The Interrelationship between Obesity, Physical Activity, Nutrition and other Determinates

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Abstract

Introduction: The prevalence of obesity has risen so rapidly in recent decades that the World Health Organisation has declared obesity a global epidemic. Changing physical activity and dietary habits are key to understanding obesity aetiology and are therefore are important components in any obesity prevention and treatment strategies. However the interrelationship that exists between obesity, diet, physical activity, and other determinates of obesity needs to be clarified. In 2009, the National Nutrition Surveillance Centre, in partnership with the Health Service Executive (HSE), as part of the HSE Framework for Action on Obesity conducted a review relating to this area.

Objective: The aim of this literature review was to explore key findings from national and international research relating to obesity, and the interrelationship between obesity, physical activity and nutrition and other determinates.

Design: This literature review draws from data reported from cross sectional studies, prospective studies, clinical trials, meta-analysis, Cochrane reviews, and national and international report papers.

Results: Combined diet and regular exercise does appear to be the most effective therapy for weight loss and also weight loss maintenance. The synergistic relationship appears to be that weight loss through dietary restriction alone results in reduction in energy expenditure, while physical activity increases energy expenditure, and the combination of the two leads to reduction in body mass, without subsequent reduction in resting energy expenditure. Regular physical activity also appears to be critical specifically for the reduction of obesity related and other chronic diseases. Behavioural change is now recognised as an important component of any response to obesity and should be incorporated into treatment for weight loss and weight maintenance.

Conclusion:

More national and international research is required to determine the best strategies for obesity prevention and treatment. Widespread promotion of regular physical activity is essential not only for weight loss and maintenance, but for many aspects of health.

1. Introduction

Obesity can be defined as a disease in which excess body fat has accumulated to an extent that health is adversely affected[1]. Body mass index (BMI) is a measurement which is widely used to estimate the prevalence of overweight and obesity within a population, and it is calculated as weight (kg)/height squared (m^2). Cut off points of 25Kg/m^2 and 30Kg/m^2 are recognised worldwide as definitions of overweight and obesity, respectively. Studies have shown that BMI is significantly correlated with total body fat for the majority of people[1]. The health concerns with obesity arise as it has been found to be a major contributing factor to serious diseases including cardiovascular disease, type 2 diabetes and hypertension[2], and the rapid increase in prevalence worldwide over the past decades, would also account for the increase in prevalence of obesity related diseases. In Europe the prevalence of obesity in most European countries has increased by 10-50% in the last ten years, with current data from individual national studies suggesting that between 10% and 20% of men and 10% and 25% of women are obese[3]. An increase was also reported in Irish studies, where data from the Lifestyle, Attitudes and Nutrition in Ireland[4] survey indicated that levels of self-reported overweight in Irish adults had increased from 32% overweight in 1998 to 36% in 2007, with levels of self-reported obesity over the same period increasing from 11% to 14%. However, based on measured data, higher percentages of respondents were found to be overweight and obese with 38% adults overweight and a further 23% obese[5,6]. Obesity in childhood is also on the increase in Ireland[2], following the worldwide trend[7], which is of major concern as long term follow up studies show that obese children tend to become obese adults[8,9].

Obesity is a multifactorial disease, and caused in the majority of cases simply by an imbalance of energy intake and energy expenditure. Energy intake is concerned with energy from dietary intake, while energy expenditure can be divided into the following components:

1. Resting metabolic rate (RMR), the largest single component of energy expenditure. RMR is synonymous with resting energy expenditure (REE) and approximates the minimum energy expenditure necessary for critical body functions. A major factor that determines resting metabolic rate is fat-free body mass (FFM). FFM consists of largely metabolically active muscle and organs and also tissues such as bone and connective tissue.
2. The thermic effect of food, which is the increase in energy expenditure that occurs after eating; and
3. Energy expended in physical activity. This varies due to differences in body mass.

The energy expended in physical activity is an important component in controlling energy expenditure. Increases in physical activity lead to increases in energy expenditure, which can induce an energy deficit[10]. In addition physical activity can also affect energy expenditure by promoting muscle growth, thereby increasing FFM. As FFM is the main determinant of resting energy expenditure, an increase in FFM can lead to a subsequent increase in RMR, which in turn can affect energy balance.

Indeed the increase in the prevalence of obesity, observed worldwide has been linked with widespread declines in the level of daily physical activity as well as increased availability of energy-dense foods and changing food habits[11]. Thus it is acknowledged that prevention and treatment must rely on modification of diet and activity patterns[1]. However it is also understood that obesity is ultimately caused by a complex interaction between genetic, behavioural and environmental factors[12] and recent studies have aimed to investigate the efficacy of current treatment strategies and to better understand the exact mechanism and potential synergy between diet and physical activity along with other determinants as an effective means for prevention and treatment of obesity.

2. Dietary therapy for the treatment of Obesity

Dietary energy restriction

Energy intake was found to be positively correlated with BMI in Irish men and women in the IUNA study[13], and the growing research to show the association between consumption of energy dense foods and obesity is impressive and convincing[2]. The basics of dietary therapy for obesity are thought to be straightforward. To lose weight, there must be an energy deficit so that intake is below expenditure therefore restrictions of calories represent the first line therapy for obesity[14]. This theory is supported by clinical trials investigating energy restriction for weight loss. In a recent trial by Heilbronn et al[15], they found significant weight loss after energy restriction for 12 weeks in a group of obese women. Likewise Wang et al[16] investigated the effect of calorific restricted diet of 1200 Kcals on body weight and subcutaneous fat. After the 6 month calorie restriction, weight and BMI were significantly reduced by 11% and total body fat was reduced by 13%. Despite these results supporting the role of calorie restriction as a lone treatment for weight reduction short term, longer term follow up studies suggest that most of the weight is regained[14,17,18]. Although behaviour clearly play a role, with long term adherence being a real issue, it would appear that weight maintenance by calories restriction may be antagonised

by a reduction in total energy expenditure, due firstly to a reduction in RMR, which is mainly as a result to a reduction in FFM[17,19], and also as eating less causes a reduction in diet related thermogenesis[20]. However it has been suggested that factors associated with dietary composition may modify these physiological adaptations to energy restriction[17].

Altering Diet Composition-High or Low Carbohydrate, Low Fat Diets and High Protein Diets

Differing thermogenic responses may result following the intake of differing nutrients, which may hence affect total energy expenditure during weight loss[17]. Therefore macronutrient composition of the diet and its effect on bodyweight reduction has been explored.

Low fat Diet, High CHO Diets

Altering the carbohydrate to fat ratio in the diet has been examined in many studies however the results have been conflicting. A large study, The CARMEN (Carbohydrate Ratio Management in European National diets) study investigated the impact of complex versus simple CHO in an ad libitum fat reduced (by 10% of total energy) regimen. After 6 months, a significant reduction in bodyweight and fat mass was observed on both diets while sustaining fat free mass[21]. A reduction in body weight and body fat have also been reported in many other studies following a low fat diet short term[22,23]. Long term low fat diets may also be important for weight loss maintenance. This was supported by observations from the National Weight Control Registry which is a registry of individuals in the United States of America who have been extremely successful at weight loss maintenance, which has shown that many weight maintainers have followed a diet, consuming approximately 24% of energy from fat[24]. The exact reason for this is unknown, and there is a paucity of studies reporting on FFM preservation and whether this is what is key to the success of limiting fat for weight maintenance. In addition although results from these studies would imply that lowering the fat content of the diet can be an effective strategy for weight loss, when fat intake is reduced it is typically associated with a spontaneous reduction in energy intake and this is believed by many to be the mechanism for weight reduction[14].

