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ECSS position statement: Exercise and obesity

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ORIGINAL ARTICLE

ECSS position statement: Exercise and obesity

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Abstract

This is a review on the effects of physical exercise on weight, body composition and health in prevention and management of obesity. Individuals who maintain or increase their physical activity also show the best weight control over a period of several years. However, physical activity at baseline of prospective studies does not consistently predict weight change, perhaps because of changes in physical activity during the follow-up. Moderate-intensity (e.g. walking) physical activity during diet-induced weight reduction leads to modest improvements in weight, abdominal fat and total fat loss. Moderate exercise 250–300 min/wk is recommended for weight reducing purposes. For maintenance of reduced body weight, more than 60 min moderate exercise per day after weight reduction might be needed. Studies have found a more pronounced training-induced loss of visceral compared to subcutaneous fat in overweight and obese subjects. Physical activity will help to lower risk of insulin resistance, type 2 diabetes, dyslipidemia, hypertension and the metabolic syndrome in overweight and obese individuals and that more pronounced effects can be attained in combination with maintained weight loss. The intensity of physical activity should be at least moderate (>30–40% VO₂max) to cause metabolic health benefits. A suitable frequency for moderate-intensity physical activity seems to be daily, and for vigorous activity every other day.

Introduction

The prevalence of obesity has increased rapidly throughout the world during the past 20 years (International Obesity Task Force, 1998). In Europe, the prevalence of obesity in adults varies between less than 10 to more than 30% (International Obesity Taskforce, 2003). Among European 13- and 15-year-old adolescents, the prevalence of overweight varies between 5 and 20%, depending on country, sex and age (Lissau et al., 2004). The highest prevalence is seen in South-East Europe in adults, as well as in adolescents (International Obesity Taskforce, 2003; Lissau et al., 2004). Obesity is related to an increased risk of many chronic diseases, namely cardiovascular heart diseases, type 2 diabetes, hypertension, stroke and cancers in colon and breast (International Obesity Taskforce, 1998). The risk of all of these diseases can be lowered by increased physical activity (US Department of Health and Human Services, 1996). Therefore, physical activity may prevent obesity-related diseases by two mechanisms, that is,

by improving weight control (prevention of weight gain, increased weight loss) and by improving the metabolic profile associated with obesity.

Physical activity and primary prevention of weight gain

A systematic analysis on the associations between physical activity and weight change has been carried out (Fogelholm & Kukkonen-Harjula, 2000). This review identified sixteen prospective, observational studies. The mean duration of the follow-up was approximately 7 years, with a range from 2 to 21 years. The outcomes were grouped according to when physical activity data were collected, that is, whether baseline, follow-up or change (from baseline to follow-up) in physical activity was compared against change in weight. Results from the studies using baseline physical activity data were inconsistent. Three studies (Klesges et al., 1992; Owens et al., 1992; Haapanen et al., 1997) reported that a large volume of physical activity predicted smaller weight change. High baseline work activity was

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associated with less weight gain in one study (Klesges et al., 1992). In contrast, two studies reported that a large volume of vigorous physical activity at baseline was associated with greater weight gains (Klesges et al., 1992; Bild et al., 1996). Finally, three studies did not find a significant association between baseline total physical activity (Williamson et al., 1993; Parker et al., 1997), or TV/VCR watching (Crawford et al., 1999), and the magnitude of weight change.

The results with physical activity data at follow-up were more consistent: four studies found that a large volume of physical activity or exercise (Haapanen et al., 1997; Williamson et al., 1993; Rissanen et al., 1991; Barefoot et al., 1998) at follow-up was associated with less weight gain. Only one study (Heitmann et al., 1997) did not find such an association. Many studies used data on physical activity from both baseline and follow-up. About half of the studies reported that an increase in physical activity was associated with less weight gain (Owens et al., 1992; Haapanen et al., 1997; Williamson et al., 1993; Taylor et al., 1994; Coakley et al., 1998; Guo et al., 1999; Fogelholm et al., 2000a).

In conclusion, individuals who maintain or increase their physical activity also show the best weight control over a period of several years. Physical activity at baseline of prospective studies does not consistently predict weight change, perhaps because of changes in physical activity during the follow-up. Unfortunately, the prospective studies did not allow quantification of, for example, time and/or energy expenditure needed for prevention of weight gain.

