteria, then retested after 2 years. Subjects who were obese (BMI at or above 30 kg.m⁻²) were found more likely to progress to Type 2 diabetes than those who were not obese [OR 4.4 (CI 1.3-13.5), p=0.04] (Qiao et al, 1996). In South African Indians with IGT, who were followed for four years, the absence of obesity was also shown to be a significant predictive factor for reversion to normal glucose tolerance [OR 4.2 (CI 1.4-13.1), p=0.013] (Motala et al, 1997).

Beneficial effects of weight loss on diabetes risk

The recently published Diabetes Prevention Study (Tuomilehto et al, 2001) conducted in Finland with a 3.2 year follow-up showed that individual diet and exercise intervention could

reduce the risk of diabetes by 58% ($p < 0.001$) in a high risk population. This was achieved with 7 sessions in the first year and regular 3 monthly sessions after that during which goals were set on how to lose weight ($>5\%$), decrease total fat intake to $<30\%$ of energy, decrease saturated fat intake to $<10\%$ of energy, increase fibre intake to >15 g/1000 kcal consumed and to exercise for at least 30 min per day. The groups were individually given guidance to increase their exercise levels (30 min per day) with a combination of endurance exercise and supervised resistance training. The control group were set goals and given general written and oral information about diet and exercise initially and then annually but no specific individualized programs were offered. The incidence of diabetes in the intervention group was significantly lower than the control group (11% vs 23%). The intervention group lost 4.7% of their body weight significantly more than the 0.9% lost by the control group. Diabetes did not develop in any of the groups who achieved 4 or 5 goals, however significantly more people were able to achieve these goals in the intervention group (49 in the intervention group vs 15 in the control group, $p < 0.001$ for each of the goals). In those who did not achieve any of the goals the incidence of diabetes was 38% vs 31% in the intervention and control groups respectively (Tuomilehto et al, 2001).

The Diabetes Prevention Program was a major clinical trial conducted in the United States over 3 years in a group of people with IGT and therefore at high risk of developing Type 2 diabetes. The intensive lifestyle intervention included instructed on a low fat diet, exercising for 150 minutes per week and behaviour modification skills. The results show on average that this group had a 7% weight loss in the first year and sustained a 5% loss for the study's duration and maintained 30 minutes of exercise per day. A 58% reduction in the risk of developing Type 2 diabetes was found compared to the control group who received only basic diet and exercise advice. This study also included another group which was treated with Metformin which was effective in reducing the risk of developing diabetes by approximately 30% (National Institutes of Diabetes and Digestive and Kidney Diseases, 2001).

People who are severely obese ($BMI > 35 \text{ kg.m}^{-2}$) are at particularly high risk of developing Type 2 diabetes. However, the risk of type 2 diabetes and associated insulin resistance can be greatly ameliorated by substantive weight loss. This was clearly demonstrated in a study where a group of 109 patients from a total of 136 individuals with severe obesity received bariatric surgery to induce marked weight loss. In the control group who did not receive surgery and who were followed on average for 4.8 years, the conversion to diabetes (by WHO criteria) was 4.72 cases per 100 person years. In contrast, among those who received surgical treatment, and who were followed on average for 6.2 years, only one individual developed diabetes (this person overcame the effect of the surgery and regained the initial weight lost), a conversion rate of only 0.15 cases per 100 person years ($p < 0.0001$). Weight loss thus prevented progression to diabetes by more than 30-fold in this patient group (Long et al, 1994). In a second study, gastric bypass surgery was used to induce marked weight loss in severely obese individuals, and it was found that 121 of 146 patients (82.9%) with Type 2 diabetes reverted to normal glucose tolerance which was maintained for up to 14 years of follow up. Furthermore, 150 of 152 patients with glucose intolerance at baseline maintained normal levels of plasma glucose, glycosylated haemoglobin and insulin up to 14 years later (Pories et al, 1995).

Summary - Obesity

- There is moderately strong evidence that underweight in childhood and adolescence followed by over-weight in middle-age will increase the risk of diabetes in adult life. Further studies are required on this question however and links need to be made to both birth weight and the pattern of growth *in utero* (*Prevention Section 7*)
- As yet there is little evidence directly linking obesity in childhood to increased risk of diabetes in adult life, although it has been shown that black South African children who gain weight rapidly have increased insulin resistance. Nevertheless, there is good evidence that overweight children are at high risk of becoming overweight adults. Overweight adults are then at increased risk of developing diabetes
- There is strong evidence, from well designed longitudinal studies highly applicable to Australia, indicating that obesity in adult life increases the risk of Type 2 diabetes particularly in men
- There is strong to moderate evidence that weight gain in adult life increases diabetes risk
- There is moderately strong evidence in at least one ethnic population that the duration of adult obesity is related to diabetes risk, but further research is needed in other populations including those of European origin
- There is moderate evidence that obesity increases the risk that people with IGT will progress to Type 2 diabetes
- In severely obese patients surgically - induced weight loss not only markedly decreases the risk of progression from impaired glucose tolerance to diabetes, but also reverses established Type 2 diabetes
- Research will be required in the future to:
 - clarify the role of early life influences (fetal, neonatal, childhood) on the development of obesity and the risk of Type 2 diabetes in later life
 - undertake large cohort studies in women where body weight and waist circumference are measured rather than self-reported

Evidence Table - Section 1

Does obesity increase the risk of Type 2 diabetes?

| Author | Evidence | | | | |
|---|-------------------|------------|----------------|-------------------|------------------|
| | Level of Evidence | | Quality Rating | Magnitude Rating | Relevance Rating |
| | Level | Study Type | | | |
| Carey VJ (1997) (Adult women – US) | III-2 | Cohort | Medium | High ⁺ | High |
| Chan JM (1994) (Adult men - US) | III-2 | Cohort | Medium | High ⁺ | High |
| Chou P (1998) (Adult men – Kinmen, Tiawan) | III-2 | Cohort | High | High ⁺ | Medium |
| Colditz GA (1990) (Adult women - US) | III-2 | Cohort | Medium | High ⁺ | High |
| Crowther NJ (1998) (Children - Sth Africa: Black Sth African) | III-2 | Cohort | Medium | High ⁺ | Low |
| Everhart JE (1992) (Adults – US: Pima Indian) | III-2 | Cohort | High | High ⁺ | Low |
| Everson SA (1998) (Adult men – Finland) | III-2 | Cohort | Medium | High ⁺ | High |
| Hanson RL (1995) (Adults – US: Pima Indian) | III-2 | Cohort | Medium | High ⁺ | Low |
| Holbrook TL (1989) (Adults - US) | III-2 | Cohort | Medium | High ⁺ | High |
| Hulman S (1998) (Children – US: African American) | III-2 | Cohort | Medium | Low | Low |
| Kahn SE (1996) (Adult men – US: Japanese) | III-2 | Cohort | Medium | High ⁺ | Low |
| Liese AD (1997) (Adults – US) | III-2 | Cohort | Medium | High ⁺ | High |
| Long SD (1994) (Adults - US) | III-2 | Cohort | Medium | High ⁺ | High |
| McCance DR (1994) (Adults & Children – US: Pima Indian) | III-2 | Cohort | Medium | High ⁺ | Low |
| Motala AA (1997) (Adults – South Africa: Indian) | III-2 | Cohort | Medium | High ⁺ | Medium |
| NIDDK (2001) (Adults – US) | II | RCT | High | High ⁺ | High |
| Njolstad I (1998) (Adults – Norway) | III-2 | Cohort | Medium | High ⁺ | High |
| Perry IJ (1995) - (Adult men - UK) | III-2 | Cohort | Medium | High ⁺ | High |
| Pories WJ (1995) (Adults & adolescents – US) | III-2 | Cohort | Medium | High ⁺ | High |
| Qiao Q (1996) (Adults - Finland) | III-2 | Cohort | Medium | High ⁺ | High |
| Sakurai Y (1997) (Adult men – Japan) | III-2 | Cohort | High | High ⁺ | Low |
| Sakurai Y (1999) (Adult men – Japan) | III-2 | Cohort | High | High ⁺ | Low |
| Shaper AG (1997) (Adult men – UK) | III-2 | Cohort | Medium | High ⁺ | High |
| Shaten BJ (1993) (Adult men – US) | III-2 | Cohort | High | High ⁺ | High |
| Sinaiko AR (1999) (Children – US) | III-2 | Cohort | Medium | High ⁺ | High |
| Srinivasan SR (1996) (Adolescents – US) | III-2 | Cohort | High | High ⁺ | High |
| Tumilehto J (2001) (Adults – Finland) | II | RCT | High | High ⁺ | High |
| Vanderpump MPJ (1996) (Adults – UK) | III-2 | Cohort | Medium | High ⁺ | High |

⁺ Indicates that obesity is associated with Type 2 diabetes: **Magnitude rating:** The direction of the effect is by '+' for a positive effect and '-' for a negative effect. Low = no statistically significant effect

Section 2: Fat distribution, total adiposity and ethnicity

Issue

Is the pattern of fat distribution an additional contributor to total adiposity in determining the risk of Type 2 diabetes and how does this vary with ethnicity?

Recommendation

Abdominal obesity is an important indicator of increased risk of Type 2 diabetes in all ethnic groups and should be a particular focus of weight loss programs.

Evidence Statements

- In men, waist circumference is a better predictor of Type 2 diabetes than waist-hip ratio particularly when used with body mass index
Evidence Level III-2
- In women, android obesity (as indicated by the waist:hip ratio or waist circumference) increases the risk of Type 2 diabetes and there is less agreement in women than in men whether BMI contributes more to risk than WHR or waist circumference
Evidence Level III-2
- Ethnic groups differ in their patterns of body fat distribution
Evidence Level III-2
- In some ethnic groups total adiposity (as indicated by BMI) is more strongly related to risk of Type 2 diabetes, while in other ethnic groups android obesity is more strongly related to risk
Evidence Level III-2

Background – Fat Distribution

Obesity in adult life is an important independent risk factor for the development of Type 2 diabetes (Ferrannini and Camastra, 1998). Anthropometry is the term for techniques used to measure the size, weight and proportions of the human body. Generalised obesity can be estimated anthropometrically using the Body Mass Index (BMI) (weight in kg divided by height in metres squared). A high BMI has been shown in many studies to be a strong determinant for the development of Type 2 diabetes both in men (Feskens and Kromhout, 1989; McPhillips et al, 1990; Haffner et al, 1990; Cassano et al, 1992; Shaten et al, 1993; Chan et al, 1994; Perry et al, 1995), and in women (Lundgren et al, 1989; McPhillips et al, 1990; Haffner et al, 1990; Kaye et al, 1991; Carey et al, 1997). However, in addition to total adiposity, the pattern of body fat distribution also appears to play an important part in determining diabetes risk.

In premenopausal women, excess body fat is deposited on the hips and thighs in what is known as a *gynoid* pattern of fat distribution (Kissebah and Krakower, 1994). In relation to health risks, this pattern of fat distribution appears relatively benign (Terry et al, 1991; Williams et al, 1997). It is of interest therefore, that women who develop Type 2 diabetes retain significantly less fat on the hips and thighs than women of the same age and total adiposity who have not developed diabetes (Stoney, 1998). Seidell and co-workers (1997), similarly found in a cross-sectional study that Type 2 diabetes was most prevalent in men and women with larger waist and smaller hip circumferences.

In men and in many women after menopause, excess body fat is deposited not on the hips and thighs, but in the abdominal region. This is known as an *android* or centralised pattern of fat distribution (Kissebah and Krakower, 1994; Kissebah, 1996). Women who develop Type 2 diabetes are characterised by their accumulation of abdominal fat (Hartz et al, 1983). As detailed below, an android pattern of body fat distribution has been implicated with increased risk of Type 2 diabetes both in men and in women.

Abdominal fat can be deposited both subcutaneously as *abdominal subcutaneous fat* and internally around the body organs as *visceral fat*. The amount of abdominal subcutaneous fat and the amount of visceral fat can now be measured by imaging techniques such as Computerised Tomography (CT) and Magnetic Resonance Imaging (MRI) (van der Kooy and Seidell, 1993). Visceral fat comprises about 10% of total fat mass whereas abdominal subcutaneous fat comprises about 20% of total fat mass (Abate et al, 1995). Interest has focussed on the important role that visceral fat plays in the metabolic consequences of obesity because of its venous drainage into the portal system and directly into the liver where excess non-esterified fatty acids and glycerol may adversely affect liver metabolism (Bouchard et al, 1993; Kissebah and Krakower, 1994). Loss of visceral fat leads to reductions in the risk of cardiovascular disease and also improves metabolic control in Type 2 diabetes (Kissebah and Krakower, 1994). Abdominal subcutaneous fat is less lipolytically active than visceral fat (Rebuffé-Scrive, 1989), but evidence is accumulating that subcutaneous abdominal fat may be an important contributor to insulin resistance (Abate et al, 1995; Abate et al, 1996; Goodpaster et al, 1997).

The waist: hip ratio (WHR) is the most commonly used simple anthropometric measure to indicate android obesity (van der Kooy and Seidell, 1993). A high WHR (WHR > 1.0 in men and > 0.85 in women) is now accepted as a method of identifying people with android obesity

(WHO, 1998). Android adiposity may also be indicated by waist circumference (National Institutes of Health Expert Committee, 1998), which may be an even better predictor of diabetes risk in men and women than the WHR. Lean and co-workers (1995) have recommended that at a waist circumference of ≥ 94 cm in men and ≥ 80 cm in women, subjects should gain no further weight; at a waist circumference of ≥ 102 cm in men and ≥ 88 cm in women, subjects should reduce their weight. In Australia, 67% of men aged 45-64 have a waist circumference >90 cm and 46% of women aged 45-64 have a waist circumference greater than 80 cm (Risk Factor Prevalence Study Management Committee, 1990). More recent Australian data from the National Nutrition Survey will soon be available.

Evidence – Fat Distribution

Generalised obesity versus regional body fat accumulation and the risk of Type 2 diabetes

In European men BMI appears to be a stronger indicator for risk of Type 2 diabetes than WHR. This is evidenced in a study based on self-reported measurements made by 51,529 American men who were followed for five years (Chan et al, 1994). After adjustment for age, smoking status, family history and quintiles of WHR, men in the highest BMI quintile (BMI at or above 35 kg.m^{-2}) had a relative risk of developing diabetes of 31.7 (CI 16.2-62.0) compared to men in the lowest quintile with a BMI less than 23 kg.m^{-2} (Chan et al, 1994). However, after adjustment for age, family history, smoking status and BMI, the risk of diabetes in men from the highest quintile of WHR (above 0.99) versus men in the lowest quintile of WHR (< 0.90) was only 1.7 (CI 1.1-2.7). An earlier, much smaller study on a cohort of 1,972 American men, came to the opposite conclusion that android adiposity might have a higher effect on diabetes risk than BMI (Cassano et al, 1992).

There is also evidence to suggest that WHR may not indicate diabetes risk as clearly as waist circumference. After adjustment for age, family history of diabetes, smoking and BMI, the relative risk of Type 2 diabetes for the highest quintile of waist circumference (102-175 cm) versus the lowest quintile (75-86 cm) was 3.5 (CI 1.7-7.0). After similar adjustment, the relative risk of Type 2 diabetes for the highest quintile of WHR (WHR > 0.99) versus the lowest quintile (WHR < 0.90) was only 1.7 (CI 1.1-2.7) (Chan et al, 1994).

In women, as in men, android obesity appears to be a strong marker for insulin resistance. In a study of 22 Australian women, the level of total and central obesity was measured both by anthropometry and by Dual Energy X-ray Absorptiometry (DXA) while insulin sensitivity was determined by euglycemic-hyperinsulinemic clamp (Carey et al, 1996). Insulin sensitivity was found to be significantly and inversely related to BMI ($r = 0.48$) but a similar level of relationship was also seen with waist circumference and the WHR ($r = -0.43$ and $r = -0.46$, respectively). Rissanen and coworkers (1997) also found in Finnish women that both the WHR and the waist circumference had a similar relationship to the metabolic risk profile.

Android obesity in women has also been shown to be associated with increased risk of Type 2 diabetes. This was indicated by anthropometry in the Nurses' Health Study where 43,582 US women who self-reported weight, waist and hip measurements, were followed from 1986 to 1994 (Carey et al, 1997). Controlling for BMI and other cofounders, the relative risk of Type 2 diabetes for the 90th percentile of WHR (WHR = 0.86) versus the 10th percentile (WHR = 0.70) was 3.1 (CI 2.3-4.1). Similarly, the relative risk of diabetes for the 90th percentile of waist circumference (92 cm) versus the 10th percentile of waist circumference (67 cm) was

5.1 (CI 2.9-8.9). The waist circumference in these American women was therefore a stronger indicator of risk than the WHR.

In the Nurses Health Study, it was also found that BMI was a stronger predictor of diabetes risk than either waist circumference or WHR. The relative risk of Type 2 diabetes for the 90th percentile of BMI (29.9) versus the 10th percentile (20.1) was 11.2 (CI 7.9-15.9) (Carey et al, 1997). This finding contrasts with that of an earlier study on women from Gothenberg, Sweden (Lundgren et al, 1989). Here the incidence of diabetes was found to increase 8 times between the highest and lowest quintile for BMI, while the incidence of diabetes increased 13.6 times between the highest and lowest quintile of WHR. Another study in Mexican Americans enrolled in the San Antonio Heart Study also found a slightly greater effect of android adiposity (measured as the centrality index or the ratio of subscapular to triceps skinfold) than total adiposity (measured by BMI) on diabetes risk in women (Haffner et al, 1991). The OR for the highest ($> 28.2 \text{ kg.m}^{-2}$) versus the lowest ($< 24.6 \text{ kg.m}^{-2}$) tertile of BMI was 2.25 (CI 0.62-8.11), while the OR for the centrality index was 3.28 (CI 0.41-26.). In a later study, where the WHR was used to indicate android obesity, it was again confirmed that android obesity had a greater effect on diabetes risk in Mexican American women than in Mexican American men (Haffner et al, 1992).

Influence of ethnicity

Android fat distribution has been implicated as a risk factor for the development of Type 2 diabetes in a number of different ethnic groups. These include Europeans (Ohlson et al, 1988); Mexican Americans (Haffner et al, 1990; Haffner et al, 1991; Wei et al, 1997); Asian Indians, Creoles and Chinese from Mauritius (Dowse et al, 1991) and Chinese living in the UK (Unwin et al, 1997).

Some ethnic groups appear to be more prone to deposit body fat in an android pattern than Europeans. For example, Indians from Mauritius have a greater amount of abdominal fat (as indicated by the WHR) for any given level of BMI than the Creole or Chinese Mauritians (Hodge et al, 1996). Another study comparing European and Pakistani men living in the UK, found that although the men had similar BMI ($25\text{-}26 \text{ kg.m}^{-2}$), the two ethnic groups had distinctly different patterns of body fat deposition (Bose, 1996). By anthropometry, the European men had more fat accumulated over both the upper and lower body, whereas in the Pakistani men, fat deposition was more concentrated on the abdomen. Asian Indian men from the United States have similarly been reported to have relatively high levels of abdominal fat (Banerji et al, 1999). Anthropometric measures moreover, may not always reveal differences in visceral fat between ethnic groups. Body fat distribution was compared by CT in 23 Afro-American and 15 European women who had similar mean BMI and WHR (Lovejoy et al, 1996). Despite the close similarity by anthropometry, African-American women by CT were found to have a smaller area of visceral fat.

It is evident that ethnicity can influence relationships between total adiposity, body fat distribution and diabetes risk. In Japanese men the WHR may have a greater influence on risk than the BMI. A cohort study of 2,228 men from the Japanese military indicated that after adjusting for WHR, the OR for Type 2 diabetes in the highest versus the lowest quintile of BMI was 1.2 (CI 0.6-2.5). In contrast, after adjustment for WHR, the OR for the highest versus the lowest quintile of WHR was 3.5 (CI 1.5-2.9) (Sakurai et al, 1995). In this study, the mean BMI of men studied was within the normal range (mean \pm SD, 23.8 ± 2.6) while the mean WHR was relatively high (0.902 ± 0.049).

In 721 Mexican Americans followed for 7.2 years, a comparison was made between BMI, waist circumference and the WHR as predictors of Type 2 diabetes (Wei et al, 1997). In this population, waist circumference was clearly found to be the best predictor of diabetes risk, the risk of diabetes being 11 times greater in those in the highest quartile for waist circumference than those in the lowest quartile (CI 4.2-28.8). In addition, by multivariate analysis, the waist circumference was the only significant predictor of Type 2 diabetes in this population, in models where WHR and BMI were also entered either separately or simultaneously. A cross-sectional study has reported that an increased WHR was associated with higher risk in non-Hispanic Whites than in Hispanics (Marshall et al, 1993). Another study compared the effect of WHR on risk of Type 2 diabetes in Asian Indians from Madras, and Mexican Americans and non-Hispanic whites recruited into the San Antonio Heart Study (Ramachandran et al, 1997). Here it was found that although the non-Hispanic whites had a WHR significantly above the other groups ($p < 0.05$), WHR was only predictive of Type 2 diabetes in the Asian Indians and the Mexican Americans.

A cross-sectional comparison has also been made of the effects of both WHR and waist circumference on risk of Type 2 diabetes in African-Americans and Jamaicans (Okosun et al, 1998). In Jamaican men ($n = 510$), a waist circumference equal or greater than 94 cm was associated with an age-adjusted OR of 2.09 (CI 1.38-3.16), whereas in African-American men ($n = 844$) this waist circumference was associated with an age-adjusted OR of 3.90 (CI 2.67-5.72). In Jamaican women ($n = 776$), a waist circumference equal or greater than 80 cm was associated with an age-adjusted OR of 3.38 (CI 1.84-6.21), whereas in African-American women ($n = 983$) this waist circumference was associated with an age-adjusted OR of 1.63 (CI 0.94-2.81).

The evidence indicates that different ethnic groups differ in the level of risk associated with waist circumference, WHR or BMI. At present, these measures provide a practical method of identifying Europeans with android obesity but grading systems developed for the European population must be applied with caution to other ethnic groups.