Low CHO Diets

Low CHO diets have also shown positive effects on short term weight loss. A recent randomised controlled trial, subjects assigned to a low CHO, high protein, high fat diet lost a greater body weight, than did those assigned to the high CHO, low fat, low calorie diet after 6 months. However no significant difference was noted after 1 year[25]. In another study by Brehm et al[26]

investigating changes in body composition following an ad libitum, low CHO diet or an energy restricted, low fat diet, reported the loss of bodyweight and fat mass was greater in the low CHO group, with no difference in reported energy intake. Similar reductions in FFM and RMR were recorded for both groups**.** These results however could not be explained by increased REE or physical activity and could not be accounted for by their reported energy intakes, and may have been down to underreporting in the low fat diet group.

High Protein Diets

The effects of high protein diets have also been investigated, but as with low fat and low CHO the evidence remains inconclusive. In a randomised controlled study by Skov et al[27], 60 overweight subjects were assigned to a control diet or an ad libitum low fat diet which was either high in protein or high in CHO. After 6 months, a significant loss of bodyweight and body fat was achieved on both the low fat diets, which was greater in the high protein group, but there was between group differences in the methodology which may have biased results. However other studies have also supported the role of high protein diets for lean tissue preservation during weight loss[28] and more weight loss[29] when compared with high CHO dietary interventions. The explanation for these observations remain unclear, although some have suggested it can increase satiety and thermogenesis, which may thus have a double impact on energy balance by altering energy intake and expenditure[30], while others believe sparing of muscle protein loss[28], and enhanced glycemic control[28,31] may also play a role. In contrast in a recent study, investigating energy restricted diets, high in protein or monounsaturated fat, no differences with respect to body composition were found[32].

At present, evidence is conflicting on the role of macronutrient composition with weight loss, and the optimal macronutrient composition has not been confirmed. There is also a scarcity of studies which have examined their role for long term weight loss and weight maintenance. More studies need to be carried out to clarify the relative merits of altering dietary composition and if they ultimately only affect weight loss by altering energy intake alone, which is a popular belief at present[14,30].

3. Physical activity intervention for the treatment of Obesity:

As increasing energy expenditure can also promote negative energy balance, an increase in physical activity could also promote weight loss. In addition weight loss due to exercise may be

associated with retention of lean body mass[17]. Although the increase in energy expenditure during the exercise period help to encourage energy imbalances, additional mechanisms may increase resting metabolism, thus further promoting energy imbalances. Given the association between lean tissue and RMR, the most apparent impact of exercise training on resting metabolism is the ability to initiate skeletal muscle growth[33]. Numerous studies have also shown that increased sedentary behaviour such as watching television and playing computer games are associated with increased prevalence of obesity, while involvement in more physically active pursuits provides protection from obesity in children[34]. In addition recent results from a 6 year observation of the Nurses' Health Study cohort suggested that 30% of new cases of obesity could be averted by adapting a relatively active lifestyle[35]. The effects of physical activity alone on body weight have also been examined in randomized controlled trials. Frey-Hewitt et al[36] randomly assigned 121 overweight men to 1 year of energy restriction or engagement in walking/jogging when compared with a control group. Although the reduction in bodyweight and fat mass in the dieters was greater than in the exercise group, exercise training was superior to dieting with respect to the maintenance of fat free mass. This was highlighted in a more recent randomized controlled trial, where no difference was reported in weight loss between diet or exercise only intervention, but exercise alone was associated with substantial reductions in total and abdominal mass, and preservation of skeletal muscle mass[37]. As the preservation of FFM is believed to be an important factor for weight maintenance, physical activity is universally promoted as a necessity for weight maintenance[38]. It has been suggested that the outcomes of exercise intervention studies with respect to body compositional alterations may to be related to the type of exercise with intensity, frequency and duration of the exercise bouts as limiting factors[17].

Intensity, Duration and Frequency

A recent Cochrane review which examined randomized controlled trials concluded that increasing exercise intensity increased the magnitude of weight loss[39]. The review did not however report data on body compositional changes, nor did it report on long term weight loss maintenance. Two studies examining the effects of low and high intensity exercise on overweight or obese adults failed to show any significant difference on body weight or body composition[40,41]. Long-term data from randomized clinical trials examining the effect of different durations of exercise on weight loss are lacking, however differing exercise intensities and durations were examined by a large randomized controlled trial, which aimed to compare the effects of different durations and intensities of exercise on 12-month weight loss and cardio-

respiratory fitness. Participants were randomly assigned to 1 of 4 exercise groups (vigorous intensity/high duration; moderate intensity/high duration; moderate intensity/moderate duration; or vigorous intensity/moderate duration. Results showed that participants randomized to vigorous exercise intensity did not have greater weight loss than those randomized to a similar dose of exercise performed at a moderate intensity. However, when data were analyzed based on the amount of exercise performed, greater levels of exercise were associated with a greater magnitude of weight loss following 12 months of treatment[30]. Based on these results, the researchers recommended that interventions should initially target the adoption and maintenance of at least 150 min/wk of moderate intensity exercise, and when appropriate, eventually progress to exercise levels of 60 min/d, which is consistent with current recommendations[42,43]**.** There have also been few studies that have examined the effect of frequency on weight loss and body composition. One study investigated the effect of moderate intensity exercise on 2 and 5 days a week with total energy expenditure being the same in 30 overweight women. After 8 weeks, a significant loss of bodyweight and body fat indicated beneficial effects regardless of the training frequency[44].

Type of exercise

Although most research studies have examined the effect of endurance exercise on weight loss, the inclusion of resistance exercise has also been found to have a role in weight loss programmes. Resistance training is a potent stimulus to increase fat free mass, muscular strength and power and thus may be an important component of a successful weight loss programme by helping to preserve FFM, while maximizing fat loss[10] and indeed some reports suggest weight training in favour of aerobic exercise to preserve or increase FFM[45]. In one study investigating whether a combination of resistance training and walking was more beneficial than resistance exercise on its own in 19 moderately obese women, results showed after 20 weeks similar increases in FFM in both groups when compared with sedentary control group, and an associated elevation in RMR in the strength trained group[46]. However these results are not supported by many larger studies especially in studies with dietary therapy. In a long term study Wadden et al[47] investigated changes in body composition, and resting energy expenditure (REE) in 128 obese women who were randomly assigned to 1 of 4 treatment conditions: diet alone, diet plus aerobic training, diet plus strength training, or diet combined with aerobic and strength training (i.e., combined training). All women received the same 48-week group behavioural program and were prescribed the same diet. Results showed that resistance exercise alone or combined with endurance exercise did not enhance weight loss compared

with endurance exercise alone. These results are also supported by other studies which have shown little or no benefit in terms of absolute weight loss by the addition of resistance exercise[48-50]. Therefore although resistance exercise may improve muscular strength in overweight adults, there is a scarcity of scientific evidence to suggest that resistance training is superior to endurance exercise for weight loss. However the ability to improve muscle strength and endurance may have an important impact on functional tasks, which may help facilitate the adoption of a more active lifestyle in sedentary overweight and obese individuals[10].

Despite the importance of exercise, there is little evidence to suggest that exercise alone produces magnitudes of weight loss similar to what can be achieved by dietary modification, or as a combined therapy.

