

## Review

# Exercise and the metabolic syndrome

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One of the first people to introduce the metabolic syndrome – or trisyndrome métabolique – in the scientific literature was Camus in 1966 [1]. However, this entity did not receive much interest until Reaven in 1988 introduced syndrome X, characterised by hypertension, impairment in glucose and lipid metabolism and insulin resistance [2]. The metabolic syndrome – also called the insulin resistance syndrome – is a multifaceted syndrome characterised by five major abnormalities: obesity, hypertension, insulin resistance, glucose intolerance (impaired glucose tolerance/non-insulin-dependent diabetes mellitus (NIDDM)), and dyslipidaemia (hypertriglyceridaemia and low HDL-cholesterol). In addition, a number of other abnormalities such as microalbuminuria, hyperuricaemia, hyperfibrinogenaemia and increased levels of plasminogen activator inhibitor I and low concentrations of tissue plasminogen activator are often associated with the syndrome. The clinical importance of the metabolic syndrome is mainly due to the clustering of simultaneously occurring atherosclerotic risk factors in the same individual [2–9].

The aetiology of the metabolic syndrome is poorly understood; both genetic and environmental factors are involved [2–9]. Possible common denominators for the various components of the syndrome are not known, although hyperinsulinaemia/insulin resistance are important characteristics in most of the abnormalities.

The prevalence rate of the metabolic syndrome varies depending on the population studied and the definition applied. As in any syndrome, not all features are present in the same individual. The

estimated prevalence rate of the metabolic syndrome in western countries is 25–35% [2–9]. On the other hand, both Ferrannini and Rupp [8, 9] have reported that only about one-third of an adult study population was free of all major characteristics of the metabolic syndrome. Since the prevalence of hypertension, insulin resistance and glucose intolerance usually increase with increasing age, the prevalence of the metabolic syndrome will probably also rise in the aging western society.

A number of pharmacological therapies can be used for the treatment of the metabolic syndrome; but the results of most of the available therapies are often unsatisfactory, although some trials with metformin and thiazolidinediones have been encouraging [6, 10, 11].

Low levels of physical activity are related to most components of the metabolic syndrome [12–16]. Consequently this offers a unique opportunity to employ increased physical activity in the prevention and treatment of the metabolic syndrome and its components. This review will focus on the role of physical activity and exercise in the prevention and treatment of the metabolic syndrome.

## Obesity

The prevalence of obesity (body mass index (BMI) > 27 kg/m<sup>2</sup>) is currently 25–35% in most western societies, and is increasing [17]. If the prevalence continues to increase, as during the past decades, by the year 2230, 100% of the adult United States population will be obese (BMI > 27.8 kg/m<sup>2</sup> for men and 27.3 kg/m<sup>2</sup> for women) – the ‘ultimate triumph of obesity’ will then be achieved [18]. Recent evidence suggests that an inactive modern lifestyle is a major aetiological factor in both the development and the rapid increase of obesity in many societies [19, 20].

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*Abbreviations:* BMI, Body mass index; 24hEE, 24-h energy expenditure; RMR, resting metabolic rate; TEF, thermic effect of food; NIDDM, non-insulin-dependent diabetes mellitus.

The characteristic feature of obesity associated with the metabolic syndrome is the android or abdominal type of obesity. Although obesity per se is associated with increased morbidity, the association is even stronger for truncal fat and particularly visceral fat [21, 22]. Both genetic and environmental factors contribute to the development of obesity and the distribution of body fat. The proposed contribution of genetics to obesity varies from 20–90% [23, 24].

Obesity has classically been considered a problem of overeating, a disorder primarily of energy intake. However, persuasive evidence suggests that much obesity is due to low energy expenditure, rather than to overeating [19, 20, 25–28]. The importance of energy expenditure in the treatment of obesity has recently been emphasized in studies on leptin, a satiety factor. Administration of leptin to *ob/ob* mice, with mutation in the endogenous leptin, led to an increase in physical activity and thermogenesis, and a decrease in weight, without a concomitant decrease in food intake [29].

Treatment of obesity is simple in theory, but not in practice. This statement is nicely supported by the vast amount of published studies evaluating various treatment modalities – around 5000 publications since 1966 – and the worldwide increase in prevalence of obesity.

In order to understand the effect of exercise and physical activity on weight reduction it is important to realize the associations between physical activity and energy expenditure. Physical activity is one of the most variable components of energy expenditure and thus physical activity/inactivity can influence both the development of obesity and the success in achieving weight loss. However, exercise as the principal method of weight loss is cumbersome, and the importance of exercise alone is easily overemphasized.