Summary – Fat Distribution

- Waist circumference in European men is a better predictor of Type 2 diabetes than waist-hip ratio particularly when used with Body Mass Index
- While there is strong evidence in European women that android obesity increases the risk of Type 2 diabetes, there is less agreement on whether the risk associated with android obesity in women is greater than that associated with total adiposity
- There is strong evidence that different ethnic groups differ in whether total adiposity (as indicated by BMI) or android adiposity (as indicated by WHR or waist circumference) is the stronger indicator of diabetes risk. There is a need for further research to establish cut-off values for waist circumference or WHR relative to diabetes risk in different ethnic groups

Evidence Table - Section 2

Is the pattern of fat distribution of more importance than total adiposity in determining the risk of Type 2 diabetes and does this vary with ethnicity?

| Author | Evidence | | | | |
|---|-------------------|---------------------------------|----------------|--------------------------|------------------|
| | Level of Evidence | | Quality Rating | Magnitude Rating | Relevance Rating |
| | Level | Study Type | | | |
| Banerji MA (1999) (Adult men – US: Asian Indian) | III-2 | Cross-sectional | Medium | High ^{A+} | Medium |
| Bose K (1996) (Adult men – UK: Caucasian & Pakistani) | III-2 | Case-control | Medium | High ^{A+; E+} | Medium |
| Carey DG (1996) (Adult women – Australia) | III-2 | Cross-sectional | Medium | High ^{A+} | High |
| Carey VJ (1997) (Adult women – US) | III-2 | Cohort | Low | High ^{A+} | High |
| Cassano PA (1992) (Adult men – US) | III-2 | Cohort | Medium | High ^{A+} | High |
| Chan JM (1994) (Adult men – US) | III-2 | Cohort | Medium | High ^{A+} | High |
| Dowse GK (1991) (Adults – Mauritius) | III-2 | Cross-sectional | Medium | High ^{A+; E+} | Low |
| Haffner SM (1990) (Adults – US: Mexican American) | III-2 | Cohort | High | High ^{A+} | Low |
| Haffner SM (1991) (Adults – US: Mexican American) | III-2 | Cohort | High | High ^{A+} | Low |
| Haffner SM (1992) (Adults – US: Mexican American) | III-2 | Cross-sectional | High | High ^{A+} | Low |
| Hodge AM (1996) (Adults – Mauritius) | III-2 | Cohort | High | Medium ^{A+; E+} | Low |
| Lean ME (1995) (Adults – Scotland, UK) | III-2 | Cross-sectional | Medium | High ^{A+} | High |
| Lovejoy JC (1996) (Adult women – US) | III-2 | Case-control | Medium | High ^{A+; E+} | High |
| Lundgren H (1989) (Adult women – Sweden) | III-2 | Cohort | Medium | High ^{A+} | High |
| Marshall JA (1993) (Adults – US) | III-2 | Case-control | Medium | High ^{A+; E+} | High |
| Ohlson LO (1988) (Adult men - Sweden) | III-2 | Cohort | Medium | High ^{A+} | High |
| Okosun IS (1998) (Adults – Jamaica; US; Nigeria) | III-2 | Cross-sectional | High | High ^{A+; E+} | Low |
| Ramachandran A (1997) (Adults – US; India) | III-2 | Cross-sectional | Medium | High ^{A+; E+} | Medium |
| Rissanen P (1997) (Adult women – Finland) | III-2 | Cross-sectional | High | High ^{A+} | High |
| Sakurai Y (1995) (Adult men – Japan) | III-2 | Cross-sectional | High | High ^{A+} | Low |
| Shin CS (1997) (Adults – Korea) | III-2 | Cohort with nested Case-control | High | Low | Low |
| Unwin N (1997) (Adults – UK: Chinese, Euroid) | III-2 | Cross-sectional | High | High ^{A+; E+} | Medium |
| Wei M (1997) (Adults – US: Mexican American) | III-2 | Cohort | High | High ^{A+} | Low |

⁺ Indicates that ethnicity or adiposity is associated with increased risk of Type 2 diabetes; ^A Adiposity; ^E Ethnicity.

Magnitude rating: The direction of the effect is by '+' for a positive effect and '-' for a negative effect. Low = no statistically significant effect

Section 3: Simple measures for body fat distribution

Issue

What simple measures can be used to determine body fat distribution?

Recommendations

Waist circumference (>100 cm for European men and >90 cm for European women) should be used in addition to body weight and body mass index (BMI) to identify individuals who should seek and be offered weight management programs.

Evidence Statements

- The waist circumference has major advantages over the WHR in its simplicity of measurement and its relation to body weight and fat distribution
Evidence Level II[#]
- Threshold values for waist circumference may be lower in older adults
Evidence Level III[#]
- Threshold values for waist circumference are population specific
Evidence Level III[#]

[#]Studies assessed using the non-intervention assessment

Background – Measurement of Fat Distribution

Simple anthropometric measures can be used to identify individuals at increased risk of Type 2 diabetes due to abdominal fat accumulation. Waist circumference and the waist to hip circumference ratio (WHR) are commonly used anthropometric measures for this purpose (Han et al, 1995; Carey et al, 1997; Ledoux et al, 1997; Han et al, 1998). Over the last decade, a high WHR (WHR > 1.0 in men and > 0.85 in women) has become accepted as the clinical method for identifying patients with abdominal fat accumulation (James, 1996). More recent evidence, however, suggests that waist circumference alone - measured at the midpoint between the lower border of the rib cage and the iliac crest - may provide a better correlate of abdominal adiposity and diabetes risk (Lean et al, 1995; Seidell, 1995; James, 1996).

These thresholds for waist circumference may be lower in European men and women over the age of 40 years. These threshold values for waist circumference are not directly applicable to people from other ethnic backgrounds although the same principles apply.

Evidence – Measurement of Fat Distribution

Advantages of using waist circumference to determine body fat distribution

The advantage of using waist circumference as an indicator of impaired health and diabetes risk was demonstrated by Lean and co-workers in a cross-sectional study of 5,887 men and 7,018 women aged 20-59 years recruited from the general population of two cities in the Netherlands (Lean et al, 1998). After adjustment for age and lifestyle, risk of Type 2 diabetes was found to be increased in men who had a waist circumference greater than 102 cm [OR 4.5 (CI 2.5-7.8)]. The risk of Type 2 diabetes was similarly found to be increased in women with a waist circumference greater than 88 cm [OR 3.8 (CI 1.9-7.3)].

Arguments advanced in favour of the waist circumference include its simplicity of measurement and its relation to body weight and fat distribution (Lean et al, 1995). In comparison with the WHR, the waist circumference appears to be a better indicator of adiposity in women. In a study of 96 women aged 16-80 years, body fat reference measures were obtained by Dual Energy X-ray Absorptiometry (DXA) (Taylor et al, 1998). In comparing the results of anthropometric measures with DXA, a positive reference test was defined as a result at or above the 75th percentile for all DXA measurements. WHR (75th percentile, 0.81) categorises only 58% of subjects, misclassifying 28 whereas the waist circumference percentile, 86.9 cm accurately classified 83% of subjects, misclassifying only eight ($p < 0.05$). In another study of 24 older men with cardiac disease where estimation of body fat by circumferences was compared with hydrostatic weighing, use of circumferences was found to be accurate, easy to administer and had low potential for error (Young et al, 1998).

Threshold values of waist circumference to identify those at risk of Type 2 diabetes

Lemieux and co-workers examined the relationship of anthropometric variables to visceral adipose tissue accumulation (by computed tomography (CT)) in 213 men and 190 women and determined how these relationships were affected by sex, age, menopausal status, and the degree of obesity (Lemieux et al, 1996). The threshold for visceral adipose tissue accumulation in this study was taken as 130 cm², since above this level, in both men and

women, substantial alterations in plasma glucose-insulin and lipoprotein profiles are observed (Despres and Lamarche, 1993).

The relationship of anthropometric variables to visceral adipose tissue accumulation also appeared age-specific. In both sexes, waist circumferences corresponding to the critical threshold of visceral adipose tissue were lower in older subjects (≥ 40 years old) than in younger individuals. Thus in men older than 40 years, a waist circumference of 90.9 cm corresponded to a visceral adipose tissue area by CT of 130 cm^2 , whereas in men aged less than 40 years, a waist circumference of 98.9 cm corresponded to a visceral adipose tissue area of 130 cm^2 . Similar age-specific differences were also seen in women (Lemieux et al, 1996). Another similar smaller study in 71 Dutch men and 34 Dutch women has also found that intra-abdominal fat area by CT was higher in older subjects for a given waist circumference (Han et al, 1997).

In the study by Lemieux and co-workers, it was found from computed regression equations, that a waist circumference of 95 cm in men or women, or a WHR of 0.94 in men and of 0.88 in women corresponded to a visceral adipose tissue area of 130 cm^2 (Lemieux et al, 1996). In this study, threshold values of waist circumference were similar in normal and overweight men and women, whereas threshold values of WHR were higher in normal than in overweight subjects (Lemieux et al, 1996). The waist circumference was argued to be a stronger and more convenient anthropometric correlate of visceral adipose tissue than the WHR since threshold values of the waist circumference appeared to be independent of the degree of obesity (Lemieux et al, 1996).

The effect of change in visceral adipose tissue on waist circumference has also been examined. In a study in 842 Swedes aged 37-60 years, it was found that over a 2-year period, for a given change in BMI, the change in visceral adipose tissue was 6 times greater in men than it was in women (Sjostrom et al, 1997). Despite this difference, the change in both the waist circumference and the WHR was similar in both sexes. The low change in visceral adipose tissue in women found in this study may have reflected the relatively high proportion who were pre-menopausal and had gynoid rather than android obesity.

A community derived random sample of men and women was studied in North Glasgow, UK, to test the hypothesis that waist circumference might be used as a single measurement to identify overweight people with android obesity (Lean et al, 1995). A group of 1,014 women and 904 men, aged 25-74 years, was recruited from the general population of North Glasgow. From this population thresholds were determined for waist circumference that identified subjects with high BMI or with high WHR. These threshold levels were then validated on a separately recruited group of 86 men and 202 women. These people were slightly younger than the first group, but exhibited a similar range of anthropometric measurements. Results indicated that a waist circumference ≥ 94 cm for men and ≥ 80 cm for women identified both subjects with high BMI ($\geq 25 \text{ kg.m}^{-2}$) and subjects with lower BMI but a high WHR (≥ 0.95 for men, ≥ 0.80 women) with a sensitivity of 96% and specificity 97.5%. A waist circumference ≥ 102 cm for men or ≥ 88 cm for women identified subjects with BMI ≥ 30 and those with lower BMI but high WHR with a sensitivity of 96% and specificity 98%. In this case only 2% of the sample was misclassified. From these data, Lean and co-workers recommended the following cut-off values: at a waist circumference of ≥ 94 cm in men and ≥ 80 cm in women, subjects should gain no further weight; at a waist circumference of ≥ 102 cm in men and ≥ 88 cm in women, subjects should reduce their weight. The waist circumference could therefore be used to identify individuals who should seek and be offered

weight management programs. These threshold values identifying men at increased risk are very similar to those obtained by Lemieux and coworkers (Lemieux et al, 1996), however, the values for women were considerably lower. This may be due to the fact that the relationship of the waist circumference to visceral adipose tissue is influenced by age (Lemieux et al, 1996).

A study was carried out to determine whether waist circumference could accurately identify android obesity in 1,513 Hong Kong Chinese men and women (Ko et al, 1996). It was found that a waist circumference ≥ 94 cm for men and ≥ 80 cm for women identified subjects with high BMI ($\geq 25 \text{ kg.m}^{-2}$), and those with lower BMI but high WHR (≥ 0.95 for men, ≥ 0.80 for women) with a sensitivity of 31% and specificity of 100%. Decreasing the waist circumference cut-off to 85 cm for men and 75 cm for women increased the sensitivity to 79.2% in men and 56.4% in women. It was concluded that a single waist circumference measurement did not allow sensitive identification of Chinese people at health risk from being overweight or from having central obesity (Ko et al, 1996). This study indicates threshold values for waist circumference may be population specific.

Summary – Measurement of Fat Distribution

- There is moderate to strong evidence, from studies applicable to Australia, that body mass index (BMI), waist to hip circumference ratio (WHR), and waist circumference are powerful independent predictors of Type 2 diabetes
- There is moderate evidence to indicate that the simplicity of measurement and its relation to body weight and fat distribution are major advantages for waist circumference over BMI and WHR
- There is strong evidence that waist circumference identifies normal-weight and overweight subjects with excess visceral adipose tissue which increases their risk of Type 2 diabetes
- There is strong evidence to suggest that in both men and women, threshold values of waist circumference corresponding to critical amounts of visceral adipose tissue are lower in those over 40 years than in younger individuals
- It has been suggested on the basis of experimental studies that in European populations, men with a waist circumference ≥ 94 cm and women with a waist circumference ≥ 80 cm should gain no further weight. In addition, men with a waist circumference ≥ 102 cm and women with a waist circumference ≥ 88 cm should reduce their weight
- These values could be changed to the closest mark on the tape measure to increase the ease of recording them for the health professional. The measurements that indicate the need to maintain weight in the European populations would be ≥ 95 cm for men and ≥ 80 cm for women and to reduce weight at ≥ 100 cm for men and ≥ 90 cm for women
- There is moderately strong evidence to suggest that threshold values for the waist circumference are population specific

Evidence Table – Section 3

What measures can be used to determine body fat distribution?

| Author | Evidence | | | | |
|---|-------------------|-----------------|----------------|------------------------------|------------------|
| | Level of Evidence | | Quality Rating | Magnitude Rating | Relevance Rating |
| | Level | Study Type | | | |
| Carey VJ (1997) (Adult women – US) | II [#] | Cohort | Low | High ^{B+; W+} | High |
| Chan JM (1994) (Adult men - US) | II [#] | Cohort | Low | High ^{B+; W+} | High |
| Han TS (1997) (Adult women – Scotland, UK; The Netherlands) | III [#] | Cross-sectional | Low | High ^{W+} | High |
| Ko GTC (1996) (Adults - Hong Kong) | III [#] | Cross-sectional | Medium | High ^{W+} | Medium |
| Lean ME (1995) (Adults – Scotland, UK) | III [#] | Cross-sectional | Medium | High ^{W+} | High |
| Lean MEJ (1998) (Adults - The Netherlands) | III [#] | Cross-sectional | Medium | High ^{W+} | High |
| Ledoux M (1997) (Adults - Canada) | III [#] | Cross-sectional | Medium | High ^{W+; B+; WHR+} | High |
| Lemieux S (1996) (Adults - Canada) | III [#] | Cross-sectional | Medium | High ^{W+} | High |
| Sjostrom CD (1997) (Adults - Sweden) | II [#] | Cohort | Medium | High ^{C+} | High |
| Taylor RW (1998) (Adults – New Zealand) | III [#] | Cross-sectional | Medium | High ^{W+; B+} | High |
| Young H (1998) (Adult men - US) | III [#] | Cross-sectional | Medium | Medium ^{W+} | High |

[#] Studies assessed using the non-intervention assessment

⁺ The measure of body fat distribution is associated with an increased risk of Type 2 diabetes; ^B BMI; ^W Waist Circumference; ^{WHR} Waist – hip ratio; ^C Computed tomography.

Magnitude rating: The direction of the effect is by ‘+’ for a positive effect and ‘-’ for a negative effect.

Section 4: Physical activity and sedentary lifestyle

Issue

What is the evidence that physical activity can decrease, and sedentary behaviour increase, the risk of Type 2 diabetes?

Recommendation

Regular physical activity is recommended to reduce the risk of Type 2 diabetes.

Evidence Statements

- In both men and women, physical activity reduces the risk of Type 2 diabetes
Evidence Level III-2
- In adults, the risk of Type 2 diabetes declines as frequency of exercise increases to 3-5 times per week
Evidence Level III-2
- In men, the risk of Type 2 diabetes declines with increased energy expended
Evidence Level III-2
- Men with low cardiovascular fitness have greater risk of IGT and Type 2 diabetes than men with high cardiovascular fitness
Evidence Level III-2
- Physical activity reduces the risk of Type 2 diabetes in older men just as well as it does in younger men
Evidence Level III-2
- Physical activity reduces the risk of Type 2 diabetes more in overweight and obese men than it does in non-obese men
Evidence Level III-2
- Exercise programs can slow the progression from IGT to Type 2 diabetes
Evidence Level II
- Exercise can reduce diabetes-related mortality
Evidence Level II

Background – Physical Activity

As societies become more sedentary with westernisation, the prevalence of Type 2 diabetes increases (Collins et al, 1994). Accordingly, as reviewed below, individuals who maintain a physically active lifestyle appear to be at less risk of developing IGT and Type 2 diabetes (Ivy, 1997).

The importance of physical activity as part of a lifestyle that protects against diabetes has only been recognised relatively recently. In 1983, Horton suggested that preventive measures to reduce the prevalence of Type 2 diabetes should not only focus on eliminating or reducing factors such as obesity that were associated with the development of insulin resistance, but should also encourage physical activity that enhanced insulin sensitivity (Horton, 1983). Since that time, increasing attention has been given to the importance of physical activity in diabetes prevention (Hardman, 1996; Ivy, 1997).

Greater levels of physical activity have been found to be inversely related to the presence of Type 2 diabetes in 14 of 16 cross-sectional studies (James et al, 1998). Moreover, findings in cross-sectional studies appear to be consistent across different ethnic groups, including Fijian Indians (Taylor et al, 1984), Micronesians (King et al, 1984), Polynesians (Taylor et al, 1983), Mauritians (Dowse et al, 1991) and Swedes (Cederholm and Wibell, 1985). Prospective cohort studies as outlined below, have now greatly strengthened these findings.

Evidence – Physical Activity

Physical activity and the risk of Type 2 diabetes in men

Increased physical activity reduces the risk of Type 2 diabetes in men. In the Physicians' Health Study, where 21,271 American men were followed for five years, men who reported exercising at least once a week had an age-adjusted risk of Type 2 diabetes of 0.64 (CI 0.51-0.82, $p < 0.003$) relative to men who exercised infrequently (Manson et al, 1992). This association was little attenuated by adjustment for BMI, indicating that physical activity is an independent risk factor for diabetes. A multicentre study of 7,735 British men has also shown that increased physical activity reduces diabetes risk. In this study, after adjustment for age and BMI, the risk of diabetes for men who reported that they engaged in moderate levels of physical activity was reduced to 0.4 (CI 0.2-0.7) relative to men who were inactive (Perry et al, 1995).

While most studies that examine the relationship between diabetes risk and physical activity have relied on self-reported exercise patterns, one recent study has examined the relationship between measured cardiovascular fitness and diabetes risk (Wei et al, 1999). In this study, which was carried out in 8,633 American men, cardiovascular fitness was estimated by a maximal exercise test on a treadmill. After a follow-up of 6 years, it was found that men who were least fit at baseline (20% of the cohort) had a 3.7 times increased risk of Type 2 diabetes (CI 2.4-5.8) compared with those with greatest cardiovascular fitness at baseline (40% of the cohort).

The protective effect of physical activity appears to be most pronounced in the heaviest men. In a study of male alumni it was demonstrated that each 2-unit increase in BMI was associated with a 21% increase in the risk of Type 2 diabetes (Helmrich et al, 1991). A greater

protective effect of exercise in overweight or obese men was also reported in the Physicians' Health Study (Manson et al, 1992).

The age at which men are exercising does not appear to affect the protective influence of increased physical activity. In the Honolulu Heart Study, the effect of physical activity on diabetes risk was found to be similar in both middle-aged and older men. Thus for men aged 45-54 at baseline the OR was 0.52 (CI 0.32-0.85) while for men aged 55-68 at baseline the OR was 0.45 (CI 0.25-0.80) (Burchfiel et al, 1995).

Physical activity and the risk of Type 2 diabetes in women

The association between physical activity and the risk of diabetes has been much less frequently studied in women than in men. In the Nurses Health Study however, 87,253 American women were followed for 8 years. It was found that women who reported that they engaged in vigorous activity once or more times per week had a reduced age-adjusted relative risk of diabetes of 0.67 (CI 0.60-0.75) compared with women who did not exercise (Manson et al, 1991).

The effect of physical activity on diabetes risk is consistent across different population groups

The effect of physical activity on diabetes risk is also evident from cohort studies in different ethnic groups. Protective effects were evident in Japanese American men enrolled in the Honolulu Heart program (Burchfiel et al, 1995), in African-American men and women participating in the Pitt County Study (James et al, 1998) and in Mexican American men (Monterrosa et al, 1995). A case-control study in Japanese military men (men with IGT, n=38; men with normal glucose tolerance, n=60) has also indicated the protective value of a high level of fitness at age 30-39 on the risk of impaired glucose tolerance 20 years later (Takemura et al, 1999). In this study, physical fitness was indicated by the shortest time in which the men had run 1,500 m when they were aged 30-39 years. The reduced risk of impaired glucose tolerance with high physical fitness at this age was 0.31 (CI 0.11-0.86, p<0.05).

Exercise intervention, diabetes progression and diabetes mortality

A recent randomised control trial carried out in the city of Da Qing, China has shown that an exercise intervention program can reduce the rate of conversion from IGT to Type 2 diabetes (Pan et al, 1997). In this study, men and women with IGT (n=577) were randomised to a control group and intervention groups including a group treated with exercise. After 6 years, the incidence of diabetes was 68% (CI 60-75%) in the control group but only 41% (CI 33-49%) in the exercise group (p<0.05). By a proportional hazards analysis, and adjusting for differences in baseline BMI and fasting glucose, the exercise intervention was associated with a 46% reduction (p<0.005) in the risk of developing diabetes. It should be noted that the cohort treated in this Chinese study was both relatively lean and physically active. The data may not therefore be directly extrapolated to people with IGT in western societies on whom further research of this nature is needed.

Increased physical activity may reduce diabetes-related mortality. In the Malmö Preventative Trial, it was found after 12 years follow-up that men with IGT (n=288) who participated in a

diet and exercise program, exhibited similar mortality to men (n=6,389) with normal glucose tolerance (6.5 vs 6.2 deaths /1,000 person years, respectively). In marked contrast, 135 men with IGT who received only conventional treatment had 14.0 deaths/1,000 person years, p=0.009 (Eriksson and Lindgarde, 1998).

The recently published Diabetes Prevention Study (Tuomilehto et al, 2001) conducted in Finland with a 3.2 year follow-up showed that individual diet and exercise intervention conducted by a dietitian could reduce the risk of diabetes by 58% (p<0.001) in a high risk population. This was achieved with 7 sessions in the first year and regular 3 monthly sessions after that during which goals were set on how to lose weight (>5%), decrease total fat intake to <30% of energy, decrease saturated fat intake to <10% of energy, increase fibre intake to >15 g/1000 kcal consumed and to exercise for at least 30 min per day. The groups were individually given guidance to increase their exercise levels (30 min per day) with a combination of endurance exercise and supervised resistance training. The control group were set goals and given general written and oral information about diet and exercise initially and then annually but no specific individualized programs were offered. The incidence of diabetes in the intervention group was significantly lower than the control group (11% vs 23%). Diabetes did not develop in any of the groups who achieved 4 or 5 goals, however significantly more people were able to achieve these goals in the intervention group (49 in the intervention group vs 15 in the control group, p< 0.001 for each of the goals). In those who did not achieve any of the goals the incidence of diabetes was 38% vs 31% respectively (Tuomilehto et al, 2001).

