The Summer Meeting of the Nutrition Society jointly with the Association for the Study of Obesity was held at Trinity College, Dublin, Republic of Ireland on 5–8 July 2004

The Boyd Orr Lecture

Cost-effective measures to prevent obesity: epidemiological basis and appropriate target groups

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Cost-effective prevention strategies to prevent weight gain and the development of obesity should be based on appropriate knowledge of the determinants of weight gain. The body of evidence on the dietary determinants of weight gain is, however, fragmentary at best, partly because inappropriate research methods are used to study the determinants of obesity under normal circumstances. Evidence from studies using experimental diets have shortcomings because of their short duration and selection of highly-motivated subjects and because the outcomes can be easily influenced by the choice of foods to be used in the intervention. Of the observational studies, many have severe methodological shortcomings, e.g. ecological studies, cross-sectional surveys and classical cohort studies in which the baseline diet is linked to subsequent weight development over long periods of time. Longitudinal studies with repeated measurements in which changes in diet and physical activity are linked to changes in weight are probably the most informative, but these studies are relatively rare. There is a great interest in interventions that are effective and efficient for the prevention of obesity. Many countries and research funding agencies seem to show a strong tendency to develop interventions for children and adolescents exclusively. It can be easily shown, however, that intervention programmes are much more likely to be cost-effective in older adults than in children, which indicates that adults should not be neglected as target populations for obesity prevention. Obesity prevention should follow a life-course approach, as currently recommended for non-communicable diseases in general by the WHO.

Obesity: Overweight: Prevention: Cost-effectiveness: Epidemiology

Recently, there has been a sharp increase in the interest in obesity as a public health problem. In particular, several reports from the World Health Organization (2000, 2003) have been instrumental in putting obesity on the agenda of ministries of health in developed and developing countries as a major concern in their nation's health.

The WHO Technical Report, *Diet, Nutrition and the Prevention of Chronic Diseases* summarises current knowledge of the main determinants of obesity (World Health Organization, 2003). The committee has agreed (World Health Organization, 2003; Swinburn *et al.* 2004) that there is convincing evidence that sedentary lifestyles and high intakes of energy-dense micronutrient-poor foods increase the risk of obesity and that regular physical activity and a high intake of dietary fibre decrease the risk of obesity.

Furthermore, they have stated that based on the current evidence it is probable that intense marketing of energy-dense foods and fast-food outlets, high intakes of sugar-sweetened soft drinks and fruit juices, and adverse socio-economic conditions are increasing the risk of obesity, while breast-feeding and home and school environments that support healthy food choices for children decrease the risk of obesity (Swinburn *et al.* 2004). Many of the factors

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that are considered to be in the category 'probable' are termed 'associated evidence and expert opinion', which means they are based on a common sense understanding of the factors that promote overeating. Following the draft release of the report, as well as during the process of adopting the WHO global strategy on diet, physical activity and health, there has been much disagreement, particularly about the level of scientific evidence for the factors that have been categorised as 'probable'. This controversy is not surprising because for many of these factors it is very likely that there will never be a randomised controlled trial with a sufficient extent of intervention and duration to establish the magnitude and direction of the associations between the factors and the development of obesity. As in most issues related to nutrition and chronic diseases it is unlikely that there will be experimental evidence with firm end points. The existing experimental evidence, however, also shows that the current study designs may not lead to the irrefutable evidence usually expected from randomised controlled trials. A good example may be the continuing debate as to whether or not low-fat diets are beneficial for the prevention of weight gain and incidence of overweight and obesity (Bray & Popkin, 1998; Willett, 1998). Low-fat diets can resemble a vegan diet or one based primarily on soft drinks and low-fat cookies and potato crisps. Selection of the foods in the experimental diets, the subjects (age, sex, ethnicity, extent of overweight or insulin resistance), the duration of the trial, the extent of adherence of the subjects all seem to have a considerable impact on the outcome of the study. For example, the CARMEN project in which a high-fat diet was compared with two high-carbohydrate diets (high in sugar or high in complex carbohydrate) has been interpreted as showing either that diets high in sugar are appropriate for weight control (Saris *et al.* 2000) or the opposite (Mann, 2004), depending on the selection of subpopulations and the interpretation of statistical power.