Twenty-four-hour energy expenditure (24hEE) has three major components – resting metabolic rate (RMR), thermic effect of exercise and thermic effect of food (TEF). RMR can be considered as the metabolic cost of maintaining the integrated systems of the body at rest and accounts for 60–80% of 24hEE. There are interindividual variations in RMR, which are to some extent determined by genetic factors [24]. Caloric restriction is associated with a decrease in RMR and TEF, thus resisting weight reduction or maintenance of achieved weight loss [26, 27]. These compensatory changes tending to return the subject to his or her initial weight contribute to the poor long-term efficacy of the treatment of obesity. However, a combination of caloric restriction with exercise enhances the maintenance of RMR and thus improves long-term results of weight reduction programmes. This is partly because weight loss by exercise does not influence RMR in an undesirable direction [30–35]. Following cessation of high intensity

exercise, RMR stays elevated for several hours. This could be due to continued increased substrate oxidation, increased body temperature, increased catecholamine levels and stimulation of protein synthesis [31, 36]. After interruption of exercise training, the exercise-induced increase in RMR returns to baseline within 3 days [37]. This only stresses the importance of life-long treatment of obesity.

Exercise preserves lean body mass, unlike dieting alone. This is crucial since the major determinant of the 24hEE is the fat-free body mass, accounting for about 80% of the variance observed in 24hEE between individuals [36–39].

Despite these desirable effects of exercise on metabolism and energy expenditure the usefulness of increased physical activity in attempts to lose weight is somewhat controversial (for reviews [26, 27]). There are several reasons for this controversy. First, obese individuals are often incapable of doing intense, long-term exercise – i. e. they cannot achieve an exercise level high enough to cause major increase in energy expenditure. Second, exercise alone is not as efficient as a potential initial weight loss strategy as caloric restriction. When exercise and a low caloric diet are combined, the weight loss is greater than with diet alone. By adding exercise to diet therapy, there is a further increase in loss of body fat and a reduction in abdominal obesity, especially in males [40]. Third, obese individuals tend to overestimate the amount of exercise and underestimate the amount of consumed food. An interview among over 200 obese subjects revealed that they ate approximately 50% more and exercised 50% less than their own records indicated [41]. The subjects also felt strongly that their obesity was due more to genetic and metabolic factors than to overeating or too little exercise. Fourth, exercise alone is a cumbersome therapy, requiring much effort, and its effects are easily overestimated. To consume the amount of energy equal to a small sandwich (125 kcal) a person has to walk briskly for 30 min. Thus, to contribute significantly to weight loss, exercise should become a part of normal daily activities in obese individuals. Fortunately, energy intake is not enhanced, in association with increased exercise among obese individuals [42].

The benefits of exercise appear to be greater in overweight men than overweight women. This may be due to a greater resistance for fat loss by exercise or a lower intensity and duration of exercise in weight loss programmes by women than men, or both [26, 27, 40, 43–45]. In any case, an equal loss of fat seems to require more exercise from women than from men. Exercise programmes are effective also in promoting loss of abdominal fat [40, 46, 47]. Whether exercise and hypocaloric diet equally reduce abdominal and gluteal fat, or whether one of the areas is preferentially influenced by exercise is not well known.

However, exercise is one of the few factors correlating with long-term body weight maintenance. Frequency of exercise again is one of the most powerful determinants of successful weight maintenance [27, 48, 49].

There are some differences between types of exercise performed. A combination of resistance training and aerobic endurance exercise appears to be most efficient in preserving fat-free body mass, compared to aerobic endurance exercise alone. Thus, an exercise programme for the treatment of obesity should include both aerobic endurance training and resistance training [50, 51]. For practical reasons resistance training might even be easier to perform for obese individuals.

## Hypertension

Hypertension is a major health problem afflicting approximately 15–30% of individuals in most western societies. However, the majority have mild hypertension and would therefore theoretically be suitable for non-pharmacological therapy [52, 53].

Epidemiological and clinical studies indicate a lower incidence of hypertension in physically active individuals, in all age groups. Likewise positive effects of physical activity and exercise on blood pressure have been reported. Exercise has even been shown to prevent age-associated increases in blood pressure [54–58]. The use of ambulatory blood pressure measurements indicate that exercise training can lower blood pressure, although less effect on diastolic pressure and blood pressure during the nighttime has been noted [59].

The above-mentioned aspects certainly make physical activity a potentially important tool both in the prevention and treatment of hypertension [60, 61].