The Diabetes Prevention Program was a major clinical trial conducted in the United States over 3 years in a group with people with IGT and therefore at high risk of developing Type 2 diabetes. The intensive lifestyle intervention included instructed on a low fat diet, exercising for 150 minutes per week and behaviour modification skills. The results show on average that this group had a 7% weight loss in the first year and sustained a 5% loss for the study's duration and maintained 30 minutes of exercise per day. A 58% reduction in the risk of developing Type 2 diabetes was found compared to the control group who received only basic diet and exercise advice. This study also included another group which was treated with Metformin which was effective in reducing the risk of developing diabetes by approximately 30% (National Institutes of Diabetes and Digestive and Kidney Diseases, 2001).

Frequency and intensity of exercise and risk of Type 2 diabetes

In men there appears to be a clear dose-response relationship between total energy expenditure in physical activity and the prevention of Type 2 diabetes. For example, in a study, which followed 5,990 American men for 14 years, with leisure-time physical activity determined by questionnaire, physical activity was measured as megajoules (MJ) of energy used. It was calculated that for every 2.1 MJ increment in weekly energy expenditure, there was a 6% decrease in diabetes risk (Helmrich et al, 1991). In agreement with this finding a study of 6,815 Japanese-American men enrolled in the Honolulu Heart program found lower risk in men who were taking the most exercise (Burchfiel et al, 1995). The age-adjusted odds ratio for the lowest versus the highest quintile of self-reported physical activity was 0.55 (CI 0.41-0.75). Similarly, it has been reported that the age adjusted risk of diabetes in American men who played vigorous sport was lower than in men who played moderate sport only; while diabetes risk was also lower in men who walked furthest each day (Helmrich et al, 1991). In addition, men who have lowest cardiovascular fitness were found to have greater risk of IGT or Type 2 diabetes than men who had moderate or high cardiovascular fitness as assessed by a

maximal exercise test on an electronic treadmill with varying speed and elevation (Wei et al, 1999). In 1,500 Finnish men, followed for a period of 10 years, the relative risk of diabetes was 1.63 (CI 0.92-2.88) in men who engaged in vigorous activity less than once a week relative to men who were vigorously active more than once a week (Haapanen et al, 1997).

Although the above studies all suggest the most vigorous activity is protective, some studies have indicated that for Type 2 diabetes prevention, there may be little additional benefit in exceeding moderate intensity activity. These studies determined a physical activity score based on the frequency and intensity of the activities reported by the subjects. In a study of British men the risk of diabetes decreased progressively as intensity of physical activity increased from light to moderate, but risk was not decreased any further in men who regularly performed vigorous physical activity (Perry et al, 1995). Similarly, the age-adjusted risk of dying or developing cardiovascular disease or diabetes was lower in men who engaged in moderate physical activity than in men who engaged in light physical activity [RR 0.53 (CI 0.44-0.64) and 0.66 (CI 0.56-0.78), respectively]. Risk was not, however, reduced any further in men who engaged in moderately vigorous or vigorous activity (Wannamethee et al, 1998).

In striking comparison to men, there is a paucity of data on the frequency and intensity of exercise in relation to risk of diabetes in women. The Nurses' Health Study reported no clear dose-response gradient in relation to frequency of exercise. Women who exercised only once a week had an age adjusted relative risk of Type 2 diabetes of 0.74 (CI 0.6-0.91) relative to sedentary women, while those who exercised four or more times a week had an age-adjusted relative risk of 0.63 (CI 0.53-0.75) (Manson et al, 1991). Another study followed 1,340 Finnish women for a period of 10 years (Haapanen et al, 1997). In this study, both a higher total amount of leisure time activity, and vigorous activity undertaken more than once a week were protective against diabetes. The study population was divided into three equal groups (each called a tertile) according to the amount of leisure time physical activity reported. The tertile of women who were least active had an age-adjusted relative risk of 2.64 (CI 1.28-5.44) relative to the tertile of women who were most active. In addition, the tertile of women who were moderately active had an age-adjusted relative risk of 1.17 (CI 0.50-2.70) relative to the tertile who were most active.

There is also evidence that exercise needs to be taken regularly for preventative effect. In the Physicians Health Study, the age-adjusted relative risk for diabetes gradually fell from 0.77 (CI 0.55-1.07) in men who exercised only once a week to 0.58 (CI 0.40-0.84) in men who exercised 5 or more times per week (p , trend =0.0002) (Manson et al, 1992).

All studies reviewed here demonstrate the risk of developing diabetes associated with some level of habitual physical activity compared to inactivity. In addition, data supports the very encouraging public health message that the greatest health benefits of increased physical activity may accrue to those who are initially most inactive (Manson et al, 1991; Perry et al, 1995; Haapanen et al, 1997; Wannamethee et al, 1998).

The majority of studies, reviewed here, rely upon self-reported estimates of physical activity. Just as the advent of doubly labelled water has shown that the obese tend to under-report food intake (Price et al, 1997), subjects may also over-report physical activity (Richman et al, 1996). If this is the case, there may be an even stronger inverse relationship between physical activity and diabetes than studies now indicate.

Obesity is known to be a primary risk factor for Type 2 diabetes (Ferrannini and Camastra, 1998) and android obesity also has an important influence on risk (Despres, 1998). As levels of physical activity are inversely correlated with obesity and can directly reduce abdominal obesity (Tremblay et al, 1990), it might be expected that physical activity would influence progression of diabetes by reduction of obesity, and specifically, reduction of abdominal fat. Adjustments for body mass index (BMI) have however, in the main, shown physical activity to be an independent risk factor. Wei and coworkers also found that the association remains after controlling for waist circumference (Wei et al, 1999).

While the evidence that increased physical activity reduces the risk of Type 2 diabetes is strong, less is known about the type of exercise that is most protective. Indeed, some forms of exercise may be contra-indicated: resistance training and high intensity exercise may be inappropriate for hypertensive individuals (Wallberg-Henriksson et al, 1998). The American College of Sports Medicine recommended that asymptomatic, apparently healthy individuals of any age who have two or more risk factors for coronary artery disease, should undergo exercise testing before participating in vigorous exercise programs (Gordon et al., 1992). Nevertheless, vigorous sports activity (expending at least 42 KJ/min) appears protective against Type 2 diabetes (Helmrich et al., 1991). Due to the greater glucose disposal that results from the use of large muscle groups, activities such as walking, swimming, or cycling appear to be preferable to exercises isolating small muscle groups such as archery, shooting, some callisthenics or some forms of weight lifting. Compliance with an exercise program is also important. Therefore flexible programs, based on accumulated lifestyle oriented activities, may be more conducive to improvements in long term exercise adherence and to improvements in metabolic health than a structured program (Jakicic et al, 1995). To this end, the recently released National Physical Activity Guidelines for Australians (Egger et al, 1999) represent an appropriate public health approach to Type 2 diabetes prevention, given that greatest attention needs to be given to those at increased risk.

Summary – Physical Activity

- There is strong evidence from well designed longitudinal studies, highly applicable to Australia, that physical activity decreases the risk of Type 2 diabetes
- There is strong evidence in men that high cardiovascular fitness protects against the development of Type 2 diabetes
- The protective effect of physical activity appears to be more pronounced in men who are obese but is as effective in older as in younger men
- There is strong evidence from a study in Chinese men and women that an exercise program will decrease the progression from IGT to Type 2 diabetes but it is not clear, however, how applicable this data will be to Australian populations
- There is strong evidence from a European study that an exercise intervention will reduce diabetes-related deaths
- There is some strong evidence that protection against Type 2 diabetes increases in a dose response relationship with physical activity but other studies indicate that moderate activity can be as beneficial as vigorous activity and further studies are required on this question, particularly in women

Evidence Table - Section 4

Does exercise decrease, and a sedentary life increase, the risk of Type 2 diabetes?

| Author | Evidence | | | | |
|---|-------------------|--------------|----------------|---------------------|------------------|
| | Level of Evidence | | Quality Rating | Magnitude Rating | Relevance Rating |
| | Level | Study Type | | | |
| Burchfiel CM (1995) (Adult men - US: Japanese) | III-2 | Cohort | High | High ⁺ | Low |
| Eriksson KF (1998) (Adult men - Sweden) | II | RCT | Medium | High ⁺ | High |
| Haapanen N (1997) (Adults - Finland) | III-2 | Cohort | High | Medium ⁺ | High |
| Helmrich SP (1991) (Adult men - US) | III-2 | Cohort | Low | High ⁺ | High |
| James SA (1998) (Adults - US: African American) | III-2 | Cohort | High | High ⁺ | Low |
| Manson JE (1991) (Adult women - US) | III-2 | Cohort | High | High ⁺ | High |
| Manson JE (1992) (Adult men - US) | III-2 | Cohort | High | High ⁺ | High |
| Monterrosa AE (1995) (Adults - US: Mexican Americans) | III-2 | Cohort | High | High ⁺ | Low |
| NIDDK (2001) (Adults - US) | II | RCT | High | High ⁺ | High |
| Pan XR (1997) (Adults - China) | II | RCT | High | High ⁺ | Medium |
| Perry IJ (1995) (Adult men - UK) | III-2 | Cohort | Low | High ⁺ | High |
| Takemura Y (1999) (Adult men - Japan) | III-2 | Case-control | High | High ⁺ | Low |
| Tumilehto J (2001) (Adults - Finland) | II | RCT | High | High ⁺ | High |
| Wannamethee SG (1998) (Adult men - UK) | III-2 | Cohort | High | High ⁺ | High |
| Wei M (1999) (Adult men - US) | III-2 | Cohort | High | High ⁺ | High |

⁺ Physical activity reduces the risk of developing Type 2 diabetes.

Magnitude rating: The direction of the effect is by '+' for a positive effect and '-' for a negative effect.

Section 5: Simple measures of physical activity

Issue

What simple measures can be used to determine physical activity and inactivity?

Recommendations

Physical activity should be measured in free-living subjects by:

- movement recorders, particularly the pedometer
- questionnaires focussing on leisure-time activities
- heart rate monitoring

Functional aerobic capacity should be measured from predictive equations based on gender, age, self-reported physical activity and body composition or BMI.

Evidence Statements

- Physical activity can be measured by the use of pedometers or accelerometers. At least 5 to 6 days recording that includes both weekdays and weekend days are required if intra-individual variance is to be kept below 5%
Evidence Level III[#]
- Questionnaires have been developed and validated for reliably measuring leisure-time physical activity
Evidence Level III[#]
- Detailed activity diaries can estimate energy expenditure accurately for populations but their inherent variability makes them less suitable for individual estimates
Evidence Level III[#]
- Heart rate monitoring can estimate total energy expenditure accurately if recording continues for at least 3 days
Evidence Level III[#]
- Functional aerobic capacity can be estimated without exercise testing by a predictive model based on gender, age, self-reported physical activity and either percentage body fat from skinfolds or BMI
Evidence Level III[#]
- Nomograms have been developed that provide an estimate of exercise capacity with age
Evidence level III[#]

[#] Studies assessed using the non-intervention assessment

Background – Measures of Physical Activity

The most accurate method available for the assessment of daily energy expenditure and physical activity in free-living subjects is the doubly labelled water method (DLW) (Schoeller and van Santen, 1982). Free-living is a term used to indicate that for the duration of the study these subjects are able to continue their normal activities outside the laboratory environment. In this method, a subject takes a drink containing the stable isotopes of hydrogen (^2H) and oxygen (^{18}O) which then enriches their body water for these isotopes. Habitual lifestyle and physical activity patterns may be continued while wash-out kinetics are determined for both isotopes in saliva samples as their concentrations in body water decline exponentially back towards natural abundance. ^2H enriches body water only whereas ^{18}O enriches both the water and bicarbonate pool; the slope of the wash-out line for ^{18}O will be steeper than the slope of the wash-out line for ^2H and the difference between these slopes will represent CO_2 production. CO_2 production can then be converted into energy expenditure via use of a food or respiratory quotient (Prentice, 1990). Although the DLW method has the very great advantage that it does not interfere with the patterns of everyday life, it is expensive and has a demanding methodology suitable only for specialised research (Bratteby et al, 1997). Other direct measures for estimating physical activity include questionnaires that can be filled out by the individual or by an interviewer (Jacobs et al, 1993), diaries to record activity (Bratteby et al, 1997), or motion sensors including pedometers (Gretebeck & Montoye, 1992), surveillance cameras or remote-reading telemetric devices (Montoye & Taylor, 1984). Indirect methods of assessing physical activity include assessment of usual energy intake by dietary measurements, measurement of body composition, physiological indicators such as pulse rate, muscular strength or fitness testing, survey of sports and recreational participation or recording of occupation (Paffenbarger et al, 1992).

Evidence – Measures of Physical Activity

Movement recorders

Physical activity in free-living subjects can be determined by a number of movement recorders. These include pedometers, which measure either distance travelled or number of steps taken and Caltrac[®] activity monitors (Gretebeck & Montoye, 1992) which measure the force in which movement is directed vertically, forwards and backwards or laterally. The simplest pedometers estimate only the number of steps taken, whereas more sophisticated pedometers estimate both distance and energy used, once the stride length is entered (Rowlands et al, 1997). A pedometer worn securely on the waist tends to be more accurate than one worn at the ankle since jolts to the ankle can cause the pedometer to count irregularly (Saris and Binkhorst, 1977). A study of the accuracy of five brands of electronic pedometers indicated that the Yamax Digiwalker DW-500[®] was particularly accurate at slow to moderate speeds (Bassett et al, 1996). Walking surface did not affect accuracy. The pedometer has the advantage of convenience of use, compatibility with most daily activities, objectivity and the possibility of repeated use. It also can be used in population studies where different ethnic groups may experience difficulty with self-report of physical activity using English and may be particularly applicable also for use in children (Rowlands et al, 1997).

Caltrac[®] activity monitors are motion sensors, which indicate activity in MJ (calculated from movement, age, gender, height and body weight). They have been reported to underestimate energy expenditure during slow walking, and overestimate energy expenditure during fast

walking (Montoye et al, 1983). Other activity monitors (eg the WAM[®] accelerometer or the Tritrac-RD3[®] three dimensional activity monitor are also available but are more expensive, and in the case of the Tritrac-RD3[®], relatively large in size (Rowlands et al, 1997).

A study was carried out in 30 healthy American men engaged in a wide variety of occupations, to determine how many days subjects would need to be monitored by pedometers or accelerometers to provide an estimate of habitual physical activity (Gretebeck & Montoye, 1992). The men were monitored during their waking hours for 7 continuous days, wearing a waist pedometer, an ankle pedometer, and three Caltrac[®] accelerometers. A repeated measures ANOVA showed that although daily measurements did not differ significantly when weekdays were compared, there were significant differences with the Caltrac[®] and waist pedometer measurements between weekend days and week-days ($p < 0.05$). At least 5 or 6 days, including both weekdays and weekend days, were required to minimise intra-individual variance (to $< 5\%$) in making physical activity determinations by these methods.

Questionnaires

Many different questionnaires have been developed to determine habitual or recent physical activity profiles. These range from a few questions, to very detailed inventories of activity patterns and they vary greatly in known reliability and validity. Ten of the most commonly used physical activity questionnaires have been evaluated in 78 American men and women with varying physical activity habits for the National Institutes of Health (Jacobs et al, 1993). Validity was studied by comparison with treadmill exercise performance, vital capacity, body fatness, the average of 14 four-week physical activity histories and the average of 14 two-day accelerometer readings. Interestingly, while most questionnaires related well to the presence of heavy intensity physical activity, fewer questionnaires related to the performance of light or moderate activity. The CARDIA physical activity and the Baecke physical activity questionnaires were found to have the highest correlation coefficient of $r= 0.83$ and $r= 0.71$, respectively. It was suggested that there is a need for future questionnaires to address this area as well as examining length of sleep, and patterns of occupational activity and household chores. While many questionnaires consider overall or habitual physical activity, others focus more specifically on leisure-time physical activity (LTPA), recognising that in western societies, LTPA makes the dominant contribution to overall physical activity. In particular, leisure-time is more susceptible to changes from health promotion or intervention programs than work-related activity (Montoye & Taylor, 1984).

A British group has developed an interview questionnaire specifically targeted at leisure-time rather than occupational physical activity (Lamb & Brodie, 1991). This questionnaire was validated in 77 British men and 41 British women from sedentary occupations. Measurements included a sit-and-reach test for lower-back flexibility, assessment of muscular strength via hand-grip dynamometry, leg power via a vertical jump test, and physical work capacity (PWC) via a submaximal cycle ergometer protocol. The LTPA questionnaire was shown to be stable following test-retest administration after a period of one month ($r=0.86$, $p < 0.0001$). The total LTPA determined by questionnaire was significantly related to PWC ($r= 0.48$, $P < 0.0001$). In addition, 'very heavy' LTPA, and 'heavy' LTPA were significantly related to PWC ($r= 0.55$ and $r=0.28$, respectively, both $p < 0.0001$), but there was no significant relationship between 'moderate' LTPA and PWC. Another group has assessed the validity and reliability of the MOSP-Q questionnaire used in the WHO-MONICA study (Roeykens et al, 1998). This questionnaire measures the average weekly time and energy expenditure spent

not only on LTPA but also during occupational work, on transport-related activities and on household chores. Subjects were 167 Belgian male and female university graduates aged 34-64 years. Test-retest reliability for the MOSPA-Q was found to be greater over the short-term than the long term. Over 38 days, test-retest reliability ranged from 0.57-0.91 whereas over 240 days reliability ranged from 0.45-0.85. Reliability for household chores and transport-related activity were found to be lower than that for LTPA or occupation-related physical activity. Validation studies indicated a strong correlation between calculated total energy expenditure and lean body mass ($r^2 = 0.53$, $p < 0.001$).

Activity diaries

The activity diary method uses a structured form to record activity levels on a scale of 1-9 for 96 periods each 15 min in length, for a day and a night. The categories range from 1: sleeping or resting in bed, to 9: maximal intensity sports activities such as competitive running. When the subject was uncertain how to code a given activity, they made an alphabetically coded note describing this activity in their diary (Bratteby et al, 1997). The method has been reported as simple and not difficult for subjects to learn and use (Bratteby et al, 1997). Records are simply processed by summing the number of 15 min periods that fall into each categorical value. The total for each category is then multiplied by the physical activity rating determined for this category of activity and by the basal metabolic rate (BMR) of the individual subject as predicted from age, sex and body weight by the Schofield equation (Schofield, 1985).

A study of 50 randomly selected 15 year old Swedish adolescents has been used to assess the use of physical activity diaries (Bratteby et al, 1997). The participants kept activity diaries for seven days and the total energy expenditure (TEE) and the ratio of TEE:BMR estimated from these diaries was compared with the results of DLW and indirect calorimetry measurements taken during the same period. Results indicated that the diary method provided a close estimate of TEE with the mean difference compared to the DLW method of 1.2%. The limits of agreement (mean difference 2 SD) were -3.47 and 3.77 MJ/day, the equivalent of a coefficient of variation (CV) of 15%. A close relationship and only a small bias was seen between the two methods but the variation was fairly large. This implies that the activity diary is suitable for providing a dose estimate of TEE in a population but is not suitable for use in making an individual estimate (Bratteby et al, 1997). Another study has compared the diary method with DLW in nine British men (Davidson et al, 1997). A detailed activity diary was kept for 9 days. Ten predefined coded activities ranging from 1, in bed to 10 walking briskly were recorded in this study in 5 min blocks. Additional codes were provided for different kinds of sports or other activities. BMR was determined with a ventilated hood respirometer and the energy cost of defined activities was measured in a calorimeter chamber and by Oxylog during leisure exercise. In this study the diary method was found to provide data $12.1 \pm 4.0\%$ (mean \pm SEM) lower than the DLW method.

Heart rate monitoring

Heart rate (HR) monitoring provides another alternative to whole body calorimetry or DLW for the measure of TEE. HR monitors are inexpensive, acceptable to subjects and are now storing minute-by-minute HR data for periods of more than 24h (Davidson et al, 1997). The appropriate use of HR monitoring, however, demands continuous monitoring for a period of at least three days (Gretebeck & Montoye, 1992). In addition, a calibration of the relationship between HR and energy expenditure at light, moderate and heavy work loads, together with a

determination of resting HR and assessment or prediction of BMR must be carried out for each individual subject (Bratteby et al, 1997). It has also been recommended that HR monitoring should be used primarily for assessing moderate to vigorous activity at heart rates > 120 bpm (Riddoch & Boreham, 1995).

A British study has compared the assessment of free-living energy expenditure in nine men, by continuous heart rate monitoring and by DLW (Davidson et al, 1997). Although all the subjects had a largely sedentary lifestyle, they had different levels of leisure time physical activity. Continuous HR monitoring was carried out over nine consecutive days. In addition, the HR - energy expenditure (EE) relationship was individually obtained for each subject using 30 min average values of HR and EE obtained during 24h whole-body calorimetry with a defined exercise protocol. Additional data points for individual leisure activities were also measured with an Oxylog portable oxygen consumption meter. After data processing, the HR-derived EE for these men was (mean \pm SEM) 6.0 ± 4.2 % higher than that estimated by DLW. Similar results have been reported in earlier studies (Schulz et al, 1989; Livingstone et al, 1990; Heini et al, 1991). One study showed no significant difference between the two methods (Lovelady et al, 1993).

As with the diary method, while HR monitoring provides accurate information on populations it is less accurate than DLW for assessment of EE in individuals. In the study carried out by Davidson and coworkers, the day-to-day CV in the estimation of TEE from HR monitoring ranged from 6.6 - 21.5% (Davidson et al, 1997).

Measurement of functional aerobic capacity

Maximal oxygen uptake (VO_{2max}) is the accepted index of cardiorespiratory fitness and functional aerobic capacity. It can be measured by exercise testing with indirect calorimetry, by the HR response at submaximal power output, or from the elapsed time to voluntary exhaustion with treadmill exercise (Jackson et al, 1990). The Rockport walking test provides an accurate estimate of VO_{2max} based on heart rate, age, sex, body weight and time necessary to complete a 2.2 km walk (Kline et al, 1987). These equations have been validated for use in women over the age of 65 (Fenstermaker et al, 1992).

Maximum oxygen uptake, either estimated or measured from the workload achieved for a given exercise protocol, may also be translated into a unitless measure, the MET, by dividing by 3.5. One MET is the quantity of oxygen consumed by the body under basal conditions. On average, this is equal to 3.5 ml oxygen/kg/min (Jette et al, 1990).