4. Combined diet and physical activity intervention:

Inducing negative energy balance is the most important aim of weight loss programmes. Physical activity in conjunction with dietary energy restriction has long been promoted as an important component of successful weight loss regimens[17], and there is compelling evidence from epidemiologic investigations and clinical trials supporting their use[51-53]. The potential synergy between the two has been discussed, and is believed that if weight loss through dietary restriction results in reduction in energy expenditure, while physical activity increases energy expenditure, the combination of the two could potentially lead to reduction in body mass, without subsequent reduction in resting energy expenditure thus improving the ability to achieve long term weight loss. Some clinical studies however have failed to show a benefit of the combined approach. Janssen et al[54] investigated the effects of 3 treatment methods; diet alone, diet with aerobic exercise, or diet with resistance exercise over a 16 week period in a group of 38 obese women. They found significant reductions in body weight, and in total and abdominal fat in each group, however the changes in body fat or weight loss were not different across any of the treatment groups. In a further study investigating whether diet alone or with exercise produced identical reductions in weight, fat mass and abdominal fat mass in a group of 48 overweight adult[55]. It must be noted that both these studies had small sample sizes and were of relatively short duration which may have affected the failure to show significance. In contrast in a recent Cochrane review and two meta-analyses of randomised controlled trials, the combined benefits of diet and exercise have been highlighted. A Cochrane review[39] which reviewed randomized controlled trials that examined body weight change using one or more physical activity intervention in adults with overweight or obesity at baseline. When compared with no treatment,

exercise resulted in small weight losses across studies. Exercise combined with diet resulted in a greater weight reduction than diet alone. The results of this review therefore support the use of exercise as a weight loss intervention, particularly when combined with dietary change. These results were also supported by two further recent meta analysis reviewing the effect of diet-plus-exercise interventions vs. diet-only interventions on long term weight loss[20,56]. In the meta analysis by Curioni & Lourenço[56] which investigated weight loss trials with an inclusion criteria of 1 year follow up of intervention, they found that diet associated with exercise produced a 20% greater initial weight loss than diet alone. The combined intervention also resulted in a 20% greater sustained weight loss after 1 y than diet alone. In both groups, however almost half of the initial weight loss was regained after 1 year. The effect of combined therapy was further studied in a meta analysis[20] which reviewed randomized controlled trials with a duration minimum of 6 months among obese or overweight adults. Results from this meta analysis showed that weight loss was greater for the diet-plus-exercise group than the diet-only group in reviewed studies, with differences significantly greater with interventions with a duration of longer that 1 year than for interventions of shorter duration. Even in studies lasting 2 years or longer, diet-plus-exercise interventions provided significantly greater weight loss than diet-only interventions. However, similar to the meta analysis by Curioni & Lourenço[56], both diet-only and diet-plus-exercise programmes were associated with partial weight regain[20]. It was reported however that intervention time was significantly associated with greater weight loss when exercise was added to the intervention programme, suggesting that prolonged active intervention may be very important for maximizing benefits in combined therapy intervention trials[20]. The beneficial role of diet and exercise in weight maintenance is also acknowledged in a report on the American national weight control registry which gives us valuable insight into the type of intervention used by successful weight maintainers. This report states that 89% of weight loss maintainers reported modifying both diet and exercise to achieve their successful weight loss. Although approaches to weight loss differed widely among the subjects, similarities were found in subjects used for successful weight maintenance, which included eating diets low in fat, and high in carbohydrates, and engaging in moderate intensity physical activity of about 60 minutes per day[24]. Thus combined diet and regular exercise can be an effective weight loss therapy for short term weight loss and also weight loss maintenance. The regain of weight noted in randomized trials may be as a result of poor compliance, which is often an issue in long term intervention studies, and indeed it has been reported that the degree of adherence to weight loss intervention is a strong predictor of weight loss[57, 58].

6. Attitudes and Behaviour towards diet, physical activity and obesity.

Research in social psychology tells us a great deal about how people make their decisions. What motivates and determines health-related behaviour is complex including past habits, beliefs, moral climate and translating intention into action, but in today's societies, there is a psychological conflict between what people want (e.g. fatty, sweet foods) and their desire to be healthy and/or slim[59]. Knowledge about healthy food choices can be a predisposing factor for the adoption of a healthy diet but it is insufficient to motivate healthy eating[59]. This has been illustrated in numerous studies despite there being high levels of knowledge about healthy foods and activities this knowledge does not generally translate into reports of consistently healthy behaviours. This may be a possible reason for the poor compliance observed in weight loss intervention trials, in addition to the rising levels of obesity, despite global public health prevention strategies. A qualitative study by Hesketh et al[60] aimed to elicit child and parent views regarding social and environmental barriers to healthy eating, physical activity and child obesity prevention programmes. Results from this study showed children and parents were generally well informed about the health value of different foods, and children as young as 5-6 years of age could identify pictures of healthy and unhealthy foods, and were aware of the nutrients contributing to their perception of foods being more or less healthy. In addition most children easily identified physically active pursuits as healthy and discussed the benefits of physical activity including fitness and fat reduction. However despite reporting good awareness and understanding about health foods, this did not generally translate into reports of consistently healthy behaviours with children exhibiting a preference for less healthy foods, and reporting that they regularly ate unhealthy foods, although they did describe home meals as being predominately healthy. They also reported frequently spending their unstructured time in sedentary pursuits. Parents were aware that their family diet and activity levels were not as healthy as they would like, despite their knowledge and awareness of what comprises a healthy lifestyle. The authors reported that lifestyle demands and pressure from their children as a result of advertising and child peer pressure were the main barriers. This illustrates the importance of the cultural and social context of an individual with regards to their behaviour and must also be considered when addressing behaviour modification.

In addition another determinant of the practice of health behaviour is a sense of self efficacy, which is the belief that an individual can practice a particular behaviour, and has been shown to affect both change and maintenance of change of a range of health behaviours including

healthy eating patterns[59], physical activity[61] and weight control[59], and this would suggest improving self efficacy in obese individuals to be important.

Understanding attitudes and motivation underlying human health behaviour is an essential part of understanding obesity aetiology, and is also recognized as playing an important role in improving prevention and treatment strategies for obesity.

The healthful lifestyle: does intervention for one health behaviour affect the other?

Based on various health psychology conceptual models such as the Health Belief model which maintains that health habits are a function of perceived vulnerability to a disorder and the belief that a particular health measure will be sufficient to overcome this vulnerability[62], it is conceivable that an improvement in one health behaviour e.g. physical activity due to participation in an exercise intervention programme could lead to a greater sense of self efficacy, a belief that other behaviour changes such as improvements in diet are possible, and an increase in motivation to adopt behaviour changes. This could potentially impact health promotion programmes, as if they target one behaviour, this could produce favourable side effects by stimulating changes in another behaviour without it being specifically targeted in health promotion. There is indeed some evidence to suggest that health behaviours may cluster together in particular patterns. Johnson et al[63] investigated the interrelationships between health behaviour in a large sample of university students. They showed that risky behaviours including drinking excessively and smoking correlated with each other, while physically active young adults had in general more healthful diets, including eating more fruit and vegetables and drinking fruit juices. However they reported that consumption of fatty foods was less consistently associated with physical activity. In addition Pate et al[64] specifically examined the associations between physical activity and other behaviours and found low physical activity was associated with several other negative health behaviours in teenagers including cigarette smoking, greater television watching, and lower fruit and vegetable consumption. A more recent cross sectional study supported these results by reporting that sedentary individuals consumed smaller amounts of foods and nutrients considered to be more healthful than the more active. For nutrients considered to be harmful such as saturated fat and trans fat, the relationship was inverse[65]. This pattern has also been reported in other cross sectional studies which have shown that physical activity is correlated with more healthful diet quality however prospective studies generally have not confirmed this association[66]. A possible reason for this is that cross-sectional studies only collect data from one point of time and provide no information on temporal