A meta-analysis of 25 longitudinal studies examining the antihypertensive effects of exercise, showed reductions in resting systolic and diastolic blood pressure of 11 and 8 mmHg, respectively. In 67% of the experimental groups statistically significant reductions were noted in systolic blood pressure; the corresponding value for the diastolic blood pressure was 70% [60]. However, the decrement in blood pressure evoked by exercise was not sufficient to produce normotension in many studies.

There are studies that have failed to show any meaningful reductions in blood pressure in association with exercise training [62]. Such observations suggest that there may in fact be certain subgroups of patients with hypertension who are more responsive to the blood-pressure-lowering effects of exercise than others [63].

The blood pressure-lowering benefits that a given hypertensive subject can expect to derive from participation in exercise programmes is dependent on body

weight, diastolic blood pressure, and the programme itself. Interestingly, the reduction in blood pressure observed with regular aerobic endurance exercise has been proposed to be due to the accumulative effects of single exercise bouts rather than to long-term adaptations to exercise. A single bout of aerobic exercise reduces blood pressure for 1 to 3 h, in a similar magnitude, as elicited by chronic exercise training [64, 65].

One of the most controversial relationships in the metabolic syndrome has been the proposed association between insulin resistance and blood pressure. It has been postulated that hyperinsulinaemia/insulin resistance plays an independent causal role in the development of hypertension; however, this has been challenged and is certainly not conclusive [66–69]. Considerable heterogeneity exists between different racial and ethnic groups, furthermore the association between insulin and blood pressure seems to be stronger among lean than obese subjects. This heterogeneity could be an explanation for the varying results obtained regarding the effect of exercise on blood pressure.

Hypertensive subjects have traditionally been discouraged from performing resistance training due to the fear of precipitating a cerebrovascular or myocardial event. However, contrary to what might be expected studies investigating the long-term effect of circuit-type resistance training on resting blood pressure have generally failed to document a deleterious effect. Instead it has been shown that chronic resistance training modifies resting blood pressure in a favourable fashion [70–72]. Circuit-type moderate-intensity, high-volume resistance training can be used effectively and safely without causing increases in blood pressure and relative size of the left ventricle [73, 74].

Taken together, some controversy exists regarding the usefulness and effectiveness of exercise in the treatment of hypertension. Women seem to derive more benefits from exercise training than men, as well as individuals with lower body weight [56, 61, 63, 75, 76]. The overall impression from the published studies is that regular exercise training provides a modest antihypertensive effect and the use of exercise therapy for the treatment of hypertension is most beneficial in the early stages of hypertension as well as for the prevention of hypertension [54, 56, 77].

## Insulin resistance

Insulin resistance, present in approximately 25% of the western population, plays a central role in the metabolic syndrome being associated with most of the other metabolic abnormalities in the syndrome [2, 5, 7]. In this syndrome insulin resistance is primarily located in the muscle tissue and limited to the

non-oxidative pathway of glucose disposal, except in association with manifest NIDDM. The association of physical inactivity and insulin resistance was first suggested over 50 years ago [78]. Since then, both cross-sectional and intervention studies have addressed the association between physical activity and insulin sensitivity (for reviews [79–81]).

In cross-sectional studies, endurance athletes demonstrate lower serum insulin levels in the fasting and postprandial state, and a greater insulin sensitivity compared to their sedentary counterparts [82–86]. Master athletes also seem to be protected against a deterioration of glucose tolerance normally associated with aging [87,88]. To what extent greater insulin sensitivity in the athletes is due to natural selection and reflects genetic endowment rather than the effect of physical training, cannot be firmly established from cross-sectional studies [89].

In intervention studies the effects of both acute and chronic exercise on insulin sensitivity have been assessed. A single bout of acute exercise enhances insulin-mediated glucose disposal in normal subjects, in insulin-resistant first-degree relatives of NIDDM subjects, in obese subjects with insulin resistance as well as in NIDDM subjects [90–94]. However, it has long been known that the increase in insulin sensitivity after an acute bout of exercise is short-lived, which stresses the importance of chronic exercise [95]. There are at least two conditions in which acute exercise not only improves insulin sensitivity, but may even worsen it. First, extreme and eccentric exercise (e.g. running downhill), decreases insulin sensitivity. A contributing mechanism is muscle damage, which is substantial during eccentric exercise [96]. Second, after extreme long-term exercise, such as a marathon run, insulin sensitivity is reduced [97]. An explanation may be the ongoing enhanced utilization of non-esterified fatty acids as fuel. However, these are extreme conditions and the intensity of such exercise is more than the great majority of individuals with the metabolic syndrome are able to perform.