As obesity is a problem that usually develops slowly over many years or decades quite heavy reliance will have to be placed on observational studies. The following section will consider the limitations and challenges of observational studies.

Dietary determinants of obesity: the limits of epidemiology

There are several main types of observational studies. One is an ecological design in which diet and measures of fatness are analysed in countries that vary in dietary patterns as well as in the prevalence of overweight or obesity. One early example of such studies is the Seven Countries Study initiated by Ancel Keys in the late 1950s. Recent analyses using chemical analyses of usual diets of the participating middle-aged male subjects have shown that at the time of the study the variation in levels of fatness (skinfolds) were mainly determined by the fibre content of the diet, as well as the extent of physical activity at work (Kromhout *et al.* 2001). A recent example of such a study is the WHO MONICA project, which involved many more participating centres, included both men and women and used food energy supply data derived from the

FAO food balance sheet (Silventoinen *et al.* 2004). This study has shown that the prevalence of obesity is associated with a higher energy supply. Such ecological studies have the merit of encompassing the impressive variations in diets, physical activity and levels of obesity in populations. However, there are many potential confounders, and also obesity and diets are assessed simultaneously, which generally ensures that these studies are usually not considered to be very convincing in terms of evidence for causal relationships between diet and obesity.

Another often-used design is cross-sectional analyses in single populations. These studies have the same limitations as the ecological studies but without the potential advantage of large variations in diets and prevalences of obesity. A review of the literature has shown that no consistent association can be identified between BMI and obesity and food intake patterns in cross-sectional studies (Togo *et al.* 2001), which probably suggests that this type of study is not very informative about possible determinants of obesity.

Usually, the epidemiological studies that rate highest in terms of scientific evidence are prospective cohort studies in which diets are assessed first and subsequently subjects are followed until they develop a particular disease during a given period. This approach fulfills one important criterion for causality, i.e. a temporal relationship between the exposure and the health outcome. Particularly for diseases that develop many years subsequent to earlier behaviour (e.g. smoking and lung cancer), this design is very powerful. For some determinants of obesity this type of study has also been shown to be less biased than cross-sectional studies. For example, in cases in which the measures of exposure are greatly influenced by the expanding fat mass, such as levels of leptin and BMR, a prospective study is more informative than other designs. Prospective studies have shown that relatively low BMR and low leptin levels are determinants of weight gain, while in cross-sectional studies it has been found that obese subjects have higher levels of leptin and higher BMR than lean subjects (Ravussin & Gautier, 1999). In the study of dietary determinants of weight gain this approach may be less useful because weight gain usually develops as a result of changes in energy balance. Baseline assessments of diets are then only informative when they predict future relative overeating and only if food habits are stable. In many periods in life when average weights are increasing, diets are not stable and patterns of physical activity may change over time. These changes may lead to biased estimates of diet and physical activity and later health outcomes. A recent study of Norwegians (Andersen, 2004) has shown that when intra-individual changes in physical activity during follow-up are taken into account the relative risk of mortality associated with baseline physical inactivity increases by 24–59 %.