Physical activity for weight reduction

A recent systematic review (Fogelholm, 2005) looked at the effects of walking on weight reduction in obese participants, with or without an energy-restricted diet. Most of the weight-reduction studies used a low-energy diet (5–7 MJ/day or 1200–1700 kcal/day), that is, the comparison was between diet only vs. diet+walking. The prescribed amount of walking varied between 150 and 225 min/wk, except for some studies with “high amount” (300 min/wk) of walking. In all studies the mean weight reduction (after 3–6 months’ treatment) was numerically better (by 0.3–2.1 kg) when walking was added to diet, but due to a large inter-individual variation this difference was not statistically significant. In earlier systematic reviews (Garrow & Summerbell, 1995; Wing, 1999) a combined exercise and diet group showed approximately 2 kg better weight reduction than the diet only group. Hence, a physical activity program, added to a low-energy diet and with prescribed total duration

corresponding to 150–225 min/wk brisk walking, is expected to improve the average weight reduction by 1–2 kg during 3–6 months.

The difference between walking only vs. control group, without added diet, seems to be as large (approximately 2 kg) as for diet+exercise vs. diet (Fogelholm, 2005). Again, because of large inter-individual variation, the between group differences were not usually significant. According to earlier reviews (Garrow & Summerbell, 1995; Wing, 1999; Ross & Janssen, 2001) the expected weight loss with 150–200 min of any moderate exercise should be approximately 3 kg in studies with 3–6 months’ duration.

Observational studies show that physical activity is efficacious for the management of obesity, if enough exercise is undertaken. Jeffery et al. (2003) reported that the actual change in total physical activity was positively correlated with weight reduction. Two other studies (Irwin et al., 2003; Jakicic et al., 2003) used post hoc analyses to quantitatively relate weight reduction with completed walking. Both studies showed a clear dose-response relation with the largest reduction in weight and total body fat in subjects walking more than 195 (Irwin et al., 2003) or 220 (Jakicic et al., 2003) min/wk.

One important study with walking solely in a fitness center needs to be mentioned: Kraus et al. (2002) found that walking or jogging 19 km/wk did not result in a significant weight change (–0.4 kg) in overweight and obese subjects, whereas 32 km/wk jogging did (–1.5 kg). This happened despite the fact that the participants were asked to maintain stable weight. The lower and higher amount corresponds to approximately 190 and 320 min/wk walking, respectively.

Studies comparing different kinds of physical activity (walking) prescriptions (long vs. short bouts; walking integrated freely into daily routines vs. structured and planned walking; at home or group-based) have been assessed in systematic reviews (Fogelholm, 2005; Hardman, 2001). The prescribed duration for walking was 90–200 min/wk. Fractionization (splitting the daily dose into 10–20-min periods instead of 30 min or longer) had a similar effect on weight loss than exercising one longer bout daily.

In conclusion, moderate-intensity (e.g. walking) physical activity during diet-induced weight reduction leads to modest improvements in weight, abdominal fat and total fat loss. However, if the prescribed level is 150–200 min/wk, the mean response is not very often significantly different from zero. Hence, 250–300 min/wk (or 35–45 min daily) seems more suitable for weight reducing purposes. This recommendation is close to the 45 min/day recommended in a report from an expert panel

(Saris et al., 2003). Splitting the daily physical activity into multiple shorter (10–20 min) periods leads to a weight loss similar to that achieved by walking an identical daily duration in one single period.

Assuming basal energy expenditure (BEE) to be 1 kcal/kg per hour and the intensity of brisk walking to 5 METs (5 times BEE) [Ainsworth et al., 2000], 150-min walking for a 70-kg individual corresponds to an energy expenditure of $2.5 \text{ h} \times 5 \text{ kcal}/(\text{kg} \times \text{h}) \times 70 \text{ kg} = 875 \text{ kcal}$, $200 \text{ min} = 1170 \text{ kcal}$, $250 \text{ min} = 1460 \text{ kcal}$ and $300 \text{ min} = 1750 \text{ kcal}$. Roughly speaking an exercise-induced weekly energy expenditure of 1000 kcal (4.2 MJ) is not enough for weight reducing purposes, whereas 1500 kcal (6.3 MJ) is the minimum recommendation.

Physical activity for maintenance of reduced body weight

Weight reduction is typically successful up to 6 months, regardless of whether the technique is diet, (Wing et al., 1998; Willett, 2003) exercise (Jakicic et al., 2003) or a combination (Jeffery et al., 2003; Wing et al., 1998). Unfortunately, maintenance of the reduced body weight is difficult and a majority of weight-reduced individuals will return towards or even beyond their initial body weight within a few years (Fogelholm & Kukkonen-Harjula, 2000).