Attempts have also been made to estimate VO_{2max} without exercise testing due to the limitations in a clinical setting of applying these techniques. A study of 2,009 American subjects (mainly men) were randomly divided into validation (n=1543) and cross-validation (n= 466) groups (Jackson et al, 1990). The validation group was used to develop two predictive models to estimate VO_{2max} based on gender, age, self-reported physical activity and % body fat from skinfolds (model 1) or gender, age, self-reported physical activity and BMI (model 2). Results were compared with VO_{2max} measured during a graded exercise stress test by the Bruce treadmill protocol (Bruce et al, 1973). The non-exercise models were found to provide accurate VO_{2max} estimates for most of the subjects studied ($r= 0.81$, SE 5.3 ml. kg^{-1} . min^{-1} (model 1) and $r= 0.78$, SE 5.6 ml. kg^{-1} . min^{-1} (model 2). Estimations were only found to be inaccurate for the most aerobically fit subjects (those with $VO_{2max} > 55$ ml. kg^{-1} min^{-1}) and were therefore deemed appropriate for use for about 96% of the adult population.

Morris and coworkers have developed a nomogram based on METs and age, for assessing a subject's ability to perform exercise (Morris et al, 1993). The exercise test results of 1,388 male patients, aged 21-89 years, without apparent heart disease, were retrospectively reviewed. This referral group, plus subgroups of active (n=346) and sedentary (n=253) men were analysed to determine norms for age and for age by decades to exercise responses, including METs, maximal HR, and maximal systolic blood pressure. These data were used to generate regression equations from which a nomogram for calculating the degree of exercise capacity from age and METs was generated. It was argued that a nomogram providing an estimate of exercise capacity to age has clinical utility, since it can be used to quantify disability, and to communicate to patients their cardiopulmonary status. It can also be used to encourage improvement in exercise capacity.

Summary – Measures of Physical Activity

- Physical activity can be measured by the use of pedometers or accelerometers. At least 5 to 6 days recording that includes both weekdays and weekend days are required if intra-individual variance is to be kept below 5%
- The use of pedometers are the most practical and objective methods for determining energy expenditure
- Questionnaires have been developed and validated for reliably measuring both total physical activity and leisure-time physical activity
- Physical activity can also be estimated by the use of detailed activity diaries. The method is not difficult for subjects to learn and use. However, while the activity diary can estimate total energy expenditure accurately for populations, the diaries have an inherent variability that makes them less suitable for making individual estimates
- Total energy expenditure can be estimated by heart rate monitoring. Recording should continue for at least 3 days. Heart rate monitoring may provide an estimate of energy expenditure that is higher than that obtained by the doubly labelled water method. Heart rate monitoring appears accurate for populations but is less accurate than doubly labelled water for individual estimates
- Functional aerobic capacity can be estimated without exercise testing by a predictive model based on gender, age, self-reported physical activity and either percentage body fat from skinfolds or BMI
- Nomograms have been developed that provide an estimate of exercise capacity with age

Evidence Table – Section 5

What simple measures can be used to determine physical activity and sendariness?

| Author | Evidence | | | | |
|---|-------------------|-----------------|----------------|----------------------|------------------|
| | Level of Evidence | | Quality Rating | Magnitude Rating | Relevance Rating |
| | Level | Study Type | | | |
| Bassett DR (1996) (Adults – US) | III [#] | Cross-sectional | Low | High ^{P+} | High |
| Bratteby LE (1997) (Adolescents – Sweden) | III [#] | Cohort | High | High ^{D+} | High |
| Bruce RA (1973) (Adults - US) | III [#] | Cross-sectional | Medium | High ^{F+} | High |
| Davidson L (1997) (Adult men - UK) | III [#] | Cohort | Medium | High ^{H+} | High |
| Fenstermaker KL (1992) (Adult women – US) | III [#] | Cross-sectional | Medium | Medium ^{F-} | High |
| Gretebeck RJ (1992) (Adult men - US) | III [#] | Cross-sectional | Medium | High ^{H+} | High |
| Heini A (1991) (Adult women – Gambia) | III [#] | Case-control | Medium | High ^{H+} | Low |
| Jackson AS (1990) (Adults - US) | III [#] | Cross-sectional | Medium | Medium ^{F+} | High |
| Jacobs DJ (1993) (Adults – US) | III [#] | Cohort | Medium | Medium ^{Q+} | High |
| Kline GM (1987) (Adults - US) | III [#] | Cross-sectional | Medium | High ^{F+} | High |
| Lamb KL (1991) (Adults - UK) | III [#] | Cross-sectional | High | High ^{Q+} | High |
| Livingstone MB (1990) (Adults – UK) | III [#] | Cross-sectional | Medium | High ^{H+} | High |
| Lovelady CA (1993) (Adult women – US) | III [#] | Cross-sectional | Medium | Low | High |
| Montoye HJ (1983) (Adults – US) | III [#] | Cross-sectional | Medium | High ^{P+} | High |
| Morris CK (1993) (Adult men – US) | III [#] | Cohort | High | High ^{F+} | High |
| Roeykens J (1998) (Adults – Belgium) | III [#] | Cohort | Medium | High ^{Q+} | High |
| Saris WH (1977) (Adult men and children) | III [#] | Cross-sectional | Medium | High ^{P+} | High |
| Schulz S (1989) (Adults – Germany) | III [#] | Cohort | Medium | High ^{H+} | High |

[#] Studies assessed using the non-intervention assessment

⁺ The method assessed is a good estimate of energy expenditure; ^P Pedometer or accelerometer; ^D Activity diary; ^F Functional aerobic capacity; ^H Heart rate monitor; ^{Q+} Questionnaires.

Magnitude rating: The direction of the effect is by '+' for a positive effect and '-' for a negative effect. Low= no statistically significant effect

Section 6: High-fat diets, GDM and fat type

Issue

Do high-fat diets increase the risk of Type 2 diabetes and how is this affected by fat type?

Recommendations

Individuals at risk of developing Type 2 diabetes should have dietary intake assessed and should receive individualised dietary advice and continued dietetic support.

Individuals at risk should consume a diet with <30% energy as fat with <10% energy as saturated fat.

Evidence Statements

- People with a high intake of dietary fat are at increased risk of insulin resistance and Type 2 diabetes
Evidence Level III-2
- People with a high intake of saturated fat intake are at increased risk of insulin resistance and Type 2 diabetes
Evidence Level III-2
- Reduction in dietary fat to <30% with restriction of saturated fat to <10% of energy intake reduces the risk of Type 2 diabetes.
Evidence Level II

Background – Dietary Fat

There is evidence from mechanistic studies that dietary fat may play a part in insulin resistance and thereby predispose individuals to Type 2 diabetes and gestational diabetes (GDM) (Storlien et al, 1996). In this model fats are not only seen as fuel sources, but also as important components of membranes, precursors to powerful metabolites such as the eicosanoids and as potent gene regulators. The development of Type 2 diabetes and GDM is therefore likely to result from the interaction between genetic predisposition and environmental factors, such as diet, exercise and the development of obesity.

Evidence – Dietary Fat

Dietary fat, dietary saturated fat and risk of insulin resistance in Type 2 diabetes

The evidence obtained from cross-sectional and cohort studies concerning the relationship between dietary fat and the development of Type 2 diabetes and GDM is summarised in Table 1. It includes six cross-sectional and five cohort studies.

The cross sectional studies compared normal, impaired glucose tolerant and diabetic subjects in male and female populations, with different ethnic backgrounds. One of the largest and strongest, the San Luis Valley Study (n=1317) compared the nutrient intakes of men and women from different ethnic backgrounds who had normal, IGT or diabetic profiles (Marshall et al, 1991). The adjusted odds ratios relating to a 40 g/day increase in fat intake to development of Type 2 diabetes and IGT were 1.51 (CI 0.85-2.67) and 1.62 (CI 1.33-5.36), respectively. While dietary intakes did not differ significantly between normal and diabetic subjects, the group of subjects with IGT consumed significantly more fat, both as g/day and as a percentage of total energy, than the normal group ($p < 0.05$). These results are in agreement with results from the California based Kaiser Permanente Women Twins Study (N=544) which showed that total dietary fat intake was weakly associated with fasting insulin concentrations, although not if activity were taken into account (Mayer et al, 1993). In addition, higher intakes of each subtype of dietary fat were positively related to fasting insulin values, although only saturated fat was significantly related to 2h post-glucose load insulin values. This later relationship with saturated fat was not seen after adjustment for obesity (Mayer et al, 1993). Dietary fat may also be important in the development of GDM. In a study (n =35), in women with GDM from an area of NSW, it was shown by three-day diet record that women who had a recurrence of GDM consumed significantly more ($p < 0.001$) energy as fat than those who did not (Moses et al, 1997).

The San Luis Valley study was also analysed on a cohort basis (1984-1988), examining the progression to disease in both the non-diabetic population and the population with IGT. In the non-diabetic sample (n=1069), total fat and saturated fatty acids resulted in a significantly higher insulin response after adjusting for age, gender, ethnicity, vigorous activity, BMI, waist circumference and total energy. No effect was seen with unsaturated fat (Marshall et al, 1997). In the smaller IGT cohort (n=134), those who developed diabetes consumed significantly more fat ($p=0.02$) and monounsaturated fat ($p=0.03$) at baseline than those who did not (Marshall et al, 1994). The effect for saturated fat was only borderline ($p=0.06$), while no effect was seen for polyunsaturated fat ($p=0.74$). The small numbers in this study did not allow for an assessment of any mediating effect by activity, obesity or sex.

Table 1: Summary of studies on the risk of dietary fat and the development of Type 2 diabetes and GDM

| Authors | Type of study | Population | No. subjects | Clinical test | Dietary method | Dietary variables | Findings |
|-------------------|-----------------|---|--------------|-------------------------------|-------------------------------|-------------------------------|--|
| Adler 1994 | cross sectional | Alaskan, normal, IGT, diabetic | 660 | OGTT | 25 item FFQ | food items | Protective effect against IGT of seal oil, salmon consumption |
| Gittelsohn 1998 | cross sectional | Native Canadians Normal, IGT, diabetic | 721 | OGTT | 34 item FFQ | food groups | Increased risk of diabetes with 'junk foods', bread and butter |
| Marshall 1991 | cross sectional | San Luis Valley population, normal, IGT, diabetic | 1317 | OGTT | 24 hr recall | Nutrients | Percentage energy total fat increased odds for NIDDM and IGT |
| Mayer 1993 | cross-sectional | Women twins in California, non-diabetic | 544 | Fasting insulins | FFQ | Nutrients | Total dietary fat associated with fasting insulin but mediated by obesity |
| Mayer-Davies 1997 | Cross-sectional | Adults in California, Texas and Colorado | 1173 | OGTT | FFQ | Nutrients | Percentage energy from total fat associated with fasting insulin |
| Moses 1997 | cross-sectional | Women with GDM \pm recurrence of GDM, NSW | 35 | OGTT | DHx and FR | Nutrients | Women with recurring GDM consumed more total dietary fat than non-recrurers |
| Colditz 1992 | cohort | Women, non-diabetic | 84,360 | self report | 61 item FFQ | Nutrients, fat source | Vegetable fat consumption inversely associated with risk of NIDDM, attenuated by obesity |
| Feskens 1990 | cohort | Men Non-diabetic | 394 | OGTT, insulin under the curve | DHx | nutrients and foods | Saturated fat detrimental to glucose tolerance, vegetable fat negatively assoc with IAUC |
| Feskens 1995 | cohort | Men non-diabetic | 338 | IAUC | DHx | nutrients and food | Total fat and saturated fat associated with development of diabetes, but fish may have a protective effect |
| Marshall 1997 | cohort | San Luis Valley non-diabetic population | 1069 | OGTT | 24hr recall | Nutrients | Total and saturated fat associated with IGT |
| Marshall 1994 | cohort | San Luis Valley IGT population | 134 | OGTT | 24 hr recall | Nutrients | Total fat consumption associated with development of Type 2 diabetes |
| Pan 1997 | RCT | IGT population China | 577 | OGTT | Not measured during the study | Not measured during the study | When educated to consume less fat people were less likely to develop Type 2 diabetes |
| Tuomilehto 2001 | RCT | Finland IGT population | 522 | OGTT | 3 day Food diary | Nutrients | Those in the intervention group were less likely to develop Type 2 diabetes. |

These studies suggest that there is an effect of dietary fat on the development of Type 2 diabetes and GDM. This effect may be mediated by obesity and possibly also by physical activity levels. This finding has been confirmed in a cross-sectional study of 1,173 non-diabetic North American adults in the Insulin Resistance Atherosclerosis study which found that total fat intake was inversely related to insulin sensitivity, but that this was not significant when adjusted for BMI and WHR (Mayer-Davis et al, 1997). Further work on the effect of dietary fat subtypes on insulin resistance was suggested.

Evidence on the effects of fatty acid sub-types seem to clearly implicate saturated fat in the development of Type 2 diabetes, and there is some indication that polyunsaturated fats may be protective. The former finding was confirmed in a cross sectional study (n=4,304) involving the analysis of phospholipid fatty acid levels as indicators of dietary intakes. Fasting serum insulin was positively associated with the percentage of saturated fatty acids in the plasma phospholipid fraction (Folsom et al, 1996). The situation with monounsaturated fats is less clear, although it may relate to the food sources of these fats.

Food consumption and risk of development of Type 2 diabetes

Two cross-sectional studies of indigenous peoples have examined the impact of foods rather than nutrients on diabetes risk. In the Native Canadian Study (n=721), a high consumption of 'junk foods' (chips, hamburgers, pizza), bread and butter, or fatty methods of food preparation were associated with increased risk of diabetes [OR 2.40 (CI 1.13-5.10); OR 2.22 (CI 1.22-4.41); OR 2.58 (CI 1.11-6.02), respectively], (Gittelsohn et al, 1998). In contrast, a study on American Indians from Alaska found that none of the subjects who ate seal oil or salmon on a daily basis developed IGT or diabetes, whereas those who consumed neither of these products had a prevalence of IGT or diabetes of 9.9% (Adler et al, 1994).

Studies involving either men or women suggested further interesting patterns concerning foods as well as nutrients. The Nurses Health Study cohort (n=84,360) found intakes of vegetable fat in women with BMI < 29 kg.m⁻² was inversely related to the risk of diabetes (Colditz et al, 1992). This effect was adjusted for BMI, but physical activity was not assessed. Analysis of data on men from a number of Finnish and Dutch cohorts in the Seven Countries Study (n=338) found that a high intake of total fat especially saturated fat increases the risk of glucose intolerance and Type 2 diabetes (Feskens et al, 1995). Increased consumption of fish during the 20-year follow up was inversely related to 2 h glucose (p<0.05). An analysis of data from the Zutphen cohort alone (n=394) found that changes in saturated fat were positively associated with IAUC (p < 0.05) independent of subscapular skinfolds indicating that saturated fat intake may be detrimental to glucose tolerance (Feskens & Kromhout, 1990). The effect for polyunsaturated fat appeared to be the reverse (Table 2).

Table 2 : Pearson Correlations (r) between Habitual Daily Intake of Selected Nutrients and Glucose Tolerance in 394 Middle Aged Men : The Zutphen Study (Feskens and Kromhout, 1990).

| | Mean | SD | Fasting glucose (r) | IAUC (r) |
|-------------------------|------|------|---------------------|----------|
| Saturated fat (g) | 49.7 | 13.1 | 0.13** | -0.05 |
| Monounsaturated fat (g) | 51.9 | 12.8 | 0.10* | -0.02 |
| Polyunsaturated fat (g) | 20.2 | 7.4 | 0.03 | 0.04 |

* p<0.01

** p<0.001

In some countries, monounsaturated fats are predominantly consumed from animal sources (meat and milk), foods which also contain substantial amounts of saturated fats. In other countries for examples, those around the Mediterranean, monounsaturated fats are consumed predominantly from vegetable sources such as olive oil (Feskens et al, 1994). In the Nurses Health Study, vegetable fat was found to be inversely related to the risk of diabetes, so it may be that the effect of monounsaturates needs to be considered in the light of dietary source (Colditz et al, 1992). Further studies are required to clarify this.

Fish has become another recognised food source linked with reduction in risk of diabetes and whilst the links with polyunsaturated fat, notably n-3 polyunsaturates is attractive, this finding also needs to be explored further.

Reduction of dietary fat decreases the risk of Type 2 diabetes

Three intervention studies have examined the effect of dietary fat reduction in preventing the development of Type 2 diabetes in people with IGT. The Da Qing study (Pan et al, 1997), a 6 year study in China, compared the effect of diet modification, increased physical activity or a combination of both, on the development of diabetes in people with IGT. The study showed a cumulative incidence of diabetes in the control group of 67.7% (written instruction) compared with 43.8% in the diet group, 41.1% in the exercise group and 46.0% in the combined diet and exercise group which all had an individual assessment followed by regular small group education sessions (weekly for 1 month, monthly for 3 months and every 3 months after that). The diet component aimed to reduce body weight to BMI <25g/m², reduce fat consumption to 25-30% of total energy intake, achieve a consumption of 55-65% carbohydrate, 10-15% protein and consuming more vegetables.

The potential effect of diet on the prevention of Type 2 diabetes was shown from the preliminary results of the Finnish Diabetes Prevention Study (Eriksson et al, 1999). In the first year of this multicentre study of overweight subjects with IGT (n=523) significant weight loss occurred in the first 212 subjects (4.7 ± 5.5 vs 0.9 ± 4.1 kg in the intervention vs control groups, p < 0.001). In addition, plasma glucose concentrations were significantly lower after intervention (fasting 5.9 ± 0.7 vs 6.4 ± 0.8 mmol/L, p<0.001; and 2-h 7.8 ± 1.8 vs 8.5 ± 2.3 mmol/L, p < 0.05). The intervention involved both intensive dietetic management (<10% saturated fat, 20% monounsaturated fat and polyunsaturated fat, or up to 25% if the surplus is from monounsaturated fat; 50% carbohydrate and 1g /kg body weight protein) plus an individualised exercise prescription. The control group received general advice on diet and exercise (Eriksson et al, 1999).

The recently published final results of the Finnish Diabetes Prevention Study confirm the preliminary findings (Tuomilehto et al, 2001). After a 3.2 year follow-up the individual diet and exercise intervention conducted by a dietitian reduced the risk of diabetes by 58% (p<0.001) in a high risk population. This was achieved with 7 sessions in the first year and regular 3 monthly sessions during which goals were set on how to lose weight (>5%), decrease total fat intake to <30% of energy, decrease saturated fat intake to <10% of energy, increase fibre intake to >15 g/1000 kcal consumed and to exercise for at least 30 min per day. They were individually given guidance to increase their exercise levels (30 min per day) with a combination of endurance exercise and supervised resistance training. The control group were set goals and given written and oral information about diet (including the target for reduction in fat intake to less than 30% of daily intake) and exercise initially and then annually. The incidence of diabetes in the intervention group was significantly lower than the

control group (11% vs 23%). Diabetes did not develop in any of the groups who achieved 4 or 5 goals, however significantly more people were able to achieve these goals in the intervention group (49 in the intervention group vs 15 in the control group, $p < 0.001$ for each of the goals). In those who did not achieve any of the goals the incidence of diabetes was 38% vs 31% in the intervention and control groups respectively (Tuomilehto et al, 2001).

The Diabetes Prevention Program was a major clinical trial conducted in the United States over 3 years in a group of people with IGT and therefore at high risk of developing Type 2 diabetes. The intensive lifestyle intervention included instructed on a low fat diet, exercising for 150 minutes per week and behaviour modification skills. The results show on average that this group had a 7% weight loss in the first year and sustained a 5% loss for the study's duration and maintained 30 minutes of exercise per day. A 58% reduction in the risk of developing Type 2 diabetes was found compared to the control group who received only basic diet and exercise advice (National Institutes of Diabetes and Digestive and Kidney Diseases, 2001).

Summary – Dietary Fat

- There is moderately strong evidence from well-designed studies applicable to Australia that dietary fat has an effect on the development of Type 2 diabetes mellitus. More research is required to determine if this is mediated by obesity
- Dietary fat may have an effect on the recurrence of gestational diabetes mellitus although more research is required in this area
- There is moderately strong evidence from well designed studies applicable to Australia that dietary saturated fat has an effect on the development of Type 2 diabetes mellitus
- There is good evidence from well designed studies applicable to Australia that unsaturated fat of vegetable origin may have a protective or neutral effect on the development of Type 2 diabetes mellitus
- More research is required to determine the effect of monounsaturated fat on the development of Type 2 diabetes, taking into account whether it comes primarily from animal or vegetable origin
- There is some evidence that fish consumption has a protective effect on the development of Type 2 diabetes mellitus

Evidence table – Section 6

Do high-fat diets increase the risk of Type 2 diabetes?

| Author | Evidence | | | | |
|---|-------------------|-----------------|----------------|---------------------|------------------|
| | Level of Evidence | | Quality Rating | Magnitude Rating | Relevance Rating |
| | Level | Study Type | | | |
| Adler AI (1994) (Adult - US: Yup'ik Eskimo & Athabaskin Indians) | III-2 | Cross-sectional | Medium | High ⁺ | Low |
| Colditz GA (1992) (Adult women – US) | III-2 | Cohort | Medium | Medium ⁺ | High |
| Eriksson J (1999) (Adults - Finland) | II | RCT | High | High ⁺ | High |
| Feskens EJM (1990) (Adult men – The Netherlands) | III-2 | Cohort | Medium | High ⁺ | High |
| Feskens EJM (1995) (Adult men – The Netherlands & Finland) | III-2 | Cohort | Medium | High ⁺ | High |
| Gittelsohn J (1998) (Adults & adolescents – Canada: Native Canadians) | III-2 | Cross-sectional | Medium | High ⁺ | Low |
| Marshall JA (1991) (Adults - US: Hispanic & Non-Hispanic) | III-2 | Case-control | Low | Medium ⁺ | High |
| Marshall JA (1994) (Adults - US: Hispanic & Non-Hispanic) | III-2 | Cohort | Medium | High ⁺ | High |
| Marshall JA (1997) (Adults - US: Hispanic & Non-Hispanic) | III-2 | Cohort | Medium | Medium ⁺ | High |
| Mayer EJ (1993) (Adult women – US: Twins) | III-2 | Cross-sectional | High | High ⁺ | High |
| Mayer-Davis EJ (1997) (Adults – US) | III-2 | Cross-sectional | Medium | High ⁺ | High |
| Moses RG (1997) (Adult women – Australia: GDM) | III-2 | Case-control | Medium | High ⁺ | High |
| NIDDK (2001) (Adults – US) | II | RCT | High | High ⁺ | High |
| Pan XR (1997) (Adults - China) | II | RCT | High | High ⁺ | Medium |
| Tumilehto J (2001) (Adults – Finland) | II | RCT | High | High ⁺ | High |

⁺ Dietary fat increases the risk of Type 2 diabetes

Magnitude rating: The direction of the effect is by '+' for a positive effect and '-' for a negative effect.

Section 7: GDM and subsequent risk of Type 2 Diabetes

Issue

Does gestational diabetes mellitus increase the subsequent risk of Type 2 diabetes in mothers and infants?