changes of health behaviours, while prospective studies examine behaviours over time and determine whether two or more behaviours are independent or tend to change one after the other. A recent randomized controlled trial[66] investigated whether initiating physical activity induces change in other health behaviours in a large sample of sedentary overweight women over a 12 month period. The authors reported changes in overall dietary intake between the exercise and the control group was not statistically different after 12 month follow up, suggesting that initiating an exercise physical activity will not induce substantial changes in other health behaviours. However the authors note a limitation of the study being that as subjects were asked to report dietary intake, underreporting of dietary intake is likely to have occurred, which may have lead to an underestimation of differences between the control and exercise group. Patterson et al[67] investigated the clustering of four health behaviours;-cigarette smoking, physical activity, diet and alcohol in a large population representative sample. They reported only 10% of the population had acceptable levels of all four behaviours, while surprisingly 25% of sample had a good diet but were the most sedentary of all lifestyle typologies. In a more recent study, Reeves & Rafferty[68] examined four healthy lifestyle characteristics-including healthy weight, regular physical activity, adequate fruit and vegetable consumption, and non smoking based on national US data from the Behavioural Risk Factor Surveillance System in 2000, which consists of annual statewide random household surveys. They found that only 3% of US adults followed a combination of these four modifiable health behaviours, identical to results from the Nurses' Health Study[69, 70]. Therefore as there is a lack of conclusive evidence on the existence of healthful behaviour clustering, it would be prudent to address and encourage both nutrition and physical activity behaviour modification in obesity treatment strategies.

Behavioural therapy as a treatment for Obesity

As behavioural change is now recognised as an important component of any response to obesity, in recent years behaviour therapy has become a useful adjunct when incorporated into treatment for weight loss and weight maintenance across all populations-from children to adults. Behavioural therapy comprises of any method to generate change in eating habits or lifestyle, including formal cognitive behaviour modification and training in behavioural skills. There have been a large number of studies and systematic reviews looking at the effectiveness of a range of behavioural interventions for obesity prevention and management in adults and children including cognitive-behavioural treatment, problem-solving approaches, family therapy and multicomponent behavioural programs which incorporate a variety of behavioural techniques.

Cognitive restructuring teaches patients to identify, challenge, and correct the irrational thoughts that frequently undermine weight control efforts. The main principle of this treatment approach include the modification of current behaviour patterns, new adaptive learning, problem solving and a collaborative approach between client and therapist, and is now used as an important part of the treatment of obesity in adults. Cognitive techniques are particularly helpful in dealing with lapse or setback situations, and an excellent technique for improving motivation in patients if weight regain occurs after weight loss[71]. Group behaviour based lifestyle intervention also appears to be useful for encouraging weight maintenance. Ash et al has shown that a group behaviour based lifestyle intervention programme was as effective as intensive individualised dietetic intervention in terms of weight loss and improvements in self-efficacy among cardiac patients. At 1-year follow-up the group intervention delivered improved weight maintenance[72]. Mulivill & Quigley[73] analysed reviews which examined diet, physical activity and behavioural approaches in the treatment of obesity and reported that there is evidence to support the role of behavioural therapy and a combination of behavioural therapy techniques in conjunction with other weight loss approaches as being effective for the treatment of adult obesity over a one year period. However weight regain seen with the individual behaviour approach suggests that it is not sufficient on its own to reduce the incidence of obesity, and that population approaches that incorporate more widespread environmental changes must also be included.

For obesity in children a whole family approach appears to be most effective[73]. Transformed dietary behaviours in the entire family including encouragement of the child and parental participation for a more healthful way of life are key factors in child weight loss[74]. Programmes which focused on the parent as being the key in the behaviour change of the child have reported promising results. Systematic reviews which investigated the treatment of obesity in children supported the use of multifaceted family based behaviour modification programmes, in which the parents take primary responsibility for treatment. The programmes comprised of diet, exercise, reducing sedentary behaviour and lifestyle counselling, with training in child management, parenting and communication skills[73, 75]. However potential problems of parental involvement have been discussed, one being parents may not identify that their child is obese or parents may accept obesity but reject that this is of any consequence[76]. It has also been reported that behavioural therapy can be more successful if given in more organized surroundings, such a specialised school or residential treatment centre using a planned mixture of beneficial techniques however this is best set aside for older children who do not mind being away from home and can make friends easily with others[76].

Regardless of the behavioural therapy used, an important practical component of achieving behaviour modification is setting realistic goals. Satisfaction with treatment is based on comparing what is obtained with what was expected, and hence if expectations are clarified and goals are realistic, this improves the chances that individuals achieve their goals, which will in turn improve self efficacy and motivation to continue with behavioural changes. Bearing this in mind, teamed with the fact that current attempts to reduce obesity levels are failing, it has now being suggested that modest weight loss (5%-10% of initial weight), in clinical practice should be advocated, with more emphasis placed on encouraging individuals to attain and maintain positive health behaviours including regular physical activity and improved diet quality which in turn can have positive effects for health.

Effects of diet and physical activity on biological risk factors for obesity related diseases.

The World Health Organisation estimated that approximately half a million people in North America and Europe died from obesity related chronic diseases in 2002, and this is set to increase by one third over the next 20 years if nothing is done[77]. As current intervention strategies are failing to result in sufficient weight loss to reduce levels of obesity, it is now thought that rather than concentrating on weight loss as a sole success endpoint, reduction in the risk of obesity related chronic disease should also be an important consideration. Modest weight loss has been associated with reduction in total mortality, reduction in total cholesterol, obesity related cancers, diabetes related deaths[2] and hypertension[78]. In addition data from longitudinal studies such as The Coronary Artery Risk Development in Young Adults Study (CARDIA)[79,80], the Atherosclerosis Risk in Communities Cohort[81-83]. and the FELS Longitudinal Study[84-86] provide evidence that the prevention of weight gain may be the easiest way to prevent the development of undesirable changes in Cardiovascular Disease (CVD) risk factors including, increased Low Density Lipoprotein (LDL) cholesterol, total cholesterol, triglycerides, fasting glucose, and decreased High Density Lipoprotein (HDL) cholesterol. Interestingly long-term data from the CARDIA study indicate that regardless of BMI, individuals that maintain a stable BMI minimized the undesirable changes in CVD risk factors that may be associated with aging[79].