Examples of periods in life during which potentially-important lifestyle changes occur in adults are the periods between 20 and 30 years of age when most individuals settle down, buy a car, get sedentary jobs, commute long distances, start a family etc. For many individuals these changes mean a drastic change in dietary habits and the way they spend their time. Other examples are pregnancy

and lactation in women, ceasing to smoke and retirement. In all these periods changes in behaviour are followed by weight change. In these cases baseline behaviour may not be very informative, it is the changes in behaviour that determine weight gain. There are also several other considerations. One is that the under-reporting of dietary intakes usually increases with increasing body weight (Heitmann & Lissner, 1995; Braam *et al.* 1998). If under-reporting occurs, evaluation of changes in diet based on repeated measures of diet may be less susceptible to this kind of bias. Finally, it is usually found at a population level that a good predictor of weight gain is weight loss in the preceding period (and vice versa). Thus, baseline dietary habits may be assessed at the end of a period of negative energy balance. In a study of women aged 30–55 years Colditz *et al.* (1990) have found that age, relative weight and previous weight change are far stronger predictors of weight change than qualitative aspects of diet. It has been shown (AJ Nooyens, TLS Visscher, AJ Schuit, WMM Verschuren and JC Seidell, unpublished results) that dietary habits, which at baseline predict weight gain, may not be predictive of weight gain when changes in that behaviour are evaluated. In some cases the association may actually be in the opposite direction.

All these issues make it very likely that analyses of longitudinal changes in diet and physical activity may lead to important insights into the modifiable determinants of major weight gain and the development of overweight and obesity. Yet, such studies are quite rare.

Most investigators have studied the relationship between baseline dietary behaviour and subsequent weight gain (Colditz *et al.* 1990; Rissanen *et al.* 1991; Parker *et al.* 1997; Stamler & Dolewcek, 1997; Coakley *et al.* 1998; Ludwig *et al.* 1999; Ball *et al.* 2002; Schulz *et al.* 2002; van Rossum *et al.* 2002; Newby *et al.* 2003). In only a handful of studies have the relationships between weight change and changes in diet been reported (Klesges *et al.* 1992; French *et al.* 1994; Sherwood *et al.* 2000; Drapeau *et al.* 2004). All these studies have failed to include changes in physical activity. As weight change is a result of a change in energy balance, changes in diet can only be interpreted on the assumption that energy expenditure remains unchanged.

It is very likely that the relationships between changes in diet and physical activity and weight gain may differ with the extent of obesity, age, sex, social class and ethnicity, and there may be a need to extend these data considerably in the future. A quick preview of the literature indicates that there actually may not be a solid basis for understanding the dietary changes that lead to weight gain and the development of obesity. If there is no critical assessment of modifiable determinants of weight gain, how can effective strategies for the prevention of overweight and obesity be developed other than by trial and error? In particular, when the requirement is for particular strategies that involve measures to be taken by governments and industry, it may be necessary to be more persuasive than merely advocating a common sense approach. Observational evidence should not replace evidence-based prevention, but it should at least be its foundation.

Cost-effective prevention of obesity: is it ever too late to start?

With the increase in the prevalence of overweight and obesity in many societies and the realisation of the impact of this increase on the burden of disease, as well as on the economy, there is a growing interest in effective obesity prevention programmes. Particular attention is usually given to programmes that are aimed at prevention of overweight in children. This consideration is justifiable, but if the implication is that prevention programmes should be limited to children and adolescents only, it may be quite inappropriate. An attempt will be made to argue that cost-effectiveness actually continuously improves with age up to much older age-groups.

The reason why interventions in children are favoured is that behaviour in children may be changed more easily than behaviour in adults, that chronic disease has early origins and that these disease processes may be more effectively altered at relatively young ages. On the other hand, it may be argued that:

1. the sharpest increase in the incidence of obesity is in adulthood. For example, based on cross-sectional analyses, the prevalence of obesity in The Netherlands increases from 4·4% in 20–29-year-old men to 14·7% in 50–59-year-old men. In women these percentages increase from 5·4 to 17·0 respectively (Visscher *et al.* 2002). These values are underestimates of the age-related increase in obesity because they are based on cross-sectional data and do not include secular increases in BMI;
2. adults usually continue to gain weight during adulthood (particularly in young adulthood);
3. adult weight gain is (independently of the extent of overweight) a risk factor for many diseases such as heart disease (Willett *et al.* 1995), type 2 diabetes mellitus (Colditz *et al.* 1995) and breast cancer (Trentham-Dietz *et al.* 2000) and all-cause and CHD mortality (Peters *et al.* 1995);
4. although for many diseases the relative risks for disease associated with obesity decrease with age, the absolute risk and population-attributable risks for disease increase with age (Seidell & Visscher, 2000). The absolute risk is the age-specific incidence rate of diseases, and is calculated from the prevalence of obesity (which increases with age) and the relative risk. Clinical thinking is dominated by a focus on the relative risk (what does this factor do for the health of a particular patient), although this variable is increasingly combined with estimates of the absolute risk (van den Hoogen *et al.* 2000). In public health the focus is mainly on population-attributable risks (what proportion of new cases in the population can be attributed to the risk factor). Some public health-oriented studies, however, also focus on the relative risk. Gostynski *et al.* (2004), for example, has reported recently that the risk (odds ratio) of hypercholesterolaemia for obese subjects *v.* normal-weight subjects decreases with age from an odds ratio of 3·4 in men aged 25–29 years to an odds ratio of 1·7 in men aged

Table 1. Weight change and risk of type 2 diabetes mellitus in the Finnish Diabetes Prevention Study (adapted from Tuomilehto *et al.* 2001)

Weight change (%) from baseline to year 1			Odds ratio for diabetes mellitus	95% CI
Quintile	Mean	Range		
1	−11	−22 to −7	0·17	0·05, 0·50
2	−5	−7 to −3	0·39	0·17, 0·92
3	−2	−3 to −1	0·87	0·43, 1·76
4	0	−1 to +1	1 (reference)	
5	+3	+1 to +16	2·18	1·15, 4·15

55–59 years. They suggest that obesity prevention is of particular importance in young adults because 'the effects of obesity are strongest' in that age group. The prevalence of obesity, however, increases from 5% in the younger men to 18% in the older men. From this increase it can be calculated that the population-attributable risk is actually the same in both age-groups (11%). The prevalence of hypercholesterolaemia also increases with age from about 11% in the younger men to about 35% in the older men. This relationship means that obesity is responsible for more cases of hypercholesterolaemia in the older men than in the younger men. It illustrates that although the relative risk for disease may decrease with age, the contribution of obesity to ill health on a population level actually increases with age;

5. prevention of weight gain and weight loss in adults have been shown to be related to dramatically-reduced relative risks for, for example, type 2 diabetes mellitus in high-risk populations (see Table 1). In the Finnish Diabetes Prevention Study (Tuomilehto *et al.* 2001) a small weight gain (3%) was found to be associated with a doubling of the risk of type 2 diabetes in high-risk individuals (obese and with impaired glucose tolerance). Weight-gain prevention may be of particular importance in those who are already overweight, since the relationship between BMI and risk of type 2 diabetes increases exponentially (Colditz *et al.* 1995);

6. interventions in children and adolescents need to be maintained for many more years or decades in order to have a considerable effect on the number of new cases of type 2 diabetes mellitus and heart disease or cancer compared with interventions in older individuals;

7. if interventions for weight-gain prevention are directed at young and older adults by supporting healthier diets and physical activity it is likely that others members of the household will also be affected. Health promotion aimed at schools is much less likely to affect the parents.

It is increasingly clear that in older men and women it may be much more informative to study changes in waist circumference rather than changes in weight. 'Waist-gain prevention' may have important public health benefits even in individuals aged ≥70 years (Seidell & Visscher, 2000; Visscher *et al.* 2001).

All these consideration make it quite likely that weight (or waist) gain prevention in adults at least up to the age of 65 or 70 years is likely to be more effective and more efficient than overweight prevention in children and adolescents over a given period of, for example, 5–10 years. This conclusion does not imply that attention and money should not be directed to health promotion programmes in children and adolescents, but that the considerable health gains that can be obtained by weight-gain prevention in adults should not neglected.

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