Recommendations

Identification of women with GDM would allow:

- postnatal clinical interventions in those with diabetes persisting after delivery
- the option to use preventive methods to reduce the risk of Type 2 diabetes

Evidence statements

- GDM is associated with increased risk of future Type 2 diabetes in the mother
Evidence Level III-2
- GDM may be associated with increased risk of Type 2 diabetes in the offspring
Evidence Level III-2

Background – Gestational Diabetes Mellitus

Gestational diabetes mellitus (GDM), now defined as diabetes (or impaired glucose tolerance) first identified in pregnancy, was originally introduced as a means of identifying women at risk of future Type 2 diabetes (O'Sullivan, 1964). The criteria adopted were subsequently found to predict perinatal mortality and morbidity (O'Sullivan, 1973). Since these studies, obstetric care and maternal and perinatal outcomes have improved, the methods for measuring blood glucose have changed and a number of methods for diagnosing GDM have been introduced. There remains no global agreement on the criteria for the diagnosis of GDM making international epidemiological comparisons and interpretations difficult. Australia currently uses the Australasian Diabetes in Pregnancy (ADIPS) criteria for GDM (Hoffman et al, 1998). The recent change in criteria for Type 2 diabetes (ADA 1997; Alberti et al, 1998) are also likely to impact on estimates of conversion rates.

A further difficulty exists with the interpretation of the data, particularly among non-European ethnic groups: the proportion of women with previously undiagnosed Type 2 diabetes is unknown and related to the structure of health care delivery. The contributions from genetic, intra-uterine and environmental factors to the development of Type 2 diabetes in a given individual and population are likely to vary substantially and change over time. As to whether the GDM itself increases the risk of future Type 2 diabetes in the mother has not been established. The issue is complicated as increasing parity may (Kritz-Silverstein 1989; Simmons 1992) or may not (Collins et al, 1991; Manson et al, 1992) be associated with an increased risk of Type 2 diabetes. Practical issues relating to studies including loss to follow up and need for long follow up periods also may impact on the estimates of progression to Type 2 diabetes and generation of data.

Investigating the possible impact of *in utero* exposure to maternal hyperglycaemia has been the subject of two follow-up studies. As GDM predicts future Type 2 diabetes in a high proportion of women and Type 2 diabetes is associated with increased rates of Type 2 diabetes in the offspring, GDM might be expected to be associated with an increased risk of Type 2 diabetes in the offspring. Whether anti-hyperglycaemic intervention during GDM reduces the risk of obesity and Type 2 diabetes in the offspring remains unknown although insulin therapy was associated with reduced adiposity by 2.8 years in one study among Polynesians and South Asians (Simmons and Robinson, 1997).

Evidence - Gestational Diabetes Mellitus

GDM and the subsequent risk of Type 2 diabetes in mothers

There are a large number of cohort studies indicating an increased risk of Type 2 diabetes in women who had GDM. Recent studies using the 75g oral glucose tolerance test on follow up are shown on Table 3.

Kjos (1995) followed up 671 Latino women for up to 6 years who had experienced past GDM but without Type 2 diabetes immediately after delivery. New cases developed at a rate of 11.1 (CI 9.3-12.9)/100 person years. The development of Type 2 diabetes was associated with the area under the oral glucose tolerance test (OGTT) curve at 4-16 weeks post-partum as well as the area under the OGTT curve during pregnancy, the gestational age at which GDM was diagnosed and the highest fasting glucose achieved during pregnancy. The authors

Table 3. Cohort studies indicating an increased risk of Type 2 diabetes in women with GDM

| <i>Group</i> | <i>No.</i> | <i>Follow-up (yrs)</i> | <i>% Type 2</i> | <i>Control</i> | <i>Reference</i> |
|----------------|------------|------------------------|-----------------|----------------|------------------|
| Navajo Indians | 111 | 11 | 53 %* | (ND) | Steinhart, 1997 |
| Copenhagen | 345 | 5.9 | 13 % | 0 % | Damm, 1989 |
| Sweden | 145 | 3-4 | 3.4 % | 0 % | Persson, 1991 |
| Australia | 881 | 17 | 40 %* | 10 % | Henry, 1991 |
| US Latinos | 671 | 6 | 55 % | (ND) | Kjos, 1995 |
| Trinidad | 60 | 3.5-6.5 | 62 % | (ND) | Ali, 1990 |
| Zuni Indians | 94 | 6 | 36 %* | 10 % | Benjamin, 1993 |

* Life Table, ND: not done

suggested that a history of GDM imparts a risk for Type 2 diabetes greater than that observed when patients are not pregnant.

The mechanisms for increased risk of GDM for Type 2 diabetes have been investigated. Damm and coworkers (1995) studied 91 women with past GDM and 33 controls using an OGTT and demonstrated reduced insulin secretion among women with past gestational diabetes. Others demonstrated a delayed insulin peak response (Catalano et al, 1986; Persson et al; 1991) while another normal insulin secretion (Efendic et al, 1987). Buchanan and coworkers (1998) studied insulin sensitivity among 122 Latino women in the third trimester. There was no association with subsequent diabetes or impaired glucose tolerance within 6 months post-partum. Defects in insulin sensitivity after adjusting for obesity have not yet been shown among women with past GDM.

Peters and coworkers (1996) followed up 666 Latino women with GDM in a past pregnancy, of which 87 subsequently became pregnant again. The additional single pregnancy among Latino women increased the risk of developing Type 2 diabetes within the subsequent 7.5 years by a rate ratio of 3.34 (CI 1.80-6.19).

GDM and the subsequent risk of Type 2 diabetes in the offspring

The 1,064 offspring of Pima women with diabetes in pregnancy were more likely to have diabetes by the age of 20-24 years than offspring of women developing diabetes after pregnancy or not at all (45% versus 8.6% versus 1.4%) (Pettitt et al, 1988). This was independent of paternal diabetes status.

Pettitt and coworkers (1983) undertook a review of the follow-up studies among 2,638 Pimas. The study attempted no new analyses but repeated the same analyses on later expanded data sets. The study demonstrated that the higher the glucose antenatally, the more hyperglycaemic the offspring by the age of 19 years. Similar findings have been shown in animal experiments and in the other long-term follow up study (Silverman et al, 1995). In a cross sectional study of 86 European and 432 Polynesian women, women with past diabetes in pregnancy were 2.05 fold more likely to have diabetic offspring than diabetic women without known past diabetes in pregnancy (Simmons 1995).

Summary - Gestational Diabetes Mellitus

- GDM is associated with an increased risk of current and future Type 2 diabetes in women and probably obesity and Type 2 diabetes in the offspring
- Whether this is causal or part of an underlying diabetogenic process is unknown
- Additional pregnancies complicated by GDM might increase the risk of Type 2 diabetes in women with past GDM
- There is also good experimental and cohort evidence to support the hypothesis that intrauterine exposure to hyperglycaemia increases the risk of Type 2 diabetes in the offspring
- Whether the hyperglycaemia in GDM (besides that from undiagnosed Type 2 diabetes) is sufficient to be causal is also unknown
- Whether anti-hyperglycaemic treatment for GDM would reduce the risk of future Type 2 diabetes in the mother or offspring is unknown

Evidence Table - Section 7

Does GDM increase the risk of Type 2 diabetes in mothers and /or infants?

| Evidence | | | | | |
|---|--------------------------|-------------------|-----------------------|-------------------------|-------------------------|
| Author | <i>Level of Evidence</i> | | <i>Quality Rating</i> | <i>Magnitude Rating</i> | <i>Relevance Rating</i> |
| | <i>Level</i> | <i>Study Type</i> | | | |
| Ali Z (1990) (Adult women – Trinidad) | III-2 | Cohort | High | High ⁺ | Low |
| Benjamin E (1993) (Adult women - US: Zuni Indians) | III-2 | Case-control | High | High ⁺ | Low |
| Buchanan TA (1998) (Adult women - US: Hispanic) | III-2 | Cohort | Medium | High ⁺ | Low |
| Catalano PM (1986) (Adult women - US) | III-2 | Case-control | Medium | High ⁺ | High |
| Damm PD (1989) (Adult women - Denmark) | III-2 | Case-control | High | High ⁺ | High |
| Damm P (1995) (Adult women - Denmark) | III-2 | Case-control | High | High ⁺ | High |
| Efendic S (1987) (Adult women - Sweden) | III-2 | Case-control | Low | High ⁺ | High |
| Henry OA (1991) (Adult women – Australia) | III-2 | Case-control | High | Medium ⁺ | High |
| Kjos SL (1995) (Adult women – US: Latino) | III-2 | Cohort | High | High ⁺ | Low |
| Persson B (1991) (Adult women - Sweden) | III-2 | Case-control | High | High ⁺ | High |
| Peters RK (1996) (Adult women - US: Latino) | III-2 | Cohort | High | High ⁺ | Low |
| Pettitt DJ (1983) (Adolescents - US: Pima Indians) | III-2 | Cohort | Medium | High ⁺ | Low |
| Pettitt DJ (1988) (Adolescents - US: Pima Indian) | III-2 | Cohort | Medium | High ⁺ | Low |
| Silverman BL (1995) (Adolescents - US) | III-2 | Case-control | Medium | High ⁺ | High |
| Simmons D (1995) (Adults & adolescents - New Zealand: Europeans, Maori, Pacific Island) | III-2 | Cross-sectional | Medium | Medium ⁺ | High |
| Steinhart JR (1997) (Adult women - US: Navajo Indian) | III-2 | Cohort | High | High ⁺ | Low |

⁺ Mothers with GDM and there infants are at increased risk of Type 2 diabetes

Magnitude rating: The direction of the effect is by '+' for a positive effect and '-' for a negative effect.

Section 8: Birth weight as a later risk of Type 2 Diabetes

Issue

How does birth weight affect later risk of Type 2 diabetes?

Recommendation

In view of the association between low birth weight and the later development of diabetes, studies are required to evaluate interventions aimed at reducing low birth weight and the impact this has on the development of Type 2 diabetes

Evidence statements

- Low birth weight is associated with increased risk of Type 2 diabetes in populations of European descent
Evidence Level III-2
- Low birth weight may be associated with increased risk of Type 2 diabetes in some populations not of European descent
Evidence Level III-2
- High birth weight is not related to future Type 2 diabetes in populations of European descent
Evidence Level III-2

Background – Birth Weight

The debate over the relative contributions of environment and genes in the aetiology of Type 2 diabetes has been added to by the hypothesis that exposure to an adverse intrauterine environment leads to permanent alterations in metabolism ("programming"). Two intrauterine mechanisms have been discussed, the "thrifty phenotype hypothesis" (Hales & Barker, 1992) and the "fuel mediated teratogenesis hypothesis" (Freinkel, 1980). These two hypotheses postulate that fetal intra-uterine under- and over-nutrition respectively lead to Type 2 diabetes in adult life. The putative manifestations of these two phenomena are a relatively low and high birth weight respectively. An alternative hypothesis for a small baby to be associated with Type 2 diabetes later on in life has been that this is a manifestation of the "thrifty genotype". That is, the fetus able to survive in an adverse intrauterine environment is genetically endowed with an ability to survive as an adult in times of poor food supply, but develops obesity and Type 2 diabetes when food is plentiful (Hattersley & Tooke, 1999).

There is a large range of confounders in prospective studies of birth weight. There is often substantial loss to follow up, in one study this being as high as 94.2% (Fall et al, 1998). The characteristics of those not followed up are unknown. The use of birth weight as a measure is fraught with difficulties relating to gestational age at delivery, impact of smoking, maternal comorbidity (eg renal disease and hypertension) and the family history of diabetes in the parents. Birth weight itself is a composite of skeletal and non-skeletal components and it may be more appropriate to use placental weight and/or other neonatal anthropometric measurements as the key indicators (eg caliper measurements, length, head circumference, ponderal index). To determine the ponderal index the birth weight is divided by the cube of the birth length.

Evidence – Birth Weight

Weight at birth and later risk of Type 2 diabetes

A low birth weight, but not a high birth weight has been associated with an increased risk of Type 2 diabetes on follow-up in a number of studies of populations of European decent. (Hales et al, 1991; Phipps et al, 1993; Fall et al, 1995; Curhan et al, 1996; Lithell et al, 1996; Olah et al, 1996; Rich-Edwards et al, 1999). In populations that are not of European decent a relationship between low birth weight and future Type 2 diabetes has been shown among Mexican Americans (Valdez et al, 1994), but not South Asians (Fall et al, 1998) or African Americans (Hulman et al, 1998). In a Danish twin study, the lighter twin was found to be more hyperglycaemic than the larger twin (Poulsen et al, 1997). Among Pima Indians, a U-shaped curve was found with high rates of Type 2 diabetes among those with the highest and lowest birth weights after 25-34 years (Pettitt and Knowler, 1998). In Indians, those who were short at birth but with a relatively high birth weight were most likely to have developed diabetes after 39-60 years (Fall et al, 1998).

While an association between fetal size and risk of future Type 2 diabetes is consistent among people of European descent, the cause for the reduced fetal size remains unclear in humans with the evidence for fetal under-nutrition being causal not being supported (Stanner et al, 1997; Mathews et al, 1999). In a prospective cohort study of 693 nulliparous women (those who have never given birth to a viable infant) in England, placental and birth weight were

unrelated to intake of any macronutrient (Mathews et al, 1999). Each milligrams increase in Vitamin C was associated with a 50.8 (4.6-97.0)g increase in birth weight and 3.2(0.4-6.1)% increase in placental weight. In a follow-up study of those exposed in utero to profound calorie restriction during the siege of Leningrad, there was no significant increase in the prevalence of the insulin resistance syndrome (Stanner et al, 1997).

The use of Ponderal Index rather than birth weight was associated with a stronger relationship with future Type 2 diabetes among Swedes (Lithell et al, 1996; McKeigue et al, 1998). A relationship was also found between insulin sensitivity as measured by euglycaemic clamp and birth weight (McKeigue et al, 1998). Among European populations, those with a low birth weight, and found to be overweight on follow up were most likely to have developed Type 2 diabetes (Lithell et al, 1996; McKeigue et al, 1998; Yarbrough et al, 1998).

Summary – Birth Weight

- Among populations of European decent, the data shows a relationship between reduced fetal size at birth and increased Type 2 diabetes in adult life
- The mechanism behind this relationship remains speculative ranging from thrifty genotype survival to epiphenomenon to fetal undernutrition
- The evidence for fetal under-nutrition being causal is not supported
- Among populations not of European decent, the data is not consistently supportive of a relationship between low birth weight and future Type 2 diabetes, and more research should be done in this area
- The evidence relating high birth weight with future Type 2 diabetes is weak

Evidence Table - Section 8

How does weight at birth affect later risk of Type 2 diabetes?

| Evidence | | | | | |
|---|--------------------------|-------------------|-----------------------|-------------------------|-------------------------|
| Author | <i>Level of Evidence</i> | | <i>Quality Rating</i> | <i>Magnitude Rating</i> | <i>Relevance Rating</i> |
| | <i>Level</i> | <i>Study Type</i> | | | |
| Curhan GC (1996) (Adult men – US) | III-2 | Cohort | Medium | High ⁺ | High |
| Fall CHD (1995) (Adult women – UK) | III-2 | Cohort | Medium | High ⁺ | High |
| Fall CHD (1998) (Adults – India) | III-2 | Cohort | Medium | Medium ⁺ | Medium |
| Hales CN (1991) (Adult men – UK) | III-2 | Cohort | High | High ⁺ | High |
| Hulman S (1998) (Adults – US: African Americans) | III-2 | Cohort | Medium | Low | Low |
| Lithell HO (1996) (Adult men – Sweden) | III-2 | Cohort | High | Medium ⁺ | High |
| Mathews F (1999) (Adult women – UK) | III-2 | Cohort | High | Low | High |
| McKeigue PM (1998) (Adult men – Sweden) | III-2 | Cohort | High | High ⁺ | High |
| Olah KS (1996) (Adult women – UK) | III-2 | Cohort | High | High ⁺ | High |
| Pettitt DJ (1998) (Adult women & children – US: Pima Indians) | III-2 | Cohort | High | High ⁺ | Low |
| Phipps K (1993) (Adults – UK) | III-2 | Cohort | High | High ⁺ | High |
| Poulsen P (1997) (Adults – Denmark) | III-2 | Cohort | High | High ⁺ | High |
| Rich-Edwards JW (1999) (Adult women – US) | III-2 | Cohort | Medium | High ⁺ | High |
| Stanner SA (1997) (Adults – Russia) | III-2 | Cross-sectional | Medium | Low | High |
| Valdez R (1994) (Adults – US: Mexican American, Non-Hispanic white) | III-2 | Cohort | Medium | Low | Medium |
| Yarbrough DE (1998) (Adult women – US) | III-2 | Cohort | Medium | High ⁺ | High |

⁺ Those with a low birth weight have an increased risk of developing Type 2 diabetes.

Magnitude rating: The direction of the effect is by '+' for a positive effect and '-' for a negative effect. Low = no statistically significant effect

Section 9: Low glycaemic index diets

Issue

Do low glycaemic index (GI) diets reduce the risk of Type 2 diabetes?

Recommendations

Diets of low energy density and containing a wide range of carbohydrate foods rich in dietary fibre and of low glycaemic index (cereals, vegetables, legumes and fruits) are recommended to reduce the risk of Type 2 diabetes.

Evidence Statement

- Diets of high glycaemic index may increase the risk of Type 2 diabetes in both men and women particularly when associated with low cereal fibre intake
Evidence Level III-2

Background – Glycaemic Index

The glycaemic index (GI) has been used to classify carbohydrate foods for various applications, including for use in diets designed for the treatment of Type 2 diabetes (Brand et al, 1991; Fontvieille et al, 1992; Frost et al, 1994; Miller, 1994; Jarvi et al, 1999). The GI classifies foods based on their potential to raise blood glucose concentrations. It is defined as:

$$GI = \frac{\text{Incremental area under blood glucose curve in response to 50g test carbohydrate} \times 100}{\text{Incremental area under blood glucose curve for 50g carbohydrate from standard food}}$$

A standardised protocol has been developed for determination of GI, which includes repeated testing in each individual subject (FAO/WHO expert consultation on carbohydrates in human nutrition, 1998). Either white bread or glucose is generally used as the standard food. A list of GI values for almost 600 individual foods has now been published (Foster-Powell & Miller, 1995), and GI can be predicted from total dietary fibre content for foods with no published value for GI (Nishimune et al, 1991). The GI can be applied to mixed meals (Hollenbeck & Coulston, 1991) and to whole diets by calculation of the weighted GI value of the meal or diet. When calculating the GI for a whole diet, an adjustment can be made for the amount of carbohydrate present in each meal (Wolever & Bolognesi, 1996).

The GI can be used, together with information about energy density, macronutrient and dietary fibre content, to guide food choices. The GI can be used to develop lists of low GI foods such as legumes, bulgur, whole grain cereals, whole grain pumpernickel breads, and pasta. Information is available for particular local and ethnic foods (Mani et al, 1990; Indar-Brown et al, 1992; Ayuo & Etyang, 1996) and different types of rice (Miller et al, 1992). It is most important that food composition is used as well as GI when making food choices. Foods high in fat have low GI but would not be recommended for inclusion in diets for individuals at risk of Type 2 diabetes, as they are energy dense. Chocolate (GI=49), icecream (GI= 61), potato crisps (GI= 54) and shortbread (GI=48) are examples of such inappropriate low GI foods. On the other hand, some high GI foods may be a good choice, for example a dietary staple such as rice, or foods with low energy density and high nutrient content, such as some vegetables (eg, carrots) (FAO/WHO Expert Consultation on carbohydrates in human nutrition, 1998). Sucrose raises blood glucose levels less than some refined starchy foods such as mashed potatoes (Wolever & Miller, 1995) but provides energy without nutrients. Nevertheless, the addition of a small amount of sucrose may be useful to improve the taste and palatability of a low GI food such as baked beans (Voster et al, 1987).

Evidence – Glycaemic Index

The Glycaemic Index and the risk of Type 2 diabetes

Few longitudinal studies have examined the relationship between the glycaemic index of the diet and the risk of Type 2 diabetes. It was examined, however, in 42,759 American men without diagnosed diabetes or heart disease, who were aged 40-75 years in 1986 (Salmeron et al, 1997a). The diet at baseline was assessed by a validated semi-quantitative food frequency questionnaire. During six years of follow-up in this study, 523 cases of Type 2 diabetes were reported. The average dietary glycaemic index value was determined for each participant as was the dietary glycaemic load which was used as an indicator of glucose response or insulin demand. This was determined by averaging the GI of the foods consumed. Data were adjusted

for age, BMI, smoking, physical activity, family history of diabetes, total energy intake (but not fat intake) and consumption of alcohol and cereal fibre. After these adjustments, the GI of the diet was found to be related to diabetes risk. Comparing the lowest and the highest quintiles for GI, the relative risk of Type 2 diabetes was 1.37 (CI 1.02-1.83, p trend= 0.03). The combination of a high glycaemic load and a low intake of cereal fibre further increased the risk of diabetes [2.17 (CI 1.04-4.54)].

A similar study has been carried out in American women (Salmeron et al, 1997b). A total of 65,173 American women, free of known diabetes, heart disease or cancer, and aged 40-65 years completed a detailed dietary questionnaire in 1986. After six years of follow-up there were 915 reported cases of Type 2 diabetes. After adjustment, as in the study in men above, the dietary GI was again found to relate to the risk of Type 2 diabetes. When the highest quintile for GI was compared to the lowest quintile, the relative risk was 1.37 (CI 1.09-1.71). Again, the combination of a high glycaemic load and a low intake of cereal fibre further increased the risk of Type 2 diabetes [2.50 (CI 1.14-5.51)]. There was no association with fruit or vegetable fibre. Significant positive associations were seen with cola beverages, white bread, white rice, french fries, and cooked potatoes.

In these studies the GI of the diet (and thereby the glycaemic load) was determined by averaging the GI of the individual foods as reported in the food frequency questionnaires. A concern of this approach is that it does not take into account other factors that may affect the GI of the food. For example fat and certain types of fibre delay gastric emptying which reduces the GI of the meal (Frost & Dornhorst, 2000). Furthermore, foods consumed at breakfast give a lower GI than the same foods consumed at lunch, the 'second meal effect' (Frost & Dornhorst, 2000). However although these other factors may influence the glycaemic response, the relative response of one carbohydrate to another remain the same (Frost & Dornhorst, 2000). Further prospective studies are required in this area before the full significance of the GI can be determined.