The role of regular physical activity specifically for the reduction of obesity related diseases appears to be very important. Evidence shows that for all populations–children, adolescents, adults and older adults, people with disabilities, and those across all races and ethnic regular physical activity reduces the risk of many adverse health outcomes including heart disease, type

2 diabetes, breast cancer and osteoporosis[42]. Indeed benefits of physical activity for the reduction of chronic health risks are seen with minimal weight loss of less than 3%. Donnelly et al[87] randomized sedentary, moderately obese females to 18 months of either continuous or intermittent exercise. After 18 months of exercise, weight loss was approximately 2% in the continuous group and approximately 1% in the intermittent group. Despite the minimal weight loss, both groups had significant improvements in HDL cholesterol and insulin. In a further randomised trial, sedentary, overweight men and women were assigned to either, a control group, a high-amount high-intensity exercise group, a low-amount exercise group, low amount high-intensity group, or a low-amount moderate-intensity group. Despite minimal weight of less than 2% loss in all groups, there were significant beneficial decreases in triglycerides and increases in HDL cholesterol. These results are supported by a recent Cochrane review examining clinical trials where exercise as a sole weight loss intervention was investigated. They reported that exercise as a sole weight loss intervention resulted in significant reductions in some of the biological risk factors for CVD and diabetes including diastolic blood pressure, triglycerides and fasting glucose even if no weight is lost[39]. There is a paucity of studies investigating the role of exercise for long term improvement in disease risk however data from the Nurse's Health Study examined the association of sedentary behaviour and television watching with the risk of obesity and type 2 diabetes during a 6-yr period. They reported regular minimal activity was associated with 9% reduction in obesity and a 12% reduction in type 2 diabetes and each 1 hour per day of brisk walking was associated with a 24 reduction in obesity and a 34% reduction in diabetes[69]. A limitation of this study is that it was observational in nature and physical activity was self-reported.

Some studies have also highlighted benefits of dietary intervention alone for risk reduction of chronic disease. A randomised controlled trial investigated whether the combination of diet alone or diet combined with aerobic or resistance exercise was associated with greater improvements in metabolic risk factors such as fasting insulin, total and LDL cholesterol. They found that weight loss occurred across all the treatment groups, which was associated with reduction in LDL and total cholesterol, and fasting insulin. However in contrast to previous studies discussed above, they reported the addition of both forms of exercise to the energy restrictive diet did not enhance the improvement of the metabolic profile[88]. In addition Heilbronn et al[15] showed that C-Reactive Protein, which is a strong inflammatory response protein that has a strong independent predictor of cardiovascular mortality was significantly reduced with modest energy restriction and weight loss. However the sample sizes in both these trials were quite

small, and the trial duration was short, and larger trials with longer duration would need to be carried out to provide more conclusive evidence.

There appears to be growing evidence to suggest that regular physical activity alone, can improve many chronic disease risk factors. There is a lack of studies that has investigated the time course of these improvements, the permanence of these improvements over time, and if there are diminishing returns for the amount of weight lost. Therefore more research is perhaps needed to strengthen the already strong evidence supporting the beneficial role of regular physical activity. It cannot be forgotten however that the role of weight reduction in risk reduction is also acknowledged, and despite lack of evidence supporting the role of dietary therapy alone, the combined approach of dietary and lifestyle modification is the most successful method to initiate weight loss.

7. Genetic determinants of Obesity

Genetic predisposition to obesity is also believed by many to play a very important role in the aetiology of obesity[12]. The genetic contribution to body weight has been established through family studies, investigations of parent-offspring relationships, and the study of twins and adopted children[89]. In addition studies in humans have now identified a number of specific genes associated with obesity. The recent discovery of the 'ob' gene has led to a renewed interest in understanding the patho-biological basis of genetic predisposition to obesity. The 'ob' gene codes a hormone called leptin which is an adipose derived protein hormone and has been found to have important effects on regulation of energy intake and energy expenditure[18]. It is believed that its main role is to increase food intake when body weight is low. When fat stores are low, levels of leptin fall and hunger and food-seeking behaviour are stimulated. An increase in body fat, however, stimulates the secretion of leptin, which circulates in the bloodstream, crosses the blood–brain barrier and acts centrally to increase satiety. This in turn reduces the overarching drive to eat, leading to a period of negative energy balance, and reduction in the secretion of leptin from fat. In this way, the system returns to equilibrium. Any mutation of 'ob' gene leads to improper coding of leptin, which has been associated with obesity[89]. Other genes are also now being identified that may contribute to obesity through dietary intake and specifically through increases in dietary fat and carbohydrate[18,90].

It has also been suggested that there may be genetic factors involved with energy expenditure and the effect of physical activity on weight control, by individuals being 'responders' and 'nonresponders' to intervention for obesity management [10]. Bouchard et al[91] examined the effect

of exercise on weight loss both within and between pairs of identical twins. Results from this study showed that the variance for changes in body weight were 6.8 times greater between pairs than within pairs, suggesting the effect of exercise may be influenced by genetic differences between individuals. However more research needs to be done to investigate this area to provide more concrete evidence. Indeed evidence with regards physiological differences in energy balance control between obese and non obese remains inconclusive. Numerous large studies have failed to find evidence to support the belief that obese people must have slower metabolic rates, either burning energy more slowly than thin people, or being metabolically more efficient[89]. In fact, the converse appears true. Energy expenditure while resting actually increases with body weight, reflecting the metabolic costs of maintaining a larger body size. After adjustment for differences in body size and composition, there is a remarkable similarity in energy expenditure between individuals[89]. Evidence from research also argues against any difference in basic physiology between the non obese and the obese. Studies conducted in controlled experimental conditions in which lean and obese individuals are over- or underfed show similar rates of weight gain or loss[89]. This evidence suggests that physiological differences between people are not the root cause of obesity.

Although it is now widely acknowledged that obesity has a genetic component, it does not appear to be a simple genetic disorder, rather there is an underlying genetic predisposition to obesity on to which environmental factors such as a constant supply of convenient, inexpensive and palatable foods, and reduced need for physical labour in jobs, and daily life are layered. Indeed the prevalence of obesity increases so rapidly in many populations that the changes cannot be attributed to changes in genetic inheritance. Continued research is needed to fully comprehend the genetics of mechanisms responsible for energy balance, the gene-environment interactions and relate these to why certain susceptible individuals passively over consume leading to obesity. This will hence lead to formulating intervention strategies which capitalise on these insights.

8. Current Recommendations

National food based dietary guidelines for healthy eating have been developed in most countries across the globe. The guidelines vary according to the specific health, behaviour, food availability, culture and economic conditions within a country, but in general stress the importance of dietary variety and balance, promote fruit and vegetable consumption, discourage excessive salt, fat, sugar and alcohol consumption, and promote physical activity. However

specific guidelines for physical activity are not as well established. Indeed Ireland is one of only a number of countries including America, Australia and Canada to have published national guidelines for physical activity. The current Irish guidelines take into account the benefits of physical activity for weight loss and weight control, and also the various other health benefits that have been associated with physical activity including lower risk of high blood pressure, type 2 diabetes[42].These guidelines specify recommendations for various population groups including children, adults, older adults and also for individuals trying and needing to lose weight. For children they recommend 60 minutes accumulative moderate to vigorous exercise every day. For adults, they recommend 30 to 60 minutes of moderate to vigorous intensity physical activity on 5 days or more. Both endurance and resistance physical activity is also recognised as beneficial. These recommendations are similar to American, Australian guidelines and Canadian guidelines[92-94]. However the Irish guidelines are the only to acknowledge that for weight loss, more physical activity than the recommended levels for the healthy adults is needed. They recommend at least 60-75 minutes per day for weight loss in adults, dependant on the individual's capabilities, and also amount of weight loss needed. The higher level is encouraged to promote weight maintenance, for which physical activity has been shown to play an important role. The growing evidence supporting the critical role of physical activity for many aspects of health highlights the need for more guidelines to be produced at national and international level, and the promotion of these guidelines should be considered as important as dietary guidelines by all involved in health and health promotion alike.