A few researchers have studied the effects of prescribed walking, as a part of a weight-maintenance intervention, on weight regain after substantial weight loss (for systematic reviews, see Fogelholm & Kukkonen-Harjula, 2000; Fogelholm, 2005). The prescribed duration of walking varied between 150 and 300 min/wk. In one study (Fogelholm et al., 2000b), the lower dose (150 min/wk) prevented weight gain better (3.5 kg or 130 g/month) than the control treatment, whereas a higher dose (300 min/wk) was not different from the control. Borg et al. (2002) did not find a difference between walking (about 225 min/wk), resistance training or control intervention. Finally, a weight-focused intervention was significantly (2.1 kg or 350 g/month) better than an exercise-focused (mostly walking, 150 min/wk) program (Leermakers et al., 1999).

Several observational studies and post hoc analyses have shown a positive dose-response between the amount (energy expenditure or duration) of physical activity and weight-loss maintenance (for a systematic review, see Fogelholm & Kukkonen-Harjula, 2000). Some of these studies included walking as the main component of physical activity (Jakicic et al., 2003; Fogelholm et al., 2000b; Borg et al., 2002; Leermakers et al., 1999; Andersen et al., 1999). The results are clear: the greater the duration

or energy expenditure of completed walking (min/week or kJ/week), the better the maintenance of reduced body weight.

Some studies have estimated the amount of total exercise (Andersen et al., 1999; McGuire et al., 1999) or walking (Borg et al., 2002), which is associated with weight-loss maintenance. The results are surprisingly similar: an average exercise energy expenditure of 9–10 MJ/wk (2200–2400 kcal/wk), corresponding to walking 70–80 min/day, seems to be associated with stable weight ($\pm 1 \text{ kg}$ for more than 1 year) after substantial weight reduction. These estimates corroborate with the recommendation by Saris et al. (2003). However, it should be noted that a smaller amount of walking may slow down, although not prevent, weight regain (Fogelholm & Kukkonen-Harjula, 2000; Jeffery et al., 2003; Fogelholm et al., 2000b).

In conclusion, the expected weight loss with 150–200 min/wk of moderate-intensity exercise should be 2–3 kg in studies with 3–6 months' duration. Hence, 250–300 min/wk (or 35–45 min daily) seems more suitable for weight reducing purposes. Splitting the daily walking into multiple shorter (10–20-min) periods leads to a similar weight loss compared with exercising an identical daily duration in one single period. For maintenance of reduced body weight, more than 60 min of moderate exercise per day after weight reduction might be needed.

The role of physical activity in adipose tissue metabolism and loss of subcutaneous vs. visceral fat in obese persons

Exercise and adipose tissue metabolism

An acute bout of exercise increases hydrolysis of triacylglycerol (lipolysis) in adipose tissue as well as oxidation of lipid in skeletal muscle in lean and obese persons (Horowitz & Klein, 2000; Stich et al., 2000; van Hall et al., 2002; Richterova et al., 2004; Mittendorfer et al., 2004). Comparisons of adipose tissue metabolism between obese and lean subjects are difficult to make due to the inherent difference in adipose tissue mass, but data indicate that sub-maximal exercise increases adipose tissue lipolysis per kg of adipose tissue less in obese subjects than in lean subjects (Mittendorfer et al., 2004). However, due to the higher amount of adipose tissue in the obese subjects, the exercise-induced increase in total adipose tissue lipolysis is generally not blunted in obese subjects. In situ studies suggest that acute exercise in lean subjects stimulates subcutaneous, abdominal adipose tissue lipolysis more in women than in men (Arner et al., 1990; Hellström et al., 1996), but it is not clear, whether this difference is also present in obese subjects.

During exercise, adipose tissue lipolysis is stimulated by an increased sympatho-adrenergic activity, and chronic endurance training has been found to increase the in situ lipolytic sensitivity to catecholamines in subcutaneous, abdominal adipose tissue of obese men (Stich et al., 1999), whereas no change has been found in obese women (Richterova et al., 2004). After a period of training subjects are able to exercise at a higher absolute intensity and probably due to this, in situ lipolysis has been found to be higher after training in obese women (Richterova et al., 2004) as well as in overweight (de Glizezinski, et al., 2003) and obese (Stich et al., 1999) men.