Summary – Glycaemic Index

- There is some evidence from large studies of a population similar to that of Australia, that a diet of high glycaemic index may contribute to an increased risk of Type 2 diabetes
- There is also evidence in these studies that the risk of Type 2 diabetes is further increased by the combination of a high glycaemic load and a low intake of cereal fibre
- Further longitudinal studies are needed in both men and women that are not based on dietary self-reporting including the use of food frequency questionnaires
- Further studies examining the influence of particular types of dietary fibre on the prevention of Type 2 diabetes should also be done

Evidence Table - Section 9

Do low glycaemic index diets reduce the risk of Type 2 diabetes?

| Author | Evidence | | | | |
|--|-------------------|------------|----------------|-------------------|------------------|
| | Level of Evidence | | Quality Rating | Magnitude Rating | Relevance Rating |
| | Level | Study Type | | | |
| Salmeron J (1997a) (Adult men – US) | III-2 | Cohort | Medium | High ⁺ | High |
| Salmeron J (1997b) (Adult women – US) | III-2 | Cohort | Medium | High ⁺ | High |

⁺ Low glycaemic diets decrease the risk of developing Type 2 diabetes.

Magnitude rating: The direction of the effect is by '+' for a positive effect and '-' for a negative effect.

Section 10: Psychosocial stress

Issue

Does psychosocial stress increase the risk of Type 2 diabetes?

Recommendation

More research is required to assess the impact of psychosocial stress or a major depressive episode in affecting the risk of Type 2 diabetes.

Evidence Statements

- Individuals with newly diagnosed diabetes may report a history of psychosocial stress
Evidence Level II[#]
- Exposure to undesirable life events may increase the risk of diabetes in later life
Evidence Level III[#]

[#] Studies assessed using the non-intervention assessment

Background – Psychosocial Stress

Stress has been considered as a possible risk factor for the development of Type 2 diabetes. Although human studies on the role of stress in the onset of diabetes are limited, there is evidence from animal studies that stress consistently produces hyperglycemia (Surwit, 1996). In humans, three categories of stress can be distinguished: chronic 'background' stress relating to the pressures of western lifestyles, acute 'occasional' or unexpected stress and stress associated with individual temperament (Ionescu-Tirgoviste et al, 1987). Reaction patterns to stress have been separated into 'defense' reactions and 'defeat' reactions (Henry, 1993). The 'defense' reaction, leading to control, is characterised by increased noradrenaline, gonadotrophins and, in men, testosterone. The 'defeat' reaction, when control is lost, is associated with activation of the hypothalamo-pituitary adrenal (HPA) axis, increase in ACTH and cortisol and decrease in gonadotrophins and peripheral sex hormones (Bjorntorp, 1997). Stress-related cortisol secretion has been found, in middle-aged men, to be strongly related to BMI, WHR, fasting insulin, glucose, blood lipids, systolic and diastolic blood pressure and heart rate (Rosmond et al, 1998). Both chronic and acute stress also alter the level of glycosylated haemoglobin (HbA_{1c}) (Cesana et al, 1985; Netterstrom et al, 1988; Kawakami et al, 1989). Indigenous populations subjected to social environmental stress associated with cultural breakdown, discrimination, loss of land, poverty and lack of control over living conditions exhibit elevated HbA_{1c} compared to populations from western cultures (Daniel et al, 1999). This elevation of HbA_{1c} was apparent in all glucose tolerance categories, which is an interesting observation due to the very high prevalence of Type 2 diabetes reported for many indigenous populations once they are subjected to urbanisation.

Evidence – Psychosocial Stress

Effect of psychosocial stress on the risk of Type 2 diabetes

The effect of mood states on HbA_{1c} levels were examined in a cross-sectional study of 51 Japanese men aged 30-60 years, employed by an electrical company (Kawakami et al, 1995). Six moods were assessed by the Profile of Mood States (POMS) questionnaire (McNair et al, 1971). Anger-hostility, but not tension-anxiety, depression, fatigue, vigour or confusion was found to be significantly and positively related to HbA_{1c} ($r= 0.30$, $p < 0.05$). This relationship was still evident after controlling for the number of cigarettes smoked per day ($p < 0.05$). There was no relationship however, between urinary catecholamine excretion and HbA_{1c} (Kawakami et al, 1995).

The effect of the psychosocial work environment on diabetes was examined in a cross-sectional study of an occupational cohort in France (Niedhammer et al, 1998). Volunteers ($n=13,226$) employed by an electrical company were given three psychosocial work environment social scores to assess psychological demands, decision latitude and social support at work. Although psychosocial work factors were found to be associated with the presence of hypertension or hyperlipidemia within the preceding 12 months, no association was found with diabetes.

Another Japanese study examined the effects of social support, stress and the effect of mood states on the presence of glucose intolerance (Fukunishi et al, 1998). Subjects were 600 Japanese aged 28 to 72 who had presented for primary health care screening and agreed to further hospital-based tests. All were given a 75g oral glucose tolerance test and placed into 3

groups on the basis of the level of impaired glucose tolerance. The subjects also took Japanese versions of three psychological questionnaires: the Stress and Coping Inventory (SCI) (Rahe and Veache, 1995), the Dealing with Illness (DWI) test (Namir et al, 1987) and the Profile of Mood States (POMS) test (McNair et al., 1971). Subjects with the greatest degree of glucose intolerance were found in the SCI to use less social support either from social groups or from individuals.

A further indication of the potential importance of emotional support from others in mitigating the effects of stress on diabetes risk was found in an American study (Krause, 1995). In this study, non-institutionalised Americans over the age of 65 years, from all states except Alaska and Hawaii were contacted using the Health Care Finance Administration (HCFA) Medicine Beneficiary Eligibility List. This list identifies all older US residents who have a social security number. Interviews were completed with 1,068 participants in which the presence of diabetes was determined by self-report, stressful life events were recorded through a 49-item checklist and social support was measured with a four item index. Logistic regression models were then developed to determine if diabetes could be predicted by combined stressful life events, social support, and the interaction between stress and social support in addition to age, sex, education and obesity. It was found that, at the lowest observed level of emotional support, an increase of one undesirable stressful life event increased the risk of diabetes by around 30% [OR 1.307 (CI 1.151-1.464)]. In contrast, at the highest observed level of emotional support, an increase of one undesirable stressful event did not increase diabetes risk [OR 0.972 (CI 0.865-1.080)]. Risk of diabetes increased with the number of stressors experienced. Thus the impact of two stressful events for those receiving the lowest level of emotional support was OR= 1.709 (CI not reported). In this study, participants were also asked to identify 3 of 8 social roles with which they identified most strongly. Stressors occurring in any of these three roles identified as important were 'salient stressors'. The risk of having diabetes in older adults with little social support who experience two salient stressors was 2.406 (CI not reported).

The role of an acute stress in the development of Type 2 diabetes has been examined in relation to a severe earthquake in Bucharest, Romania on 4 March, 1977 (Ionescu-Tirgoviste et al, 1987). This earthquake had a magnitude of 7.2 on the Richter scale and lasted 75 sec. The number of patients newly registered with diabetes at a single Bucharest clinic was compared for the month of May one year previously, 2 months after or 1 year after the earthquake. In May, 2 months after the earthquake (156 cases) were registered, a significantly higher number of patients than in May one year before the earthquake (98 cases) or in May one year after the earthquake (101 case), $p < 0.001$. In 24 cases, the onset of diabetes occurred within days after the earthquake.

To examine the role of chronic and subacute stress as a factor present at diabetes onset, 300 newly diagnosed Romanian patients aged 36-76, with newly diagnosed diabetes were given a detailed stress-related questionnaire (Ionescu-Tirgoviste et al, 1987). Past chronic or subacute psychosocial stress was recorded in 44% of cases, and the incidence was higher in women (54%) than in men (39%). In 44% of cases, the stress levels experienced were rated 'extreme', in 37% stress levels were rated 'severe' and in 19% stress levels were rated 'moderate' according to WHO criteria.

Summary – Psychosocial Stress

- There is moderate evidence from a study in Japanese men that anger-hostility levels are positively related to levels of HbA_{1c}. Similar studies on the effects of mood states on HbA_{1c} levels are required in European populations
- There is moderate evidence that exposure to stressful events may increase risk of diabetes and that this increased risk may be mitigated if individuals receive social support. Studies are needed to fully determine physiological mechanisms for these environmental influences
- More research is required into the possible links between stress and Type 2 diabetes as more sensitive methods for measuring markers of acute and chronic stress are developed

Evidence Table - Section 10

Does psychosocial stress increase the risk of Type 2 diabetes?

| Author | Evidence | | | | | |
|---|-------------------|--------|---|----------------|---------------------|------------------|
| | Level of Evidence | | | Quality Rating | Magnitude Rating | Relevance Rating |
| | Level | Rating | Study Type | | | |
| Eaton WW (1996) (Adults – US) | II [#] | High | Cohort | Medium | High ⁺ | High |
| Fukunishi I (1998) (Adults – Japan) | III [#] | Medium | Cross-sectional | Medium | Medium ⁺ | Low |
| Garvard JA (1993) | III [#] | High | Case-control | Medium | High ⁺ | High |
| Ionescu-Tirgoviste C(1987) (Adults – Romania) | III [#] | Medium | Cohort Cross-sectional Case-control | Low | Medium ⁺ | Low |
| Kawakami N (1995) (Adult men – Japan) | III [#] | Medium | Cross-sectional | Medium | Medium ⁺ | Low |
| Krause N (1995) (Adults – US) | III [#] | Medium | Cross-sectional | Medium | High ⁺ | High |
| Leedom L (1991) (Adults – US) | III [#] | Medium | Case-control | Medium | High ⁺ | High |
| Niedhammer I (1998) (Adults – France) | III [#] | Medium | Cross-sectional | Medium | Low | High |
| Palinkas L (1991) (Adults – US) | III [#] | Medium | Cross-sectional | High | Medium ⁺ | High |
| Wing RR (1990) (Adults – US) | III [#] | Medium | Case-control | Medium | High ⁺ | High |

[#] Studies assessed using the non-intervention assessment

⁺ Psychosocial stress or a major depressive disorder is increased in those with diabetes

Magnitude rating: The direction of the effect is by '+' for a positive effect and '-' for a negative effect. Low = no statistically significant effect

Section 11: Diet and physical activity to prevent excessive weight gain

Issue

What forms of diet and physical activity can be recommended to prevent excessive weight gain in childhood and throughout adult life?

Recommendations

Programs of diet and exercise education in children should include parental involvement and use behavioural techniques to reinforce lifestyle change.

Adults who have achieved weight loss require ongoing programs involving diet, exercise and social support to prevent weight regain, however the effectiveness of specific long term programs requires further evaluation.

Evidence Statements

Childhood

- Family therapy is more effective in preventing obesity in children than conventional therapy
Evidence Level II
- Repeated visits to a multi-disciplinary health care program focussing on dietary change can effectively induce weight loss in children and adolescents. Outcomes are better in children seen at least every 2 months over a year than in children seen less often
Evidence Level III-2
- Intervention programs to increase exercise levels in addition to dietary change can reduce obesity in children
Evidence Level II
- Obese children placed on low energy diets exhibit significantly better long- term weight loss than their obese parent
Evidence Level III-2
- Programs in children aimed at reducing sedentary behaviour produce more body fat loss after a year than programs aimed at increasing exercise activity or programs that combine both approaches
Evidence Level II

Evidence Statements continued

Adults

- Mass media campaigns have not successfully prevented weight gain in adult communities
Evidence Level I
- Targeted dietary and exercise programs based on an empowerment model comprising diabetes awareness sessions, exercise groups and cooking demonstrations have prevented weight gain in a high-risk population
Evidence Level III-2
- Although body weight can be readily lost during active supervised treatment, it is often gradually regained during the subsequent unsupervised period
Evidence Level II
- In men taught dietary and exercise strategies, waist circumference may decline during active supervised treatment and remain reduced for up to 2 years
Evidence Level III-2
- Men and women who have successfully maintained long-term weight loss report the continued consumption of a low energy, low fat diet
Evidence Level II
- In women, an ad libitum low fat diet is superior to a low energy diet in maintaining weight after a major weight loss
Evidence Level II
- Exercise is an important determinant in the success of weight maintenance after a weight loss program
Evidence Level I
- A program of combined diet and exercise is more effective in maintaining weight loss than either diet alone or exercise alone
Evidence Level II
- Weight gain after a weight loss program is positively associated with hours of weekly television viewing
Evidence Level III-2
- In women, increased general physical activity is as effective as aerobic exercise in preventing weight regain after a period of weight loss
Evidence Level II
- In women, the threshold of energy expenditure required for weight maintenance is 80 minutes/day of moderate activity or 35 minutes/day of vigorous activity added to a sedentary lifestyle
Evidence Level III-2

Adults continued

- In men and women, neither aerobic training nor weight training exercise programs are able to reverse the decline in resting metabolic rate that occurs after significant weight loss
Evidence Level II
- Continued contact with a therapist may aid weight maintenance after a weight loss program
Evidence Level I
- Recruitment of overweight people into weight reduction programs together with their family and friends influences weight loss but not long-term weight maintenance
Evidence Level II
- Overweight men with impaired glucose tolerance who followed a long-term program of diet and exercise exhibited reduced all-cause mortality after a period of 12 years
Evidence Level III-2

Background – Prevent Weight Gain

Obesity, especially adiposity around the abdomen, appears to be the major potentially modifiable determinant of insulin resistance and the risk of developing Type 2 diabetes (Ferrannini & Camastra, 1998). Rates of overweight and obesity have increased dramatically in recent years in Australia, as has happened in most other Westernised countries (Australian Institute of Health & Welfare, 1998; Hill & Peters, 1998). Such weight gain appears to be the response to an environment where energy output is insufficient to counteract the effects of inappropriately high energy intakes resulting from the widespread availability of convenience and other energy dense foods (Hill & Peters, 1998). Thus it would appear that the best hope of reducing the prevalence of Type 2 diabetes in the Australian community lies in the early prevention of excessive weight gain both by changing energy intake and increasing energy expenditure.

It is important to target programs establishing healthy eating and physical activity patterns to children so that they can then be maintained life-long (Huon et al, 1999). It has been suggested that there are three time-periods that appear to be particularly appropriate in which to concentrate efforts for obesity prevention in adults (Wing, 1995). These periods are young adulthood, from the ages of 25 to 35 while the subjects are still healthy and the perimenopausal period in women. Programs are also needed to prevent long-term weight regain in adults who have successfully completed weight loss programs (Glenny et al, 1997). The best way to structure interventions for the prevention of obesity still requires much clarification both at the community and individual level (Wing et al, 1995). To date, health professionals, the general public and policy makers have been too reluctant to recognise that obesity poses a serious public health threat (Hill & Peters, 1998).

Evidence – Prevent Weight Gain in Childhood

Prevention of obesity in children

In a recent systematic review Glenny (1997) identified only one study focussing on the prevention rather than the treatment of obesity in children. The study examined the prevention of progression to severe obesity in Swedish school children aged 10-11 years with a body mass index (BMI) $>23 \text{ kg.m}^{-2}$. Conventional therapy (dietary counselling by a dietitian and regular visits to a paediatrician) was compared to conventional therapy plus family therapy (six sessions over 12 months) and to a no treatment group. Treatment continued for 14-18 months. At one year follow-up the proportion of children with severe obesity (BMI $>30 \text{ kg.m}^{-2}$) was significantly less in the family therapy group than in the control group (5% versus 29% respectively, $p < 0.02$). In contrast, the proportion of children with severe obesity in the conventionally treated group did not differ significantly from the control group. Based on these results, Glenny (1997) has argued that the family therapy approach for prevention of childhood obesity should now be trialed in other age groups and settings and on larger numbers of children.

Treatment of obesity in childhood

A retrospective study carried out in San Paulo, Brazil has evaluated the impact of a multi-disciplinary program on weight control in children and adolescents (Valverde et al, 1998).

Treatment consisted of individual visits to a paediatrician and psychologist and to a nutritionist for dietetic counselling at one urban centre. Information was offered to enable patients to make adequate food choices, avoiding excessive energy or fat intake and consumption of high energy density foods. This advice was reinforced at regular clinic visits (visits ranged for 2-20 with a mean interval between visits of 2 months). Over the period of a year, 198 children and adolescents were counselled (mean age was 9.25 years mean body mass index (BMI) 24.26 kg.m⁻² (range 19.1-40.31)). At each visit, an adjusted BMI was calculated for the child to relate actual BMI to BMI values at the 50th percentile for a reference population of the same age. Progress in weight control was then evaluated as percentage change in adjusted BMI. For both boys and girls, adjusted BMI declined significantly between the first and last clinic visit (p<0.0012, p<0.000 respectively). Significantly better outcomes occurred among children who made six or more clinic visits (p<0.000). Variables such as percent body fat, body shape at the first visit, family obesity pattern, length of obesity and pubertal stage did not significantly affect outcomes.

An American study (Epstein et al, 1995b) examined the effects of increasing activity on weight change in obese children. Obese children from 61 families recruited by newspaper advertisement were randomised into three treatment groups. In each family at least one parent was also willing to attend the treatment sessions. The treatment of one group aimed to decrease sedentary behaviour, the treatment of the second group aimed to increase exercise activity while the third group reinforced both aims. The study did not include a control group without intervention. Children in all groups and participating parents were instructed in the 'Traffic Light Diet'. In this diet *Green* foods (primarily vegetables) are low energy and nutritious; *Yellow* foods, for example skim milk or apples, are higher in energy but contain important nutrients whereas *Red* foods, such as potato chips and sweets, are high in energy but low in nutrients. Children were instructed to limit *Red* foods to seven or less per week. After four months on the program, children in the group in which sedentary behaviour had been targeted now showed a significantly higher preference for high intensity exercise activities than children in the combined group or in the group in which increased physical activity had been targeted (p<0.02). After one year on this program, the group in which sedentary behaviour had been targeted exhibited a greater decrease in percentage body fat as estimated by bioelectrical impedance, than the combined group or the group in which increased physical activity had been targeted (p<0.05).

A study by Johnson and coworkers (1997) also used the 'Traffic Light' diet to treat childhood obesity. Thirty-two obese children aged 8 - 16 years were given information alone (control group), or they were given a dietary program preceded or followed by an exercise program. While the impact of combined exercise and dietary interventions endured at a five-year follow-up, children in the control group remained morbidly obese.

Treating childhood obesity appears to be more effective than treating adults. Children aged 8 years and older and an obese parent from 113 families participating in treatment programs were studied (Epstein et al, 1995a). Both children and adults were placed on similar energy-reduced diets based on the 'Traffic Light' system. After 6 months, 5 or 10 years the children were found to have a significantly greater decrease in percentage body weight than their parents (p<0.001, p<0.022 and p<0.001 at 6 months, 5 or 10 years, respectively).

Obesity is growing as a health problem in children from many ethnic groups. An intervention program in Singapore has addressed the problem of obesity in Malay, Chinese and Indian children (Ray et al, 1994). The study sample comprised 1,128 obese preschool children aged

3-6 years who either attended 17 Primary Health care clinics with their family or saw a dietitian for counselling on diet and exercise. After one year, 456 children (40.4%) had reduced their obesity while 228 (20.2%) had attained a normal weight. Progress varied significantly with ethnicity ($p < 0.05$) with Indian children reducing obesity more than Chinese children, who in turn reduced their obesity more than the children of Malay origin. Pima Indian children in the United States also have very high rates of obesity. In 1996 a diabetes primary prevention program called Quest was implemented as a pilot study at one elementary school for Pima Indian children in kindergarten and grades 1 and 2. The program included classes in nutrition, increased physical activity and a structured school breakfast and lunch program. Results however, have not yet been reported (Cook & Hurley, 1998). Although some other intervention programs have also been recently reviewed, the number of programs designed explicitly for children remains low (Huon et al, 1999).

Despite the poverty of data on preventative and treatment programs for obesity in children, it appears that preventing excessive weight gain in childhood is an achievable goal. Hill & Peters (1998), point out nevertheless that we still need to know more about how children's eating habits develop and how they can be modified to foster a preference for less energy dense foods. In addition, we need a better understanding of factors affecting physical activity patterns in children and how these patterns develop and continue into adulthood (Hill & Peters, 1998). Successful treatment programs for obesity in children use behaviour modification techniques (Huon et al, 1999) integrated with advice for improved nutrition and proper eating habits, regular exercise and a more active lifestyle, and are supported by continued family involvement (Glenny, 1997). Overweight children and their families during therapy should be treated with sensitivity and compassion, avoiding any emphasis on physical appearance or relating obesity to lack of willpower (Barlow & Dietz, 1998). In addition, care needs to be taken that messages targeted to prevent childhood obesity do not unintentionally promote inappropriate weight loss in underweight adolescent girls dissatisfied with their body image and size (O'Dea, 1998).

There is also a need for long term follow-up studies to determine the most effective dietary, exercise and psychological strategies for use in preventing obesity in children. Decreasing sedentary behaviour appears to be one of the most effective approaches (Epstein et al, 1995b). Television watching, a sedentary activity much enjoyed by children, may also prompt increased food intake via food commercials (Jeffery et al, 1982). Television viewing has been cross-sectionally and prospectively related to obesity (Dietz & Gortmaker, 1985). It has also been suggested that increased intake of low energy density, fibre-rich foods may aid prevention of weight gain in overweight children (Kimm, 1995).

The role of parental involvement in weight management of children appears important but requires clarification in further studies (Glenny, 1997). While there may be benefit in younger children attending therapy sessions with a parent (Israel et al, 1994), in adolescents outcomes may be better if the child and parent attend support sessions separately (Glenny, 1997). Parental involvement in the success of juvenile weight management may also extend much further than the support role since parental attitudes to their child's obesity, their belief that obesity can be modified by lifestyle factors and their readiness to make lifestyle changes may all influence outcomes (Barlow & Dietz, 1998).

Evidence – Prevent Weight Gain in Adult Life

Preventing obesity in adults

A systematic review based on one database (Medline 1966-96) which sought large studies (>50 participants) of long duration (> 2 years) has identified three community based prospective cohort design studies that aimed to reduce the prevalence of obesity through education via the mass media (Douketis et al, 1999). In each study, no significant difference in body weight was evident between control and test communities during a 3 to 7 year follow-up period. Each study however, was found to suffer methodological problems, which limited their conclusions (Douketis et al, 1999). These included the lack of defined criteria to specify a successful outcome, a high rate of withdrawal, and a reliance on self-reported body weight. A more recent study (Dyson et al, 1997) has examined the effects of basic (n=116) or reinforced (n=111) healthy-living advice on body weight in self-referred hyperglycemic but non-diabetic volunteers at five centers, two in France (Lyon, Toulouse) and three in Britain (Exeter, Leicester, Oxford). After three months, the mean body weight in both groups showed a significant reduction of 1.5 kg ($p < 0.001$) but at follow-up after one year, weight loss was non-significant in each case.