9.Conclusion

This review shows the strong interrelationship between physical activity, nutrition and obesity, in addition to recognising the important role of other factors involved in obesity aetiology including attitudes and behaviours and also genetic factors. There is ever growing evidence from epidemiology studies and intervention trials, supporting the role of combined dietary and exercise modification to best prevent and treat obesity. It is believed that the combination of the two reduces body mass, but by preserving FFM does not lead to subsequent reduction in resting energy expenditure thus improving the ability to achieve long term weight loss. However the exact mechanism has yet to be confirmed, and there is a need for further research to better understand the interactions and synergy between diet and physical activity in obesity.

Encouragement and facilitation of behavioural change is also very important, and has promising implications for intervention strategies in obesity management. However the ever rising levels of obesity both in Ireland and worldwide suggests that current strategies are failing to have a sufficient impact, which could be as they do not offer the range and depth of intervention needed. Therefore further research regarding the effectiveness of differing combinations of obesity prevention and treatment programmes is required. It must also be noted that interventions based on improving nutrition and physical activity can be effective for individuals, but shifting the population distribution will also require interventions that target elements of the obesogenic environment also, which must be considered with utmost importance in public health strategies. In addition more emphasis needs to be placed on building public awareness and advocating the importance of physical activity, which is a critical public health issue due not only to it being key to weight loss and weight loss maintenance, but also independently for many aspects of health. The recent publication of national guidelines for Ireland will greatly facilitate the promotion of physical activity, and may indeed lead to the development and implementation of more successful obesity prevention and treatment strategies in the future.

References

1. World Health Organisation (2000). Obesity, preventing and managing the global epidemic: Report of the WHO consultation of obesity. WHO, Geneva.
2. National Taskforce on Obesity (2005).Obesity: The policy challenges. The report of the National Taskforce on Obesity, Dublin.
3. International Obesity Task Force (IOTF) and European Association for the Study of Obesity (EASO) (2002).Position paper; Obesity in Europe-the case for action. London. www.iotf.org
4. Harrington, J., I. Perry, et al. (2008). SLAN 2007: Survey of Lifestyle, Attitudes and Nutrition in Ireland. Dietary Habits of the Irish Population. Dublin, Department of Health and Children. Health Behaviour in School Aged Children Ireland. Available at the following link: http://www.nuigalway.ie/hbsc/
5. Friel, S., Nic Gabhainn, S. & Kelleher, C. (1999). The National Lifestyle Surveys: Survey of Lifestyle, Attitudes and Nutrition (SLÁN) and the Irish Health Behaviour in School-Aged children survey (HBSC): Regional Report. Dublin, Department of Health and Children.
6. Morgan K, McGee H, Watson D, Perry I, Barry M, Shelley E, Harrington J, Molcho M, Layte R, Tully N, Van Lente E, Ward M, Lutomski J , Conroy R and Brugha R (2008). SLÁN 2007: Survey of Lifestyle, Attitudes & Nutrition in Ireland. Main Report. Dublin, Department of Health and Children.
7. Wang Y and Lobstein T (2006). Worldwide trends in childhood overweight and obesity. *Int J Pediatr Obes* **1**, 11-25.
8. Doak CM, Visscher TLS, Renders CM and Seidell JC(2006). The prevention of overweight and obesity; a review of interventions and programmes*. Obes Rev* **7,** 111-136.
9. Dehghan M, Akhtar-Danesh N and Merchant AT (2005). Childhood Obesity, prevelance and prevention. *Nutr J* **4**, 24.
10. Jakicic JM, Clarke K, Coleman E, Donnelly JE, Foreyt J, Melanson E, Volek J and Volpe SL (2001). Appropriate intervention strategies for weight loss and prevention of weight regain for adults. *Med Sci Sports Exerc* 33, 2145-2156
11. Yach D, Stuckler D and Brownell KD (2006). Epidemiologic and economic consequences of the global epidemics of obesity and diabetes. *Nat Med* **12**, 62-66.
12. Newbold RR, Padilla-Banks E and Jefferson WN (2009). Environmental eoestogens and obesity. *Mol Cell Endocrinol* **304,** 84-89.
13. McCarty SN, Robson PJ, Flynn A, and Gibney MJ (2002). Health and Lifestyle variables that predict body mass index and body fat distribution in a nationally representative sample of Irish adults. *Proc Nutr Soc* **61,** 169.
14. Rolls BJ and Bell EA (2000). Dietary approaches to the treatment of obesity. *Med Clin North Am* **84,** 401-418.
15. Heilbronn LK, Noakes M and Clifton PM (2001). Energy restriction and weight loss on very low fat diets reduce C-Reactive Protein concentrations in obese healthy women. *Arterioscler Thromb Vasc Biol* **21**, 968-970.
16. Wang J, Laferrère B, Thornton JC, Pierson RN, Pi-Sunyer FX (2002). Regional Subcutaneous-fat loss induced by caloric restriction in obese women. *Obes Res* **10,** 885-890.
17. Stiegler P and Cunliff A (2006) The Role of Diet and Exercise for the Maintenance of fat free mass and resting metabolic rate during weight loss. *Sports Med* **36**, 239-262.
18. Nammi S, Koki S, Chinnala KM. and Boini KM (2004). Obesity: An Overview on its current perspectives and treatment options. *Nutr J* **3**,3.
19. Menozzi R, Bondi M, Baldini A, Venneri MG, Velardo A and Del Rio G(2000). Resting metabolic rate, fat free mass, and catecholamine excretion during weight loss in female obese patients. *Brit J Nutr* **84,** 515-520.
20. Wu T, Gao X,Chen M and Van Dam RM (2009).Long term effectiveness of diet plus exercise interventions vs. diet only interventions for weight loss: a meta-analysis. *Obes Rev* **10,** 313-323.
21. Saris WHM, Astrup A, Prentice AM, Zunft HJF, Formiguera X, Verboeket-van de Venne WPHG, A Raben A, Poppitt SD, Seppelt B, Johnston S, T H Vasilaras TH and Keogh GF (2000). Randomised controlled trial of changes in dietary carbohydrate diets on body weight and blood lipids: the CARMEN study. *Int J Obes* **24**, 1310-1318.