Exercise and fat loss

The increase in adipose tissue lipolysis in response to acute as well as chronic endurance exercise indicates that endurance training could be able to promote a loss of adipose tissue in obese subjects, and indeed this has been shown to be the case (Ross & Janssen, 2001; Irwin et al., 2003; Donnelly, et al., 2003; Gutin et al., 2002; Slentz et al., 2004; Gan et al., 2003; Ross et al., 2000, 2004). Also, it has been found in short-term studies (≤ 16 weeks) that fat loss increases with amount of energy spent on exercise (Ross & Janssen, 2001; Irwin et al., 2003; Slentz et al., 2004). Exercise-training-induced fat loss is usually more prominent in men compared with women (Donnelly et al., 2003) and varies positively with initial body fat levels (Ballor & Keeseey, 1991). This might be due a greater compensation in energy intake by women compared with men and by lean compared with obese people.

In situ studies suggest that acute exercise in lean subjects stimulates adipose tissue lipolysis more in subcutaneous abdominal than femoral/gluteal adipose tissue (Arner et al., 1990; Horowitz et al., 2000). It is not clear, if this site difference is also present in obese subjects. Lipolytic sensitivity towards catecholamines is higher in intra-abdominal than in subcutaneous adipose tissue, and this could indicate that acute exercise would stimulate lipolysis more in visceral compared with subcutaneous adipose tissue. Visceral adipose tissue lipolysis is indeed stimulated during acute exercise (van Hall et al., 2002), but the relative increase in visceral compared with subcutaneous adipose tissue lipolysis during exercise has not been thoroughly evaluated.

Loss of subcutaneous abdominal fat versus limb fat

As potential site differences in adipose tissue lipolysis exist, exercise training could influence fat depots differently, and this has been evaluated in several recent, well-controlled studies, where previously sedentary overweight and obese subjects performed

endurance training but did not diet. In one study an 8-month training program of middle-aged men and women reduced subcutaneous abdominal and limb skinfolds to the same extent ("low amount" ~ 1250 kcal/week: $\sim 9\%$; "high amount" ~ 2000 kcal/week: $\sim 19\%$) (Slentz et al., 2004). In another study men participating in a 10-week training program (~ 160 min/week of moderate intensity exercise) showed a decrease in limb fat mass by $\sim 3\%$ and a decrease in abdominal fat mass by $\sim 5\%$ as determined by DXA scanning (Gan et al., 2003). Magnetic resonance imaging (MRI) further showed, that both the visceral and the subcutaneous, abdominal adipose tissue was reduced by $\sim 5\%$.

In conclusion, according to the mentioned as well as previous studies in obese and overweight subjects (Ross et al., 2002), there does not seem to be a large variation in training-induced loss of subcutaneous abdominal fat and limb fat.

Loss of visceral fat versus subcutaneous fat

In other recent studies the focus has been on the relative loss of visceral versus subcutaneous adipose tissue. A 3-month training program (4900 kcal/week) in men decreased visceral adipose tissue mass by ~ 1 kg ($\sim 28\%$) and whole-body subcutaneous adipose tissue mass by ~ 4 kg ($\sim 17\%$) (Ross et al., 2000). The relative reduction in visceral adipose tissue was significantly higher than the relative reduction in subcutaneous adipose tissue. Similarly, a 3-month training program (3500 kcal/week) in women decreased visceral adipose tissue mass by a little less than 1 kg ($\sim 30\%$) and whole-body subcutaneous adipose tissue mass by ~ 6 kg ($\sim 15\%$) (Ross et al., 2004). In another study young men and women were randomized to either a 16-month training program consisting mainly of walking (2000 kcal/week) or to continue their sedentary living (Donnelly et al., 2003). In women the training program induced a significantly higher loss of total and subcutaneous abdominal fat in the intervention group than in the control group, but a significant change in visceral fat was not seen. In men a significant change in abdominal fat was not seen.

In another study obese adolescents were randomized to either physical training and lifestyle education or lifestyle education alone for 8 months, and those who trained ≥ 2 days/week (≥ 500 kcal/week) lost a significantly higher amount of whole body and visceral fat, but not subcutaneous abdominal fat, than the control group (Gutin et al., 2002). Furthermore, in postmenopausal women a 12-month training program (~ 175 min/week of moderate intensity exercise) reduced abdominal subcutaneous (5%) as well as visceral (6%) fat significantly (Irwin et al., 2003).