More success was achieved in another prospective study (Simmons et al, 1998) that assessed the impact of a 2-year pilot diabetes risk reduction program in a high risk population of Western Samoans living in Auckland, New Zealand. This involved comparison of two church congregations of similar socio-economic status led by the same pastor, a control congregation (n=115) and an intervention congregation (n= 67). The culturally specific intervention followed an empowerment model and comprised diabetes awareness sessions, exercise groups and cooking demonstrations. At 2 year follow-up, body weight had remained stable (0 ± 4.8 kg) at the intervention church but had increased by 3.1 ± 9.8 kg at the control church ($p=0.5$). In the intervention church there was an associated increase in the proportion exercising regularly (+22% versus -8% in the control group, $p < 0.05$) and consumption of key fatty foods was also significantly reduced (eg; the proportion cutting the fat off meat had increased, $p < 0.001$). In an Indian study (Viswanathan et al, 1997) 262 non-diabetic adults who had one or more parents with Type 2 diabetes were given individualised dietary and exercise programs and followed up after four or more years. During this period 26% exhibited no weight change, 31% exhibited a decrease in body weight while 42% became more overweight. Weight gain occurred mainly among those who had not adhered to dietary and exercise prescriptions and 59% of those with weight gain developed Type 2 diabetes.

Maintenance of reduced weight levels after a period of weight loss

Dietary therapy for obesity, assessed in a recent systematic review (Douketis et al, 1999), is usually effective during the period of active supervised treatment, but is then followed by a pattern of gradual weight regain during the subsequent unsupervised follow-up period.

One such example is a New Zealand study where 39 men and women followed an *ad libitum* intake, low fat eating program while taking placebo tablets as the control arm of a study on a weight-loss drug (Carmichael et al, 1998). Mean body weight after 6 months on this program was significantly lower than baseline ($p < 0.0001$) but during an additional 3 months unsupervised follow-up body weight began to increase (mean increase from 6-9 months, 0.7 kg, $p=0.06$). Another study (Grodstein et al, 1996) examined the efficacy of weight maintenance in 192 Americans who had participated in a commercial weight loss program involving 12 weeks on a formula diet. After a follow-up period of about three years, mean weight (by self-report) was only modestly less than at baseline (102.6 kg and 105.9 kg, final and initial weight, respectively). Forty percent of subjects gained back more than they had lost during the diet. Weight regain in the 12 months after a weight loss program has also been reported by Jeffery and coworkers (1995) in 74 women who lost weight either by dietary energy or by dietary fat restriction. It was also seen at one-year follow-up in a study of 46 non-diabetic women who had a family history of Type 2 diabetes (Pascale et al, 1995). At a 96-week follow-up of 22 obese women who had undertaken a weight loss program, only 10% of the initial weight loss was maintained (Weinstock et al, 1998). Also in a study of self-referred subjects with hyperglycaemia ($n=227$), randomised to reinforced or basic healthy-living advice, although mean body weight reduced by 1.5 kg by 3 months, weight returned to baseline after 1 year (Dyson et al, 1997). Due to this phenomenon of weight regain after therapy, it is considered imperative to build effective weight maintenance strategies into any weight loss program (Glenny et al, 1997).

Shick and coworkers (1998) determined the habitual diet of a group of American who sustained an average weight loss of 30kg (minimum 13.6 kg) and maintained this for an average of 5.1 years (minimum 1 year). Both women ($n=355$, mean age 45 years) and men ($n=83$, mean age 50 years) reported continued consumption of a low energy, low fat diet (mean 23.5 and 24.3% energy from fat, respectively). In another study Toubro and Astrup (1997) compared the efficacy of an *ad libitum* low fat diet with a reduced energy diet in maintaining weight after major weight loss. Subjects were 41 obese Danish women (BMI 27-40) who had achieved similar weight loss after either 8 weeks on a low energy diet (2 MJ/day) or 17 weeks on a reduced energy diet (5 MJ/day). After one year on the weight maintenance program, the group randomised to the *ad libitum* low fat diet had maintained 13.2 kg (CI 8.1-18.3 kg) of an initial 13.5 kg weight loss. The group randomised to a reduced energy diet had maintained 9.7 kg (CI 6-13.3 kg) of an initial 13.8 kg weight loss. One year later, at follow-up 65% of the group given the low fat diet versus 40% of the group given the reduced energy diet had maintained a weight loss greater than 5 kg ($p < 0.07$). The rate of the initial weight loss did not influence these long-term outcomes.

Obese subjects are usually already moderately physically active but are limited in their ability to perform higher intensity exercise. While exercise without dietary change does not seem effective in promoting weight loss, it has been argued that exercise combined with dietary change is very important in promoting weight maintenance following a period of weight loss (Miller et al, 1997). In a systematic review of the literature from 1969-1994, Miller and coworkers (1997) found evidence that diet plus aerobic exercise was more effective than a

program of either diet alone or exercise alone in maintaining weight loss at one year follow-up. Whereas those on diet and exercise maintained 77% of their initial weight loss at one year, those on diet alone maintained only 56% and those on exercise alone maintained only 53% of initial weight loss. Grodstein and coworkers (1996) followed up participants in a commercial weight loss program. Three years after initial weight loss, the strongest predictor of continued weight loss was exercise frequency. For every hour of exercise per week, there was a decrease of 2.0 kg (CI-3.0-1.0 kg) after adjustment for months of regular exercise, hours per week watching television and age. Conversely, television was associated with an increase in weight. For each hour of television watching per week, there was an increase of 0.3 kg (CI 0.1-0.5 kg). Another study (Andersen et al 1999) has assessed the effects of the type of exercise on the success of a weight maintenance program. Forty obese women (mean age 42.9 years) attended a university-based weight management program where they were given a low energy diet (5 MJ/day) combined with either structured exercise (step aerobics) or increased physical activity in a moderate lifestyle change (eg increased walking, stair-climbing) for 16 weeks. After the program, both aerobic and lifestyle groups exhibited similar mean weight loss (-8.3 and -7.9 kg, respectively). Participants in the aerobic group assessed at one-year follow-up had regained 1.6 kg while participants in the lifestyle group had regained 0.08 kg ($p=0.06$). Weight regain was significantly higher in those who reported the lowest physical activity levels during follow-up (lowest versus highest $p<0.001$). Wadden and coworkers (1998) also followed weight change in 77 obese women treated for 48 weeks with diet alone or diet combined with supervised aerobic exercise, supervised strength training or both aerobic exercise and strength training. Weight losses were similar in all groups and at one year follow-up participants had regained 35-55% of their lost weight. Yet those who reported exercising regularly in the 4 months preceding the follow-up assessment regained significantly less weight than those who did less exercise ($r=-0.44$, $p<0.005$).

Exercise has also proved effective in maintaining long-term loss of abdominal fat in the Australian "GutBuster" program (Egger et al, 1996). These studies focussed on reduction in waist girth in men rather than on weight loss. Using instructors qualified in exercise or dietetics, the men were taught strategies to reduce dietary fat, increase dietary fibre, and to increase low intensity movement (both planned and incidental). In the program they were also allowed to 'trade' additional moderate alcohol intake for a period of increased movement. In the first study 42 men completed the 6 weeks course and were followed up 2 years later when waist and hip circumferences, body weight and BMI still showed a significant reduction from baseline (all $p<0.001$). Waist circumference had declined in 64% of these men between week 6 and the 2-year follow-up. No change was evident in the waist:hip ratio due to the finding that both hip and waist circumferences had declined. In a second study, 87 men were followed up for one year after the initial 6 week course while 37 were followed up after completing both the initial course and an additional 6 week 'advanced' course. In these groups, 39% of men and 67% of men respectively, achieved a greater waist loss after 12 months than exhibited at 6 weeks.

A prospective study has been carried out to determine how much exercise is required to optimise weight maintenance (Schoeller et al, 1997). Thirty-two American women, mean age 38 years, were recruited within three months of achieving a documented mean targeted weight loss of 23 kg. The women were followed for 12 months while undertaking habitual exercise. At study entry, total energy expenditure (TEE) was measured with doubly labelled water while the resting metabolic rate (RMR) and the thermic effect of a meal (TEM) were measured by respiratory gas exchange. Physical activity was measured by 7-day recall, 3-day heart rate monitoring and as TEE minus (RMR + TEM). At 3,6,9 and 12 months body weight

was measured and physical activity was estimated by 7-day recall. For analysis, subjects were divided into three groups on the basis of physical activity at baseline. After one year, physically active women (mean TEE:RMR=1.89, n=9) gained 2.5 ± 3.1 kg; less physically active women (mean TEE:RMR=1.64, n= 15) gained 9.9 ± 10.5 kg while sedentary women (mean TEE:RMR=1.44, n= 15) gained 7.0 ± 5.9 kg ($p<0.01$). Retrospective analyses of weight regain as a function of energy expended in physical activity indicated that there was a threshold of physical activity required for weight maintenance. In women the apparent threshold was at 47 KJ per kg body weight per day, corresponding to an expenditure of 80 minutes per day of moderate activity or 35 minutes per day of vigorous activity added to a sedentary lifestyle.

Mechanisms where by exercise aids weight maintenance have not yet been established. It has been suggested that exercise might help maintain the resting metabolic weight, which characteristically falls after weight loss, making it easier to regain weight. Ballor and coworkers (1996) addressed this issue in a study. Eighteen men and women between the ages of 55 and 70 years were randomised to aerobic training (by treadmill exercise) or weight training for a period of 12 weeks following a successful weight loss program (mean loss 9 ± 1 kg). Although the aerobic training group lost significantly more additional weight than the weight training group ($p< 0.05$), neither type of training reversed the depression of resting metabolism that had occurred following weight loss.

Continued contact with the therapist may also aid weight maintenance. This conclusion was reached by Glenny et al, (1997) following a systematic review of five long term studies that examined the effects of continued contact. However, since not all studies reviewed showed the benefit of contact it was also argued that more research is needed to develop new and more effective weight maintenance programs. A recent study by Wing & Jeffrey (1999) has also indicated the importance of social support in the success of weight maintenance programs. Overweight subjects (n=166) were recruited either alone or with three friends or family members. Each subject was then randomised to receive 16 weeks treatment on a diet and exercise program with or without an additional social support intervention. A higher proportion of subjects recruited with friends and receiving social support completed the 10 month study (95%) than in other groups (75-83%, $p=0.048$). Both recruitment approach and social support intervention had significant effects on weight maintenance ($p< 0.4$ and $p< 0.009$, respectively). For participants who were recruited alone but received social support during weight loss, 24% maintained their weight loss in full. In contrast, for participants who were recruited with friends but also received social support during weight loss, 66% maintained their weight loss in full. Despite these benefits at 10 months, by 16 months no significant difference remained evident in weight loss between the four different treatment groups.

Diet and exercise in people with Impaired Glucose Tolerance (IGT)

Despite the difficulties in adults of maintaining weight loss in the longer term, there is emerging evidence that participation in diet and exercise programs can be of substantial benefit. A recent study in Sweden has shown that overweight men with IGT (n= 288) who followed a long-term intervention program of physical exercise and dietary change exhibited significantly reduced all-cause mortality after a 12 year follow-up. This change in men with IGT on an intervention program was relative to men with IGT (n= 135) who had received

routine treatment (mortality 6.5 versus 14.0 per 1,000 person years at risk respectively, $p=0.009$) (Eriksson & Lindgärde, 1998).

Although weight loss through diet or diet and exercise programs can be effective in reducing obesity, the longer-term maintenance of body weight at the reduced level may be very difficult. Extensive review of the literature suggests that a weight maintenance program should be a priority after the initial first six months of weight loss therapy. Such a weight maintenance program should employ a combination of low-energy diets, increased physical activity and behavioural therapy and should be continued indefinitely (National Institutes of Health Expert Committee, 1998).

Summary – Prevent Weight Gain

- Although children are a very important target group, there is an evident paucity of studies examining the prevention of obesity in childhood and adolescence
- There is evidence that family therapy has efficacy in preventing obesity in prepubertal children. Further studies are needed to extend this finding to other age groups
- There is good evidence, although in a population group rather dissimilar to Australia, that weight loss in children is improved by repeated visits (made at least once every two months) to a health care centre
- There is good evidence that the addition of exercise to a dietary program for children will aid obesity reduction . Programs aimed at reduction of sedentary behaviours are even more effective than programs aimed at increasing physical activity
- In adults there is strong evidence that mass media campaigns are unable to prevent community weight gain
- There is good evidence that dietary and exercise programs using a culturally specific empowerment model comprising of diabetes awareness sessions, exercise groups and cooking demonstrations can be successful when targeted at a high-risk population
- There is strong evidence that adults regain weight in the long-term once active supervision for weight loss ceases
- In men there is very good evidence that a focus on loss of waist circumference will produce long-term loss of abdominal fat
- Adults who successfully maintain weight loss long-term are those more likely to consume a low fat diet
- There is very strong evidence that a program of diet combined with exercise is necessary to maintain weight loss in the long term
- In adults continued contact with the therapist in the longer term plus the support of family and friends will aid long-term weight maintenance

Evidence Table - Section 11

Do diet and exercise interventions prevent weight gain or promote weight loss in childhood or adult life?

| Author | Evidence | | | | |
|--|-------------------|---|----------------|--|------------------|
| | Level of Evidence | | Quality Rating | Magnitude Rating | Relevance Rating |
| | Level | Study Type | | | |
| Anderson RE (1999) (Adult women - US) | II | RCT | Medium | High ^{D & A+} | High |
| Ballor DL (1996) (Adults - US) | II | RCT | Medium | Medium ^{D&A+; D&W+} | High |
| Carmichael HE (1998) (Adults - New Zealand) | II | RCT | Medium | High ^{D+} | High |
| Douketis JD (1999) | I | Systematic review | Medium | Medium ^{D+; P+; S+; B+} | High |
| Dyson PA (1997) (Adults - France; UK) | II | RCT | High | Low | High |
| Egger G (1996) (Adult men - Australia) | III-2 | Cohort | Medium | High ^{D & B+} | High |
| Epstein LH (1995a) (Children & Parents - US) | III-2 | Cohort | Medium | High ^{B(c)+; B-} | High |
| Epstein LH (1995b) (Children - US) | II | RCT | Medium | High ^{B+} | High |
| Eriksson KF (1998) (Adult men - Sweden) | III-2 | Cohort | Medium | High ^{D&A+} | High |
| Glenny AM (1997) | I | Systematic review | High | High ^{B(c)+, B+; D&A+; P+, S+,} | High |
| Grodstein F (1996) (Adults - US) | III-2 | Cohort | Low | Low | High |
| Huon GF (1999) (Children) | III-2 | Systematic review of Case-control studies | Low | Low | High |
| Israel AC (1994) (Children - US) | II | RCT | Medium | Low | High |
| Jeffery RW (1995) (Adult women - US) | II | RCT | Medium | Low | High |
| Johnson WG (1997) (Children - US) | II | RCT | Medium | High ^{D&A+} | High |
| Miller WC (1997) (Adults) | I | Systematic review | Medium | High ^{D+; D&E+} | High |
| Pascale RW (1995) Adult women - US) | II | RCT | Medium | Medium ^{D+} | High |
| Ray R (1994) (Children - Singapore) | III-2 | Cohort | Medium | High ^{B+} | Medium |
| Schoeller DA (1997) (Adult women - US) | III-2 | Cohort | Medium | High ^{A+} | High |
| Shick SM (1998) (Adults - US) | III-2 | Cohort | Medium | High ^{D+} | High |
| Simmons D (1998) (Adults - New Zealand) | III-2 | Cohort | Medium | High ^{B+} | Medium |
| Toubro S (1997) (Adults - Denmark) | II | RCT | Medium | High ^{D+} | High |
| Valverde MA (1998) (Children & adolescents - Brazil) | III-2 | Cohort | Low | Medium ^{D&B+} | Low |
| Viswanathan M (1997) (Adults - India) | III-2 | Cohort | Medium | High ^{D&A+} | Medium |
| Wadden (1998) (Adult women - US) | II | RCT | Medium | Medium ^{D+; A+; W+} | High |
| Weinstock RS (1998) (Adult women - US) | II | RCT | Low | Medium ^{D+; D&A+; D&W+} | High |
| Wing RR (1999) (Adults - US) | II | RCT | Medium | High ^{B+} | High |

D = diet; A = aerobic training; W = weight training; S = surgery; B = behaviour therapy; P = pharmacological; c = children; a = adults

* Intervention decreased the risk of developing Type 2 diabetes .

Magnitude rating: The direction of the effect is by '+' for a positive effect and '-' for a negative effect. Low = no statistically significant effect

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2.5

Prevention Guideline Search Strategy And Yield

Electronic Databases Searched:

Medline
Embase
CINAHL
Cochrane
Psychinfo for Stress information only

Terms used to search the databases:

Detailed within the table below

Search Inclusion Criteria:

Where possible the searches were limited by the English Language and Human Research. The databases were searched for the following years of publication: Medline 1990 - 99; Embase 1988 - 99; CINAHL 1982 - 99; Cochrane 1993 - 99; PsycINFO 1984 - 99. Unless other year ranges are specified on the table.

Other searching:

Reference lists at the end of review articles of particular relevance were hand searched.
Relevant articles were solicited from expert colleagues and organisations.
Local and international clinical practice guidelines were reviewed for relevant references

Abbreviations:

The database searched has been indicated next to each set of keywords using the following abbreviations. M= Medline, EM= Embase, CO= Cochrane and CI= CINAHL. All EMBASE and Medline searches were done using english language En.La and human as a limit. Other abbreviations used were NIDDM= non-insulin-dependent diabetes mellitus; di=diagnosis; pc= prevention and control; ep= epidemiology; DM= Diabetes Melitus; BW= body weight; IGT= impaired glucose tolerance; Type 2= Type 2 diabetes; PSS= Psychosomatic Stress; St= Stress; Dp= depression; Sm&RP= Smoking and related phenomena; Sm= Smoking; Ex= Exercise; PA= physical activity; BIA= Bioelectrical impedance; DEXA= dual energy X-ray; Wt= weight; Edu= education; Comp= Composition; Gp=group; Gps= groups; WHR= waist to hip ratio; Dist= distribution; sf= skinfolds; ad= adipose; ob= obesity; BMI= body mass index; Thpy= therapy

Identified = number of articles which matched the mesh terms listed or contained the text terms in each particular database

Relevant = those articles considered relevant to the questions being asked after viewing titles or abstracts

Articles identified by other strategies = articles identified by hand searching, other searches for other questions, or from colleagues

Total for Review = Those articles which were relevant to the question, contained original data or were systematic reviews of original articles and met the following criteria.

Criteria used to determine the suitability of articles for review

In assessing the evidence the following criteria were used to determine the suitability of studies:

1. **Papers or editorials that present original data**
2. Papers that examine parameters in relation to cardiovascular disease or mortality risk and the risk of Type 2 diabetes
3. Papers that examine a parameter in relation to development of the metabolic syndrome, and impaired glucose tolerance and Type 2 diabetes
4. Studies of longitudinal design, however in some areas where there are few of these studies the cross-sectional studies were also assessed.
5. Population studies in populations similar to the population of Australia eg: small studies on American Indians or Japanese may not have been included.
6. The paper was published in the English Language.
7. Articles based on human studies
8. Articles were obtained from journals able to be accessed within our Library network or ordered through an interlibrary loan.

| QUESTIONS | | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | Total No. Reviewed and Graded | HIGHEST LEVEL OF EVIDENCE |
|-----------|---|--|---|----------------------|---|------------------|----------|-----------|-----------|----------|-------------------------------|---------------------------|
| 1. | <u>Does obesity in adult life and/or in childhood increase the risk of Type 2 diabetes?</u> | <p>Total for Question</p> <p>(DM/ OR DM.mp OR NIDDM/OR NIDDM.mp) AND (ob/ OR ob.mp OR BW/ OR BW.mp OR wt loss/) AND (risk/ OR risk.mp OR risk factors/ OR prediabetic state/ OR risk assessment/) limit adult M</p> <p>(DM/ OR DM.mp OR NIDDM/ OR NIDDM.mp) AND (ob/ OR ob.mp OR BW/ OR BW.mp) AND (risk/ OR risk.mp) AND limit child M</p> <p>(DM/ OR DM.mpOR NIDDM/ OR NIDDM.mp AND (ob/ OR ob.mp/ OR BW/ OR BW.mp) AND (child/ OR child.mp OR childhood/) M</p> <p>(NIDDM/ OR Type 2.mp) AND (ob/ OR ob.mp) AND risk.mp AND child.mp CI</p> <p>(NIDDM/ OR Type 2.mp) AND (ob/ OR ob.mp) AND risk.mp CI</p> <p>(NIDDM/ OR Type 2.mp) AND (risk/ OR risk.mp) AND (ob/ OR ob.mp) EM 88-99</p> <p>(ob.mp AND risk.mp AND diabetes.mpCO</p> | <p>3289</p> <p>1351</p> <p>370</p> <p>872</p> <p>1</p> <p>52</p> <p>567</p> <p>76</p> | 51 | 0 | 29 | 0 | 25 | 3 | 1 | 17 | II |

| QUESTIONS | | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE | |
|---|--|---|-------------------------|----------------------|---|------------------|----------|-----------|-----------|----------|----------------------------|---------------------------|--|
| 2 | <u>Is the pattern of fat distribution an additional contributor to total adiposity in determining the risk of Type 2 Diabetes and does this vary with ethnicity?</u> | Total For Question | 1631 | 44 | 1 | 22 | 0 | 16 | 6 | 0 | 22 | II | |
| | | (DM/ OR DM.mp OR NIDDM/ OR NIDDM.mp) AND (ad tissue/ OR ad tissue.mp OR body Comp/ OR body Comp.mp) AND (risk/ OR risk.mp OR ethnic Gps/ OR ethnic Gp.mp OR Asian/) M | 245 | | | | | | | | | | |
| | | (DM/ OR DM.mp OR NIDDM/ OR NIDDM.mp) AND (waist.mp OR hip/ OR hip.mp OR ad tissue.mp) AND (risk/ OR risk.mp) M | 255 | | | | | | | | | | |
| | | (DM/ OR DM.mp OR NIDDM/ OR NIDDM.mp) AND (fats/ OR fat.mp OR body.mp) AND (risk/ OR risk.mp) AND (supply and Dist/ OR Dist.mp) M | 206 | | | | | | | | | | |
| | | (NIDDM/ OR Type 2.mp OR NIDDM.mp) AND (ad tissue/ OR ad tissue..mp OR body Comp/ OR body Comp.mp OR body constitution/ OR body constitution.mp Or fat Dist.mp OR risk factors/ OR body fat.mp OR adiposity.mp) AND measure..mp M Complete | 86 | | | | | | | | | | |
| (NIDDM/ OR Type 2.mp) AND (Body fat/ OR fat Dist.mp) AND adiposity.mp AND (risk/ OR risk.mp) EM 94-99 | 17 | | | | | | | | | | | | |