22. Sloth B, Krog-Mikkelsen I, Flint A, Tetens I, Björck I, Vinoy S, Elmståhl H, Astrup A, Lang V and Raben A (2004). No difference in body weight decrease between a low-glycaemic-index and a high-glycaemic-index diet but reduced LDL cholesterol after 10 week ad libitum intake of the low-glycaemic index diet. *Am J Clin Nutr* **80,** 337-347.
23. Astrup A, Grunwald GK, Melanson EL, Saris WHM and Hill JO (2000). The role of low fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. *Int J Obes* **24**, 1545-1552.
24. Wing RR and Hill JO (2001). Successful weight loss maintenance. *Ann Rev Nut* **21,** 323-341.
25. Foster GD, Wyatt HR, Hill JO McGuckin BG, Carrie Brill C, Mohammed BS, Philippe O. Szapary PO, Rader DJ, Edman JS and Samuel Klein S (2003). A randomised trial of a low carbohydrate diet for obesity. N Engl J Med **348,** 2074-2081.
26. Brehm BJ, Spang SE, Lattin BL, Seeley RJ, Daniels SR and D'Alessio DA (2005). The role of energy expenditure in the differential weight loss in obese women on low fat and low carbohydrate diets. *J Clin Endocr Metab* **90,** 1475-1482.
27. Skov, A. R., Toubro, S., Ronn, B., Holm, L. & Astrup, A. (1999) Randomized trial on protein vs carbohydrate in ad libitum fat reduced diet for the treatment of obesity. *Int J Obes* **23**,528-536.
28. Layman DK, Boileau RA, Erickson DJ, Painter JE, Shiue H, Sather C and Christou DD (2003). A reduced ratio of dietary carbohydrate to protein improves body composition and blood lipid profiles during weight loss in adult women*. J Nutr* **133**, 411-417.
29. Baba NH, Sawaya S, Torbay N, Habbal Z, Azar S and Hashim SA (1999). High protein vs high carbohydrate hypoenergetic diet for the treatment of obese hyperinsulinemic subjects. *Int J Obes* **23,** 1202-1206.
30. Jakicic JM, Marcus BH, Gallagher KI, Napolitano M and Lang W (2003). Effect of exercise duration and intensity on weight loss in overweight sedentary women *JAMA* **290,** 1323-1330.
31. Farnsworth E, Luscombe ND, Noakes M, Wittert G, Argyiou E and Clifton PM (2003). Effect of a high protein, energy restricted diet on body composition, glycaemic control, and lipid concentrations in overweight and obese hyperinsulinaemic men and women *Am J Clin Nutr* **78,** 31-39.
32. Luscombe-Marsh ND, Noakes M, Wittert GA, Keogh JB, Foster B and Clifton PM (2005). Carbohydrate restricted diets high in either monounsaturated fat or protein are equally effective at promoting fat loss and improving blood lipids. *Am J Clin Nutr* **81,** 762-772.
33. Ryan AS (2000). Insulin resistance with aging: effects of diet and exercise. *Sports Med* **30,** 327-346.
34. Swinburn B and Egger G (2002).Preventive strategies against weight gain and obesity**.** *Obes Rev* **3:**289-301.
35. Hu FB, Li TY, Colditz GA, Willett WC and Manson GE (2003). Television watching and other sedentary behaviours in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA* **289**, 1785-1791.
36. Frey-Hewitt B, Vranizan KM, Dreon DM and Wood PD (1990). The effect of weight loss by dieting or exercise on resting metabolic rate in overweight men. *Int J Obes* **14,** 327-334.
37. Ross R, Janssen I , Dawson J, Kungl AM, Kuk JL, Wong SL, Nguyen-Duy TB, Lee S, Kilpatrick K and Hudson R (2004). Exercise induced reduction in obesity and insulin resistance in women: a randomised controlled trial. *Obes Res* **12,** 789-798.
38. Donnelly JE, Blair SN, Jakicic JM, Manore MM, Rankin JW and Smith BK (2009). Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Med Sci in Sports and Exercise* **41** 459-471.
39. Shaw K, Gennat H, O'Rourke P and Del Mar C (2006). Exercise for overweight or obesity. *Cochrane Database Syst Rev*. CD003817.
40. Grediagin MA, Cody M, Rupp J. Benardot D and Shern R (1995). Exercise intensity does not affect body composition change in untrained, moderately overfat women. *J. Am Diet. Assoc* **95**, 661-665.
41. Duncan JJ, Gordon NF and Scott CB (1991). Women walking for health and fitness; how much is enough? *JAMA* **266***,* 3295-3299.
42. Department of Health and Children and Health Service Executive (2009). The National Guidelines for Physical Activity Ireland.www.getirelandactive.ie

43. Institute of Medicine (2002). Dietary Reference Intakes for Energy, Carbohydrates, Fiber, Fat, Protein and Amino Acids (Macronutrients): A Report of the Panel on Macronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Washington, DC: National Academies Press.
44. Sykes K, Choo LL and Cotterrell M (2004). Accumulating aerobic exercise for effective weight control. *J R Soc Health* **124**, 24-28.
45. Walberg JL (1989). Aerobic exercise and resistance weight-training during weight-reduction: implications for obese persons and athletes. *Sports Med* **7,** 343-356.
46. Byrne HK and Wilmore JH (2001). The effects of a 20 week exercise training program on resting metabolic rate in previously sedentary, moderately obese women. *Int J Sport Nutr Exerc Metab* **11,** 15-31.
47. Wadden TA (1997). Exercise in the treatment of obesity: effects of four interventions on body composition, resting energy expenditure, appetite, and mood. *J Consult Clin Psychol* **65**, 269-77.
48. Geliebter A., Maher MM, Gerace L. Gutin B., Heymsfield SB. and Hashim SA(1997). Effects of strength or aerobic training on body composition, resting metabolic rate, and peak oxygen consumption in obese dieting subjects. *Am. J. Clin. Nutr.* **66**, 557-563.
49. Kraemer WJ, Volek JS, Clarke KL, Gordon SE, Puhl SM, McBride JM, Triplett-McBride NT, Putukain M, Newton RU, Hakkinen K, Bush JA and Sebastianelli WJ(1999). Influence of exercise training on physiological and performance changes with weight loss in men. *Med Sci Sports Exerc* **31,** 1320-1329.
50. Kraemer WJ, Volek JS, Clarke KL, Gordon SE, Incledon T, Puhl SM, Triplett-McBride NT, McBride JM, Putukain M and Sebastianelli WJ(1997). Physiological adaptations to a weight loss dietary regimen and exercise program in women *J App Phys* **83,** 270-279.
51. Thompson WG, Holdman NR, Janzow DJ, Slezak JM, Morris KL and Zemel MB (2005). Effect of energy reduced diets high in dairy products and fiber on weight loss in obese adults. *Obes Res* **13,** 1344-1353.
52. Wing RR, Acton KJ, Birch LL, Jakicic JM, Sallis JF, Smith-West D, Jeffery RW and Surwit RS (2001). Lifestyle changes related to obesity, eating behaviour and physical activity. *Diacare* **24,**117-123.
53. Svendsen OL, Hassager C and Christiansen C (1993). Effect of an energy-restrictive diet, with or without exercise, on lean tissue mass, resting metabolic rate, cardiovascular risk factors and bone in overweight postmenopausal women *Am J Med* **95,** 131-140.
54. Janssen I, Fortier A, Hudson R and Ross R (2002). Effects of an energy restrictive diet with or without exercise on abdominal fat, intermuscular fat, and metabolic risk factors in obese women. *Diacare* **25,** 431-438.
55. Larson-Meyer DE, Heilbronn LK, Redman LM, Newcomer BR, Frisard MI, Anton S, Smith SR, Alfonso A, Ravussin E and The Pennington Calerie team (2006). Effect of calorie restriction with or without exercise on insulin sensitivity, β-Cell function, fat cell size and ectopic lipid in overweight subjects. *Diacare* **29,** 1337-1344.
56. Curioni CC and Lourenço PM (2005). Long-term weight loss after diet and exercise: a systematic review. *Int J Obes* **29,** 1168-1174.
57. Dansinger ML, Gleason JA, Griffith JL, Selker HP and Schaefer EJ (2005). Comparison of the Atkins, Ornish, Weight Watchers and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *JAMA* **293,** 43-53.
58. Heshka S, Anderson JW, Atkinson RL, Greenway FL, Hill JO, Phinney SD, Kolotkin RL, Miller-Kovach K and Pi-Suyner FX (2003). Weight loss with self- help compared with a structured commercial program; a randomized trial. *JAMA* **289**, 1792-1798.
59. Gracey D, Stanley N, Burke V, Corti B and Beilin LJ (1996). Nutritional knowledge, beliefs and behaviours in teenage school children. *Heal Ed Res* **11,** 187-204.
60. Hesketh K, Waters E, Green J, Salmon L and Williams J(2005). Healthy eating, activity and obesity prevention: a qualitative study of parent and child perceptions in Australia *Heal Prom Int* **20,** 19-26.
61. Dishman RK, Motl RW, Saunders R, Felton G, Ward DS, Dowda M and Pate RR (2004). Self efficacy partially mediates the effect of a school based physical activity intervention among adolescent girls *Prev Med* **38,** 628-636.