The effect of chronic exercise on insulin sensitivity has been studied in various settings. Physical training has mostly been shown to improve insulin sensitivity in healthy humans regardless of age [82, 98–101], in obese non-diabetic subjects [102, 103], in both patients with insulin-dependent diabetes and NIDDM [104–107]. Regular exercise is also beneficial in older subjects and improves insulin sensitivity even to levels typical of younger individuals, independent of changes in weight and body composition [99–101]. The improvement in insulin sensitivity has generally been considered to be proportional to the rise in physical fitness or  $\dot{V}O_{2\max}$ . However, exercise induced improvements in insulin sensitivity also occur in the absence of changes in  $\dot{V}O_{2\max}$  or BMI [108].

If exercise training is discontinued, the improvements gained in both insulin sensitivity and glucose

tolerance will vanish within a few days. Improved insulin sensitivity has been demonstrated 12–48 h after the exercise bout, but is virtually unmeasurable 3–5 days after the last exercise session [109–112]. However, this can be rapidly regained by one single bout of exercise [109, 113]. If physical exercise is minimal during leisure time, insulin sensitivity is low, and a few days bed rest is associated with substantial insulin resistance [114]. This means that the impact of exercise on insulin sensitivity, independent of the intervening effects of concomitant changes in body weight/body composition and residual effects of the last exercise session, need to be assessed and taken into consideration. Segal and co-workers focussed on this aspect [115]. A 12-week 4 h per week course of exercise training at 70% of  $\dot{V}O_{2\max}$  did not influence insulin sensitivity in lean, obese or NIDDM subjects, despite a 27% increase in  $\dot{V}O_{2\max}$  (i.e. a similar increase as observed in several other exercise studies). Basal hepatic glucose production was reduced by 22% in the diabetic men. The authors conclude that when intervening effects of the last exercise bout and changes in body composition are taken into account, exercise training per se leading to increased cardio-respiratory fitness had no independent effect on insulin sensitivity.

It has recently been demonstrated that acute exercise can normalize a defect in insulin-stimulated glucose transport-phosphorylation in insulin-resistant subjects. However, in these insulin-resistant normal-weight individuals an additional defect in glycogen synthase activity was observed, which could not be normalized even after chronic exercise. However, total glucose disposal increased 22% after the first exercise session and the overall increase was 42% after 6 weeks of exercise among insulin-resistant first-degree relatives of NIDDM subjects. Thus, the observed improvements in insulin sensitivity by exercise might reflect both a carry-over effect of the last bout of exercise as well as a chronic effect [94]. We have shown that 12 weeks of circuit-type resistance training, not affecting  $\dot{V}O_{2\max}$ , improves insulin sensitivity by 38% in insulin-resistant first-degree relatives of NIDDM patients [116] a percentage similar to that of Perseghin et al. [94].

Insulin resistance is often present in individuals who are not overtly obese, such as first-degree relatives of NIDDM subjects. These 'metabolically obese normal-weight' individuals appear to be quite common in the general population, and they could be an important target population for exercise therapy, for the prevention of the metabolic syndrome [68, 117, 118]. Also, the potential role of a diminished physical work capacity in the pathogenesis of insulin resistance among first-degree relatives of NIDDM subjects has recently been stressed [119].

## Glucose intolerance

It has been estimated that at least 25% of the incidence of NIDDM may be attributable to a sedentary lifestyle. Consequently the beneficial effects of exercise and physical activity in prevention of NIDDM are numerous [120, 121].

Exercise is considered a cornerstone in the treatment regimen for individuals with manifest NIDDM, and aerobic endurance exercise has traditionally been advocated as the most suitable exercise mode [122–124]. The recommendation that exercise training can be used as a therapeutic means to lower blood glucose levels in NIDDM subjects stems primarily from the fact that exercise has pronounced effects upon the metabolism of glucose. Exercising muscle may indeed increase glucose uptake 7- to 20-fold [125].

However, in contrast to the quite promising theoretical considerations based on exercise studies in healthy and obese individuals, the metabolic benefits in patients with NIDDM are not unequivocally convincing.

The beneficial effects of aerobic endurance exercise training alone on long-term glycaemic control and glucose tolerance in NIDDM subjects seem to be of minor importance, despite other positive metabolic effects in NIDDM subjects [122–124, 126, 127]. Despite the discouraging results from aerobic endurance exercise studies, we evaluated the effect of circuit-type resistance training and observed a significant improvement in long-term glycaemic control ( $HbA_{1c}$ : 8.8–8.2%) without concomitant changes in body weight or  $VO_{2max}$  (unpublished data). Resistance training has previously been associated with increases in capillary-to-muscle fibre ratio and glycogen storage capacity [128, 129].

Prospective, observational studies that have assessed the relationship between physical activity and risk of NIDDM have consistently shown a marked reduction in NIDDM among physically active individuals compared with their sedentary peers in both sexes.