In conclusion, it is apparent that the picture, with regards training-induced loss in visceral compared to subcutaneous fat, is not entirely clear, but many of the mentioned as well as previous studies (Ross et al., 2002) found a more pronounced training-induced loss of visceral compared to subcutaneous fat in overweight and obese subjects.

Physical activity and comorbidities of obesity

Overweight and obesity, and visceral fat in particular, are associated with a considerably increased risk of co-morbidities, such as type 2 diabetes, hypertension and cardiovascular disease (National Institutes of Health and National Heart, Lung and Blood Institute, 1998). In 1999 the role of physical activity in the prevention and treatment of these co-morbidities was reviewed by a consensus committee of the American College of Sports Medicine and a consensus statement was published (Grundy et al., 1999). Since then, additional randomized controlled trials (RCTs) and meta-analyses on this topic have been published. These will be included in this report and conclusions based on the currently available evidence will be drawn.

Insulin resistance, impaired glucose tolerance and type 2 diabetes

The ACSM consensus committee stated in 1999 that physical activity improves insulin action, and thus reduces insulin resistance in obese subjects. Alone or in combination with weight loss it retards the transition from impaired glucose tolerance to type 2 diabetes (Grundy et al., 1999). This statement was based on a limited number of RCTs and mostly based on non-randomized studies (Kelley & Goodpaster, 1999). Since then, several RCTs on the effects of exercise on insulin resistance in overweight and obese subjects have been published.

Two RCTs by Ross and colleagues in overweight and obese men (BMI > 27 kg/m²; n = 52) (Ross et al., 2000) and women (n = 54) (Ross et al., 2004) compared the effects of 12 weeks of exercise (700 [men] or 500 [women] kcal/day of brisk walking or jogging) with that of diet-induced weight loss or control. They found that similar exercise- and diet-induced weight loss (8%) improves insulin sensitivity in overweight and obese men and women to a similar extent, but exercise without weight loss had no effect. Insulin sensitivity was measured 4 days after the last exercise bout and thus the short-lived effects of an acute exercise bout (1–3 days) were not measured.

McAuley et al. (2002) performed an RCT in 79 overweight insulin-resistant men and women comparing a moderate (according to current guidelines)

and an intensive (low saturated and total fat intake, high fiber intake, exercise at least 5 × per week for 20 min at 80–90% of maximal heart rate) lifestyle modification including diet and exercise on insulin sensitivity. Both groups lost a comparable amount of weight (3–5 kg) and waist circumference after 4 months' intervention, but insulin sensitivity was only improved significantly in the intensive intervention group.

An RCT in 154 sedentary overweight and obese (25–35 kg/m²) subjects by Houmard et al. (2004) compared three groups with different volumes and intensities of exercise (12 miles/wk walking at 40–55% VO₂peak, 12 miles/wk jogging at 65–80% VO₂peak or 20 miles/wk jogging at 65–80% VO₂peak) and a control group after a 6-month intervention. Weight loss was kept at a minimum, but was not completely prevented. Insulin sensitivity was measured approximately 24 h after the last exercise bout. Insulin sensitivity increased in all three intervention groups. The increase was larger with longer total exercise duration.

Stewart et al. (2005) studied in an RCT in 115 overweight and obese men and women between 55 and 75 years, the effect of a 6-month exercise training program (exercise sessions 3 × per week according to ACSM guidelines) on insulin sensitivity. Body weight and body fat were significantly reduced, but no change in insulin sensitivity was found. However, changes in abdominal body fat were negatively correlated with changes in insulin sensitivity.

Three large RCTs, the Da Qing trial, the Finnish Diabetes Prevention Study (DPS) and the Diabetes Prevention Program Research trial, have provided convincing evidence that lifestyle intervention, including regular exercise and moderate diet-induced weight loss, can prevent or delay progression to type 2 diabetes in subjects with impaired glucose tolerance, who are usually also overweight or obese (Pan et al., 1997; Lindstrom et al., 2003; Knowler et al., 2002). In addition the Da Qing trial showed that this effect was similar with an exercise intervention alone and that the effect was the same in lean and overweight individuals (Pan et al., 1997). A secondary analysis of the Finnish DPS trial showed that a higher level of leisure time physical activity reduced the risk of developing type 2 diabetes independent of changes in diet or body weight (Laaksonen et al., 2005).