| QUESTIONS | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE |
|---|---|-------------------------|----------------------|---|------------------|---------|----------|-----------|----------|----------------------------|---------------------------|
| 2. <u>Is the pattern of fat distribution an additional contributor to total adiposity in determining the risk of Type 2 Diabetes and does this vary with ethnicity?</u> | (NIDDM/ OR Type 2.mp) AND (risk/ OR risk.mp) AND (ob/ OR ob.mp) AND (ethnic Gp/ OR ethnic.mp) EM 94-99 | 33 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp) AND (risk/ OR risk.mp) AND (ob/ OR ob.mp) AND EM 88-93 | 155 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp) AND (body fat/ OR fat Dist.mp OR adiposity.mp OR ob/) AND (risk/ OR risk.mp) EM 88-93 | 125 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp) AND (risk/ OR risk.mp) AND (ethnic difference/ OR ethnic.mp) AND (ob/ OR ob.mp) EM 88-93 | 12 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp) AND (ob/ OR ob.mp) AND asians/OR ethnic Gps/ OR hispanics/ OR minority Gps/ OR ethnic.mp) CI | 37 | | | | | | | | | |
| | (Anthropometry.mp OR sf thickness/ OR WHR.mp OR ad tissue Dist/ OR sf.mp OR BMI/) AND (measures.mp OR body Wts and measures/) AND (Ad tissue/ OR body Comp/ OR adiposity.mp OR body constitution OR fat Dist.mp) CI | 161 | | | | | | | | | |

| QUESTIONS | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE |
|-----------|--|---|----------------------|---|------------------|---------|----------|-----------|----------|----------------------------|---------------------------|
| 2. | <u>Is the pattern of fat distribution an additional contributor to total adiposity in determining the risk of Type 2 Diabetes and does this vary with ethnicity?</u> | <p>(body Wts and measures/ OR measures.mp) AND (Ad tissue/ OR body Comp/ adiposity.mp OR body constitution/ OR fat Dist.mp) AND (tomography, X-ray computed/ OR computed tomography.mp OR electric impedance/ OR BIA.mp OR DEXA.mp) CI</p> <p>Body.mp AND fat.mp AND risk.mp AND diabetes.mp CO</p> <p>[(Fat.mp AND body.mp) OR (body.mp AND comp.mp) OR (body.mp AND constitution.mp) OR (body.mp AND patterning.mp)] AND anthropometry.mp CO</p> <p>Ethnicity.mp AND diabetes.mp AND risk.mp CO</p> <p>[Body comp/OR (Body.mp AND comp.mp) OR body constitution/ OR (body AND constitution)OR body patterning/ OR (body.mp AND patterning.mp) OR fat body/ OR (fat AND body)] AND [body-weights-and-measures/ OR (body.mp AND weights.mp AND measures.mp)] CO</p> | 97 | | | | | | | | |
| | | 20 | | | | | | | | | |
| | | 161 | | | | | | | | | |
| | | 8 | | | | | | | | | |
| | | 13 | | | | | | | | | |

| QUESTIONS | | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE | |
|-----------|--|---|-------------------------|----------------------|---|------------------|----------|-----------|-----------|----------|----------------------------|---------------------------|--|
| 3 | <u>What simple measures can be used for body fat distribution?</u> | Total For Question | 926 | 19 | 0 | 18 | 0 | 12 | 5 | 0 | 17 | II | |
| | | (DM/ OR NIDDM/ OR NIDDM.mp OR DM.mp) AND body.mp AND (fat/ OR ob/ OR adiposity.mp OR BW/) AND (waist.mp OR hip/ OR hip.mp) M | 245 | | | | | | | | | | |
| | | (DM/ OR DM.mp OR NIDDM/) AND (body Comp/ OR body Comp.mp M | 112 | | | | | | | | | | |
| | | (Ad tissue/ OR body comp/ OR body constitution/) AND weights and measures/ AND (BMI/ OR Anthropometry/ OR sf thickness/ OR WHR.mp) M | 0 | | | | | | | | | | |
| | | (Ad tissue/ OR body comp/ OR body constitution/) AND weights and measures/ AND (electric impedance/ tomography, OR x-ray computed/ OR dual energy x-ray.mp) M | 0 | | | | | | | | | | |
| | | (NIDDM/ OR Type 2.mp) AND (ad tissue OR body Comp OR fat DistOR body Wts) AND (measuresOR skinfold thickness OR sf OR WHR OR DEXA OR computed tomography) CI | 43 | | | | | | | | | | |

| QUESTIONS | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE |
|---|--|-------------------------|----------------------|---|------------------|---------|----------|-----------|----------|----------------------------|---------------------------|
| 3. <u>What simple measures can be used for body fat distribution?</u> | (Anthropometry.mp OR sf thickness/ OR sf.mp OR ad tissue Dist/ OR WHR.mp) AND (body Wts and measures/ OR measures.mp) AND (Ad tissue/ OR body Comp/ OR adiposity.mp OR body constitution fat Dist.mp) CI | 132 | | | | | | | | | |
| | (body Wts and measures/ OR measures.mp) AND (Ad tissue/ OR body Comp/ adiposity.mp OR body constitution/ OR fat Dist.mp) AND (electric impedance/ OR tomography, Xray computed/ computed tomography.mp OR BIA.mp/ OR DEXA.mp) CI | 97 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp) AND (Body fat/ OR fat Dist.mp) AND adiposity.mp AND (risk/ OR risk.mp) EM 94-Feb 99 | 17 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp) AND (risk/ OR risk.mp) AND (ob/ OR ob.mp) EM 88-93 | 155 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp) AND (body fat/ OR fat Dist.mp OR adiposity.mp OR ob/) AND (risk/ OR risk.mp) EM 88-93 | 125 | | | | | | | | | |
| Body AND fat AND risk AND diabetes CO | 0 | | | | | | | | | | |

| QUESTIONS | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE |
|-----------|---|-----------------------------------|----------------------|---|------------------|----------|----------|-----------|----------|----------------------------|---------------------------|
| 4. | <u>Does physical activity decrease, and a sedentary life-style increase, the risk of Type 2 diabetes?</u> | Total For Question 1470 | 34 | 2 | 14 | 0 | 6 | 8 | 2 | 14 | II |
| | (DM/ OR DM.mp OR NIDDM/ OR diabetes.mp) AND (Ex/ OR Ex.mp OR sedentary/ OR PA.mp) AND (risk/ OR risk.mp) M | 923 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp) AND (Ex/ OR Ex.mp OR therapeutic Ex/ OR sedentary.mp OR PA) AND (risk.mp OR prevent.mp OR weights and measure/ OR data collection methods/) Sept CI | 52 | | | | | | | | | |
| | (PA.mp OR PA/) AND estimate.mp) EM 88-99 | 299 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp) AND (risk/ OR risk.mp) AND (Ex.mp OR Ex/) EM 88-99 | 104 | | | | | | | | | |
| | (Diabetes.mp OR DM/) AND [(physical.mp AND activity.mp) OR physical fitness/ OR ex Thy/ OR ex/]AND (risk.mp OR risk/ OR risk factors/ OR risk assessment/) CO | 32 | | | | | | | | | |
| | DM/ AND (risk/ OR risk factors/ OR risk assessment/) CO | 49 | | | | | | | | | |

| QUESTIONS | | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE |
|-----------|---|--|-------------------------|----------------------|---|------------------|---------|----------|-----------|----------|----------------------------|---------------------------|
| 4. | <u>Does physical activity decrease, and a sedentary life-style increase, the risk of Type 2 diabetes?</u> | [Physical fitness/ OR (physical.mp AND fitness.mp) OR ex Thpy/ OR (ex.mp and Thpy.mp) OR Ex/ OR Ex.mp] AND [body wts and measures/ OR (body.mp and weights.mp and measures.mp) OR weights and measures/OR [weights.mp and measures.mp]] CO | 11 | | | | | | | | | |

| QUESTIONS | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE | |
|-----------|---|--|--|---|------------------|-----------|----------|-----------|-----------|----------------------------|---------------------------|-----------|
| 5. | <u>What simple measures can be used to determine physical activity and sedentariness?</u> | Total For Question DM/ OR DM.mp OR NIDDM/ AND (DM/ OR DM.mp) (Ex/ OR Ex.mp OR PA.mp) AND (Wts and measures/ OR measure.mp) M NIDDM/ OR Type 2.mp) AND (risk.mp OR prevent.mp) AND (Ex/ OR Ex.mp PA./ OR therapeutic Ex/) CI (NIDDM/ OR Type 2.mp) AND (Ex/ OR sedentary.mp) AND risk.mp CI (PA.MPOR PA.mp) AND estimate.mp EM 88-99 (NIDDM/ OR Type 2.mp) AND (risk/ OR risk.mp) AND (Ex.mp OR Ex/) EM 88-93 Diabetes.mp AND physical.mp AND activity.mp AND risk.mp CO | 610 129 27 13 390 28 23 | 26 | 1 | 19 | 0 | 3 | 16 | 0 | 19 | II |

| QUESTIONS | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE |
|---|--|-------------------------|----------------------|---|------------------|---------|----------|-----------|----------|----------------------------|---------------------------|
| 6. <u>Do high-fat diets increase the risk of Type 2 diabetes and/or Gestational Diabetes Mellitus and how is this affected by fat type?</u> | Total For Question | 622 | 14 | 0 | 11 | 0 | 9 | 2 | 0 | 11 | II |
| | DM/ OR DM.mp OR NIDDM/ AND (DM/ OR diabetes.mp) AND (dietary fats/ OR fats/ OR dietary fat.mp OR fat.mp) AND intake/ AND (risk/ OR risk.mp M | 165 | | | | | | | | | |
| | Diabetes, gestational/ OR gestational diabetes.mp) AND (fats/ OR fat/ OR fat.mp) AND (diet/ OR diet.mp) M | 13 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp OR DM, gestational/ OR gestational diabetes.mp) AND (dietary fats OR fat.mp) AND diabetic diet/ OR diet/ OR diet, fat restricted/ OR diet, reducing/ OR diet.mp) AND risk.mp CI | 14 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp) AND (risk OR risk.mp) AND (dietary fat/ OR fat intake/) EM 88-99 | 54 | | | | | | | | | |
| | (pregnancy DM/ OR gestational diabetes.mp) AND (risk OR risk.mp) AND (dietary fat.mp OR fat intake/) EM 88-99 | 3 | | | | | | | | | |
| | (NIDDM/ OR Type 2.mp) AND (risk OR risk.mp) AND (fat/ OR fat.mp) EM 88-99 | 251 | | | | | | | | | |
| Diet AND Diabetes AND Risk CO | 122 | | | | | | | | | | |

| QUESTIONS | | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE |
|-----------|---|--|---|----------------------|---|------------------|----------|-----------|-----------|----------|----------------------------|---------------------------|
| 7. | <u>Does Gestational Diabetes Mellitus increase the subsequent risk of Type 2 diabetes in mothers and/or their infants ?</u> | <p>Total For Question</p> <p>(Diabetes, gestational/ OR gestational diabetes.mp OR GDM.mp) AND (DM/ OR DM.mp OR NIDDM) AND (risk/ OR risk.mp) M</p> <p>NIDDM/ AND (birth Wt/ birthWt.mp OR fetal macrosomia/) M Complete</p> <p>NIDDM/ AND (birth Wt/ birthWt.mp OR fetal macrosomia/) AND risk factors/ M Complete</p> <p>(NIDDM/ OR Type 2.mp) AND (gestational diabetes.mp OR DM, gestational/) AND (risk.mp OR high-risk pregnancy/) CI</p> <p>NIDDM/ AND Risk factors/ AND gestational.mp. CI</p> <p>NIDDM/ pc,di and ep and gestational diabetes/ CI</p> <p>(pregnancy DM/ OR gestational diabetes.mp) AND (NIDDM/ OR Type 2.mp) AND (risk/ OR risk.mp) EM 88-99</p> <p>{ gestational diabetes/OR (gestational.mp and diabetes.mp)] AND [NIDDM/ OR NIDDM.mp] CO</p> | <p>629</p> <p>414</p> <p>73</p> <p>20</p> <p>19</p> <p>11</p> <p>10</p> <p>77</p> <p>5</p> | 31 | 0 | 18 | 0 | 10 | 8 | 0 | 18 | II |

| QUESTIONS | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE | |
|-----------|---|---|----------------------|---|------------------|-----------|----------|-----------|----------|----------------------------|---------------------------|-----------|
| 8. | <u>How does weight at birth affect later risk of Type 2 diabetes?</u> | <u>Total For Review</u> | 428 | 22 | 0 | 18 | 0 | 18 | 0 | 0 | 18 | II |
| | | (DM/ OR DM.mp OR NIDDM.mp) AND (DM/ OR diabetes.mp) AND (birth Wt/ OR birth Wt.mp OR birthWt.mp) AND (risk/ OR risk.mp) M | 262 | | | | | | | | | |
| | | NIDDM/ AND (birth wt/ OR birthwt.mp OR fetal macrosomia/) M Complete | 73 | | | | | | | | | |
| | | (NIDDM/ OR non-insulin-dependent diabetes.mp OR Type 2.mp) AND (birth Wt.mp OR birth Wt/) CI | 3 | | | | | | | | | |
| | | DM/ AND (Birth Wt/ OR birth Wt.mp OR macrosomia/) EM 88-93 | 68 | | | | | | | | | |
| | | DM/ pc, di, ep. AND (birthWt.mp OR birth Wt/ OR macrosomia/) EM 94-99 | 22 | | | | | | | | | |
| | | Type.mp AND 2.mp AND Diabetes.mp AND Birth.mp AND Wt.mp CO | 0 | | | | | | | | | |
| | | NIDDM.mp AND (birth.mp AND Wt.mp) OR birthWt.mp OR macrosomia.mp CO | 0 | | | | | | | | | |

| QUESTIONS | | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE | |
|-----------|---|---|-------------------------|----------------------|---|------------------|----------|----------|-----------|----------|----------------------------|---------------------------|--|
| 9. | <u>Do low glycaemic index diets decrease the risk of Type 2 diabetes?</u> | Total For Question | 531 | 18 | 0 | 2 | 0 | 2 | 0 | 0 | 2 | II | |
| | | (DM/ OR DM.mp OR NIDDM.mp) AND (DM/ OR diabetes.mp) AND (glycaemic/ OR abstracting and indexing OR index.mp OR glyceic.mp) AND (risk/ OR risk.mp) M | 124 | | | | | | | | | | |
| | | (DM/ OR DM.mp OR NIDDM.mp) AND DM/ OR diabetes.mp AND dietary carbohydrates/ OR dietary carbohydrate.mp AND (risk/ OR risk.mp) M | 44 | | | | | | | | | | |
| | | (NIDDM/ OR Type 2.mp) AND (glycaemic index.mp OR glyceic index.mp) AND (risk/ OR risk.mp) CI | 1 | | | | | | | | | | |
| | | (NIDDM/ OR Type 2.mp) AND (glycaemic index.mp OR glyceic index.mp OR glyceic.mp OR glycaemic.mp) AND (risk/ OR risk.mp) EM 88-93 | 62 | | | | | | | | | | |
| | | NIDDM/ OR Type 2.mp AND (risk/ OR risk.mp) AND (Glycaemic index.mp OR glyceic) EM 94-99 | 148 | | | | | | | | | | |
| | | Diabetes.mp AND diet,mp AND risk.mp CO | 152 | | | | | | | | | | |

| QUESTIONS | | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE | |
|-----------|---|--|-------------------------|----------------------|---|------------------|----------|----------|-----------|----------|----------------------------|---------------------------|--|
| 10. | <u>Will psychosocial stress increase the risk of Type 2 diabetes?</u> | Total For Question | 2586 | 32 | 0 | 15 | 0 | 9 | 6 | 0 | 15 | II | |
| | | (DM/ OR DM.mp OR NIDDM) AND (DM/ OR diabetes.mp) AND (Sm/ OR Sm.mp) AND (risk/ OR risk.mp) M | 1517 | | | | | | | | | | |
| | | (DM/ OR DM.mp OR NIDDM/) AND (DM/ OR diabetes.mp AND (psychosocial AND St/OR St.mpOR Dp/OR Dp.mp OR anxiety/ OR anxiety.mp) AND (risk/ OR risk.mp) M | 128 | | | | | | | | | | |
| | | NIDDM/ OR Type 2.mp) AND (St, psychological/ OR psychological St.mp OR Dp/ OR Dp.mp) CI | 25 | | | | | | | | | | |
| | | NIDDM/ AND Smoking.mp CI | 36 | | | | | | | | | | |
| | | (NIDDM/ OR Type 2.mp OR IGT/ OR IGT.mp OR 'IGT'.mp) AND (emotional St OR psychological aspect OR PSS OR St OR cigarette Sm OR Dp) EM 94-99 | 75 | | | | | | | | | | |

| QUESTIONS | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE | |
|-----------|--|---|----------------------|---|------------------|---------|----------|-----------|----------|----------------------------|---------------------------|--|
| 10. | <u>Will psychosomatic stress increase the risk of Type 2 diabetes?</u> | (St/ OR PSS.mp OR emotional St/ OR mental St/ OR St.mp OR Dp/ OR Dp.mp OR cigarette Sm/ OR 'Sm&RP'/ OR Sm/ OR Sm.mp) AND IGT.mp OR IGT/) EM 88-93 | 15 | | | | | | | | | |
| | | (NIDDM/ OR Type 2.mp OR NIDDM.mp) AND (emotional St/ OR PSS.mp OR mental St/ OR St/ OR St.mp OR cigarette Sm/ OR Sm/ OR Sm.mp OR 'Sm and Sm related phenomena'/ OR Dp/ OR Dp.mp) EM 88-93 | 155 | | | | | | | | | |
| | | (NIDDM/ OR Type 2.mp OR NIDDM.mp) AND (emotional St/ OR psychological aspect/ OR PSS.mp OR St/ OR cigarette Sm/ OR Sm/ OR Sm.mp OR Dp/ OR Dp.mp) EM 94-99 | 403 | | | | | | | | | |

| QUESTIONS | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE |
|--|---|-------------------------|----------------------|---|------------------|---------|----------|-----------|----------|----------------------------|---------------------------|
| 10. <u>Will psychosomatic stress increase the risk of Type 2 diabetes?</u> | (DM/ OR Type 2.mp OR NIDDM.mp) AND (psychosomatic disorders/ OR PSS.mp OR tobacco Sm OR Sm.mp OR social St/ OR psychological St OR OR St/ OR St.mp PI | 137 | | | | | | | | | |
| | NIDDM.mp AND St.mp AND pyschosomatic St.mp AND Sm.mp CO | 6 | | | | | | | | | |
| | NIDDM.mp AND St CO | 8 | | | | | | | | | |
| | NIDDM.mp AND psychosomatic.mp AND St.mp CO | 0 | | | | | | | | | |
| | Type.mp AND 2.mp AND stress.mp AND Diabetes.mp CO | 0 | | | | | | | | | |
| | NIDDM/ AND smoking/ CO | 9 | | | | | | | | | |
| | NIDDM/ AND (St/ OR St-psychological/ OR Sm/ OR Dp/ OR Anxiety/) CO | 17 | | | | | | | | | |
| | NIDDM.mp AND (St/ OR [St.mp-AND psychological.mp] OR Sm.mp OR Dp.mp OR Anxiety.mp) CO | 55 | | | | | | | | | |

| QUESTIONS | | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE | |
|-----------|--|--|-------------------------|----------------------|---|------------------|----------|-----------|-----------|----------|----------------------------|---------------------------|--|
| 11. | <u>What forms of diet and physical activity are recommended to prevent excessive weight gain in childhood and throughout adult life?</u> | Total For Question | 2834 | 80 | 5 | 45 | 9 | 21 | 12 | 3 | 45 | I | |
| | | (DM/ OR DM.mp OR NIDDM) AND diabetes.mp AND (ob/ OR ob.mp OR Ex/ OR Ex.mp) AND (risk/ OR risk.mp AND Limit (En.La AND human) M | 1614 | | | | | | | | | | |
| | | (DM/ OR DM.mp OR NIDDM) AND (DM/ OR diabetes.mp AND Edu.mp OR Edu/ OR health Edu/OR health Edu.mp) AND (risk/ OR risk.mp AND (En.La AND human) M | 489 | | | | | | | | | | |
| | | (Wt gain/ OR Wt gain.mp OR Wt loss/ OR Wt loss.mp) AND (diet/ OR diet, fat – restricted/ OR diet.mp OR Ex/ OR Ex.mp) AND (risk.mp OR prevent.mp) CI | 148 | | | | | | | | | | |
| | | (Wt gain/ OR Wt gain.mp OR ob/ OR ob, morbid/ OR ob.mp) AND (diet/ OR diet, fat –restricted/ OR diet, reducing/ OR restricted diet/ OR diet.mp OR Ex/ OR Ex.mp OR PA.mp OR PA.mp) AND (Edu/ OR health Edu/ OR Nutrition Edu/ OR patient Edu/ OR Edu.mp) AND (risk.mp OR prevent.mp) CI | 49 | | | | | | | | | | |

| QUESTIONS | KEY WORDS | NO. ARTICLES IDENTIFIED | NO RELEVANT ARTICLES | ARTICLES IDENTIFIED BY OTHER STRATEGIES | TOTAL FOR REVIEW | LEVEL I | LEVEL II | LEVEL III | LEVEL IV | TOTAL NO REVIEWED & GRADED | HIGHEST LEVEL OF EVIDENCE |
|--|---|-------------------------|----------------------|---|------------------|---------|----------|-----------|----------|----------------------------|---------------------------|
| 11. <u>What forms of diet and physical activity are recommended to prevent excessive weight gain in childhood and throughout adult life?</u> | (diabetic diet/ OR diet/ OR diet.mp OR PA.mp OR PA/ Ex.mp OR Ex/) AND (Wt gain/ OR Wt gain.mp OR morbid ob/ OR ob/ OR ob.mp) AND prevent.mp EM 88-99 | 257 | | | | | | | | | |
| | (Primary and Prevention/ OR Risk/ OR Risk-assessment/ OR risk-factors/ OR risk-managemnet/) AND (Obesity/ OR obesity-in-diabetes/ OR obesity-morbid/ OR weight-gain/ OR weight-loss/) CO | 114 | | | | | | | | | |
| | (Physical-fitness/ OR Ex-Thpy/ OR Ex/ OR diabetic-diet/ OR diet/ OR diet-Thpy/ OR diet-fat-restricted/ OR diet-reducing/) AND (obesity/ OR obesity-in-diabetes/ OR obesity-morbid/ OR weight-gain/ OR weight-loss/) limit review CO | 11 | | | | | | | | | |
| | Diabetes.mp AND diet,mp AND risk.mp CO | 152 | | | | | | | | | |