62. Rosenstock IM, Strecher VJ and Becker MH (1988). Social learning theory and the Health Belief Model. *Health Educ Q* **15,** 175-183.
63. Johnson MF, Nichols JF, Sallis JF, Calfas KJ and Hovell MF (1998). Interrelationships between physical activity and other health behaviours among university women and men *Prev Med* **27,** 536-544.
64. Pate RR, Heath GW, Dowda M and Trost SG (1996). Associations between physical activity and other health behaviours in a representative sample of US adolescents *Am J Pub Health* **86,** 1577-1581.
65. Gillman MW, Pinto BM, Tennstedt S, Glanz K, Marcus B and Friedman RH(2001). Relationships of physical activity with dietary behaviours among adults. *Prev Med* **32,** 295-301.
66. Rhew I, Yasui Y, Sorensen B, Ulrich CM, Neuhouser ML, Tworoger SS, Chubak J, Bowen DJ and McTiernan A (2007). Effects of an exercise intervention on other health behaviours in overweight/obese post-menopausal women *Contemp Clin Trials* **28,** 472-481.
67. Patterson RE, Haines PS and Popkin BM (1994). Health lifestyle patterns of US adults *Prev Med* **23,** 453-460.
68. Reeves MJ and Rafferty AP (2005). Healthy lifestyle characteristics among adults in the United States, 2000 *Arch Inter Med* **165,** 854-857.
69. Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG and Willett WC (2001). Diet, lifestyle and the risk of type 2 diabetes mellitus in women. *N Engl J Med* **345**, 790-797.
70. Stampfer MJ, Hu FB, Manson JE, Rimm EB and Willett WC. (2000). Primary prevention of coronary heart disease in women through diet and lifestyle. *N Engl J Med* **343;**16-22.
71. Wadden TA and Foster GD (2000). Behavioural treatment of obesity *Med Clin N Amer* **84,** 441-461.
72. Ash S, Reeves MM, Bauer JD, Dover T, Vivanti AP, Leong C K, O'Moore-Sullivan Tand Capra SM (2006) A randomised controlled trial comparing lifestyle groups, individual counselling and written information in the management of weight and health outcomes over 12 months *Int J Obes* **30,** 1557-1564.
73. Mulvihill C and Quigley R (2003). The management of obesity and overweight: An analysis of reviews of diet, physical activity and behavioural approaches. Health Development Agency, London. www.had.nhs.uk/documents/obesity_evidence_briefing.pdf
74. Lobstein T, Baur L and Uauy R for International Obesity Taskforce (2004). Obesity in children and young people: a crisis in public health *Obes Rev* **5,** 4-85.
75. Wilson P, O Meara S, Summerbell C and Kelly S (2003). The prevention and treatment of childhood obesity *Qual Saf Health Care* 12, 65-74.
76. World Health Organisation (2007) The challenge of Obesity in the WHO European Region and the strategies for response. Copenhagen: WHO, Regional Office for Europe. .http://www.euro.who.int/document/e89858.pdf
77. World Health Organisation (2002). The world health report: reducing risks, promoting healthy life. WHO, Geneva.
78. Moore LL, Visioni AJ, Qureshi MM, Bradlee ML, Ellison RC and D'Agostino R (2005). Weight loss in overweight adults and the long term risk of hypertension. The Framingham Study *Arch Intern Med* **165***,* 1298-1303.
79. Lloyd-Jones DM, Liu K, Colangelo LA, Yan LL, Klein L, Loria CM, Lewis CE and Savage P(2007). Consistently stable or decreased body mass index in young adulthood and longitudinal changes in metabolic syndrome components: the Coronary Artery Risk Development in Young Adults Study. *Circulation* **115**, 1004-1011.
80. Norman JE, Bild D, Liu K and West SD(2003). The impact of weight change on cardiovascular disease risk factors in young black and white adults: the CARDIA study. *Int J Obes*, 27, 369-376.
81. Truesdale KP, Stevens J and Cai J (2007).Nine-year changes in cardiovascular disease risk factors with weight maintenance in the Atherosclerosis Risk in Communities Cohort *Am J Epidemiol* **165**, 890-900.
82. Truesdale KP, Stevens J, Lewis CE, Schreiner PJ, Loria CM and Cai J (2006). Changes in risk factors for cardiovascular disease by baseline weight status in young adults who maintain or gain weight over 15 years: the CARDIA study. *Int J Obes* **30**, 1397-1407.
83. Truesdale KP, Stevens J and Cai J (2005). The effect of weight history on glucose and lipids: the Atherosclerosis Risk in Communities Study *Am J Epidemiol* **161**, 1133-1143.

84. Schubert CM, Rogers NL, Remsberg KE, Sun SS, Chumlea WC, Demerath EW,Czerwinski SA,Towne B and Siervogel RM(2006). Lipids, lipoproteins, lifestyle, adiposity and fat-free mass during middle age: the FELS Longitudinal Study. *Int J Obes* **30**,251-260.
85. Siervogel RM, Wisemandle W, Maynard LM, Guo SS, Chumlea WC and Towne B (2000). Lifetime overweight status in relation to serial changes in body composition and risk factors for cardiovascular disease: the FELS Longitudinal Study. *Obes Res* **8,** 422-430.
86. Siervogel RM, Wisemandle W, Maynard LM, Guo SS, Roche AF, Chumlea WC and Towne B (1998). Serial changes in body composition throughout adulthood and their relationships to changes in lipid and lipoprotein levels. The FELS Longitudinal Study. *Arterioscler Thromb Vasc Biol* **18**,1759-1764.
87. Donnelly JE, Jacobsen DJ, Snyder Heelan KA, Seip R and Smith S (2000). The effects of 18 months of intermittent vs continuous exercise on aerobic capacity, body weight and composition, and metabolic fitness in previously sedentary, moderately obese females *Int J Obes Relat Metab Disord* **24**, 566-572.
88. Janssen I, Katzmarzyk PT & Ross R. (2004) Duration of overweight and metabolic health risk in American men and women. *Ann. Epidemiol.* **14**: 585−591**.**
89. Farooqi S and O' Rahilly S (2006). Genetics of obesity in humans *Endocr Rev* **27,**710-718.
90. Bauer F, Elbers CC, Adan RAH, Loos RJF, Onland-Moret NC, Grobbee DE, Van Vliet-Ostaptchouk JV, Wijmenga C and Van der Schouw YT(2009). Obesity genes identified in genome-wide association studies are associated with adiposity measures and potentially with nutrient-specific food preference *Am J Clin Nutr* **90,** 951-959.
91. Bouchard C, Tremblay A and Despres JP, Theriault G, Nadeau A, Lupien PJ, Moorjani S, Prudhomme D and Fournier G (1994). The response to exercise with constant energy intake in identical twins *Obes Res* **5,** 400-410.
92. U.S. Physical Activity Guidelines Advisory Committee (2008). 2008 Physical activity guidelines for Americans. www. health.gov/paguidelines
93. Department of Health and Ageing (2005). The National Physical Activity Guidelines for Australians. www.health.gov.au
94. Public Health Agency of Canada (2002). Canada's physical activity guide to healthy active living. www.phac-aspc.gc.ca/pau-uap/paguide