In conclusion, weight loss improves insulin sensitivity in overweight and obese individuals independent of the way the weight loss is attained (diet or exercise). An acute exercise bout also improves insulin sensitivity, but this effect is short-lived (1–3 days) and therefore requires very regular exercise. Consistent evidence that exercise training alone has

a more long-lasting effect on insulin sensitivity is lacking. Regular exercise prevents or delays the progression to type 2 diabetes in overweight and obese individuals with impaired glucose tolerance even in the absence of changes in body weight.

Dyslipidemia

In 1999 it was concluded that in order to raise HDL cholesterol and reduce triglyceride concentrations by physical activity in overweight and obese individuals, a moderate weight loss of at least 4.5 kg has to be attained (Grundey et al., 1999). Beneficial effects on LDL are only seen in combination with weight loss induced by a low fat weight-reducing diet.

Two meta-analyses have recently been published on this topic (Carroll & Dudfield, 2004; Kelley et al., 2005). Conclusion of the meta-analysis by Carroll and Dudfield (Carroll & Dudfield, 2004), based on 15 RCTs, is that longer term regular supervised exercise training of moderately vigorous intensity, even in the absence of clinically significant weight loss (<5% of body weight) is associated with modest improvements in the dyslipidemic profile, by raising HDL cholesterol (+4.6%) and lowering triglycerides (TG) (-21%) among middle-aged and older overweight/obese adults, but that exercise alone may not be sufficient to normalize the atherogenic dyslipidemia associated with the metabolic syndrome.

Based on their meta-analysis including 13 RCTs in overweight or obese subjects, which included only three of the RCTs from the meta-analysis of Carroll and Dudfield, Kelley et al. (2005) conclude that aerobic exercise decreases TG (-11%) in overweight and obese adults independent of changes in body weight. However, the authors mention that there is no consensus to support a reduction of cardiovascular risk with the reduction of TG observed. LDL and HDL cholesterol changes were not significant, but they were associated with changes in body weight.

In conclusion, exercise reduces TG and possibly increases HDL levels in overweight and obese subjects even in the absence of significant weight loss. Reduction in LDL concentration, which is currently the primary target for therapy in dyslipidemia, appears to be dependent on weight loss.

Hypertension

The evidence, that aerobic exercise reduces blood pressure independent of weight loss and initial body mass index, was already strong in 1999 (Grundey et al., 1999). The mean blood pressure reduction was -3.4/-2.4 mmHg, but was larger in hypertensive than normotensive individuals. Adding exercise

to an energy-reduced diet did not reduce blood pressure significantly more than diet alone. The effect of exercise alone is often not large enough to fully normalize blood pressure in patients with hypertension. Additional RCTs since 1999 have not changed this general picture.

Metabolic syndrome

The metabolic syndrome is a cluster of risk factors, including abdominal obesity, hypertriglyceridemia, low HDL, hypertension and insulin resistance, associated with increased risk of type 2 diabetes and cardiovascular disease (Roberts & Barnard, 2005). In the US 4.6, 22.4, and 59.6% of normal weight, overweight and obese men have the metabolic syndrome (Park et al., 2003). Physical inactivity in people with the metabolic syndrome is associated with an even greater risk.

The Oslo Diet and Exercise study is an RCT in 219 men and women with the metabolic syndrome (BMI >24 kg/m² and elevated diastolic blood pressure, triglycerides, total cholesterol and low HDL cholesterol) and compared the effects of a 1-year intervention (exercise alone, exercise plus energy-restricted diet, energy restriction alone or control) on changes in insulin resistance. The exercise intervention did not change insulin resistance, but it was reduced in both energy-restricted groups (Torjesen et al., 1997).

In an RCT, Watkins et al. (2003) studied the effect of a 6-month intervention with exercise only (3-4 times per week for 35 min at 70-85% HRR) or diet and exercise in 53 subjects with the metabolic syndrome. Body weight reduction was more pronounced in the diet and exercise group compared to the exercise only group and so was insulin resistance. Diastolic blood pressure was only reduced in the diet and exercise group, plasma lipid profile was not affected by either intervention (Watkins et al., 2003).

Christ et al. (2004) performed a non-randomized study in 52 hypertensive men and women with the metabolic syndrome and compared the effects of diet-induced weight loss with or without aerobic exercise (2 times per week for 40 min at 60-80% HRR) over a period of 36 months. Both interventions reduced body weight, waist-to-hip ratio, plasma triglycerides, HbA_{1C}, and increased HDL cholesterol compared to a control group that only received blood pressure treatment with an ACE inhibitor. The addition of exercise to the diet did not result in further improvements of the metabolic parameters.

Katzmarzyk et al. (2003) performed a subanalysis of participants of the HERITAGE Family Study with the metabolic syndrome at baseline (*n* = 105). This is a non-randomized trial on the effect of

exercise (3 times per week for 30–50 min at 50–70% $\text{VO}_{2\text{max}}$) on risk factors for cardiovascular disease and type 2 diabetes in sedentary adults. After the 20-week intervention 30% of the participants with metabolic syndrome at baseline were no longer classified as having the metabolic syndrome.

A secondary analysis of the Diabetes Prevention Program RCT in 3234 subjects with impaired glucose tolerance showed that the lifestyle intervention (designed to achieve and maintain a 7% weight loss and 150 min of exercise per week) reduced the development of the metabolic syndrome among those subjects (45%) that did not have metabolic syndrome at baseline by 41% (Orchard et al., 2005).

In conclusion, the above overview shows that physical activity will help to lower risk of insulin resistance, type 2 diabetes, dyslipidemia, hypertension and the metabolic syndrome in overweight and obese individuals and that more pronounced effects can be attained in combination with maintained weight loss.

Recommendations

How much physical activity is efficacious for the management of obesity?

The recommended amount of physical activity for the management of obesity depends on several issues, such as the magnitude of desired outcome, recent history of weight change and dietary habits. The amount of 150–200 min/wk (25–30 min daily) moderate-intensity activity may improve cardiorespiratory fitness (in unfit individuals) and insulin sensitivity, but a marked effect on weight, body composition or fat distribution should not be expected in most individuals. Increasing the total duration of physical activity to about 250–300 min/wk (35–45 min daily) should bring about beneficial changes in weight, body composition and HDL-cholesterol. Possibly much more than 300 min/wk of moderate physical activity may be needed to prevent weight regain after substantial weight loss. It must be noted, however, that since weight change is a function of energy balance, the daily dietary energy intake has a substantial effect on the level of physical activity needed for weight control.

Is moderate physical activity enough for management of obesity?

An important question is if moderate physical activity is enough, or whether more vigorous activity is needed for better weight control and health. The added impact of high-intensity activity (vs. moderate intensity) on weight and insulin sensitivity seems negligible (provided energy expenditure is kept

constant) [Houmard et al., 2004]. High-intensity activity may have an additional positive effect on LDL-lipoproteins (Kraus et al., 2002; Halbert et al., 1999). Nevertheless, some cross-sectional studies show that a combination of high duration of both walking activity (or low duration of leisure inactivity) and vigorous exercise activity is optimal for weight control (Martinez-Gonzalez et al., 1999) and prevention of cardiovascular heart diseases (Manson et al., 2002). Moreover, individuals who are successful at long-term maintenance of substantial body weight loss, expend almost 30% of their weekly physical activity energy in vigorous activities, such as running (Klem et al., 1997). Some high-intensity activities might simply be needed to increase the total energy expenditure up to efficacious levels. Perhaps walking or other moderate-intensity activities as the only mode of physical activity are too time-consuming.

The recommendations are as follows:

- *Dose* (amount, volume) corresponding to an energy expenditure of 1500–1750 kcal/week (e.g. 250–300-min brisk walking weekly) during weight reduction and suggestively also for primary prevention of weight gain; 2000–2500 kcal/week (e.g. 400–500 min brisk walking weekly) may be needed to prevent weight regain after weight reduction.
- The *intensity* of physical activity should perhaps be at least moderate ($>30\text{--}40\%$ $\text{VO}_{2\text{max}}$) to cause metabolic health benefits. High intensity exercise ($>70\%$ $\text{VO}_{2\text{max}}$) may bring additional health benefits for lipid metabolism, but not for any other outcome variables.
- A suitable *frequency* for moderate-intensity physical activity seems to be daily, and for vigorous activity every other day.
- The *daily dose* can be split into shorter (10–20 min) segments without a loss of health responses.
- Lifestyle activity leads, when performed at sufficient intensity, to similar effects on weight and metabolic health as more structured (aerobic) exercise with the same intensity and volume.
- Strength (resistance) training improves muscle fitness without compromising metabolic health. Therefore, strength training is a natural component of a health-enhancing physical activity recommendation.

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