The risk reduction when comparing the most active to the most sedentary has been between 0.64–0.94. Of interest is that the strongest protective effect has been observed among those at highest risk for NIDDM. This includes overweight individuals, hypertensive individuals, and those with a family history of NIDDM [130–132]. Also epidemiological studies indicate that individuals who are physically active are much less likely to develop NIDDM than individuals who have a very sedentary lifestyle [133, 134]. The prospective 6-year Malmö feasibility study showed that it is possible through intervention to actually induce and maintain changes in lifestyle in individuals with impaired glucose tolerance leading to prevention/postponement of manifest NIDDM [135].

However, at present, data are limited with respect to the intensity, frequency, and duration of exercise

that will be most effective in reducing the occurrence of NIDDM. According to the estimates derived from the prospective studies, the potential reduction in the risk of NIDDM associated with regular moderate and vigorous exercise, compared with a sedentary lifestyle, is 30–50% [136].

While the evidence is substantial that endurance exercise can reduce the risk of glucose intolerance and NIDDM, the evidence that aerobic endurance exercise training is beneficial in the treatment of NIDDM is not particularly strong. However, studies using prolonged, vigorous exercise training protocols have produced more favourable results.

## Dyslipidaemia

Dyslipidaemia associated with the metabolic syndrome is primarily characterised by hypertriglyceridaemia and low levels of HDL-cholesterol [137–139].

The effects of exercise on lipid and lipoprotein profiles are fairly well known. Early studies emphasizing the differences in total cholesterol and triglycerides between individuals involved in endurance-type exercise and sedentary subjects, have been confirmed by later studies [140]. According to the present consensus, physically active individuals have higher levels of HDL- and HDL<sub>2</sub>-cholesterol and lower levels of triglycerides, VLDL- and dense LDL-cholesterol, compared to sedentary individuals [141, 142]. Furthermore, intervention studies have shown that unfavourable lipid and lipoprotein profiles respond favourably to exercise training [141, 142]. The full significance of these findings can only be understood when considering the associations between lipids and lipoproteins, exercise and coronary heart disease morbidity and mortality [143–145]. Studies concerning the effect of exercise on lipids and lipoproteins among subjects with the metabolic syndrome are scarce, due to the lack of precise definition of the syndrome. However, it is likely that subjects with the metabolic syndrome would also benefit from exercise as do other individuals with dyslipidaemia. Exercise training increases the ability of muscle tissue to take up and oxidize non-esterified fatty acids and increases the activity of lipoprotein lipase (LPL) in muscle [139, 146, 147]. The combined effects of exercise and diet among NIDDM subjects resulted in a decrease in total and LDL-cholesterol, while HDL-cholesterol increased (for review [148]). However, it is difficult to separate dietary influences from the effects of exercise on serum lipids and lipoproteins.

Both the separate and combined effects of diet and exercise in reducing body weight and improving serum lipid and lipoprotein profiles among overweight subjects have been evaluated. Both treatment modalities caused significant increases in HDL-, HDL<sub>2</sub>- and

HDL<sub>3</sub>-cholesterol and decreases in triglyceride levels [149]. Aerobic endurance exercise training and a low-fat diet can normalize the metabolic profile of obese women, even if their adiposity remains higher than that of lean women [150].

In the studies by Lampman and Schteingart [151] exercise training alone increased insulin sensitivity, improved glucose tolerance, and lowered triglyceride and cholesterol levels among non-diabetic, non-obese subjects with hypertriglyceridaemia. However, these improvements did not occur when exercise training alone was performed by similar patients with impaired glucose tolerance.

Thirty-one obese premenopausal women participated in a 6 month exercise programme with exercise sessions four to five times a week.  $\dot{V}O_{2\max}$  increased and carbohydrate and lipid metabolism improved in the study group as reflected by decreases in total and LDL-cholesterol and an increase in the ratio of HDL- to LDL-cholesterol. However, as changes in body weight correlated significantly with changes in carbohydrate metabolism parameters and VLDL-triglycerides, the authors suggested that body fat loss may be a determinant factor in the improvement of carbohydrate and lipid metabolism following aerobic exercise training in obese women [152]. In the study by Hurley et al. [72] resistance training resulted in a 13% increase in HDL-cholesterol and a decrease in total cholesterol to HDL-cholesterol ratio despite an unchanged maximal oxygen uptake. These plasma lipoprotein changes are similar to those seen with aerobic exercise training [153].

Considering the evidence indicated above, it is very likely that exercise, either alone or combined with proper diet, is effective in improving serum lipid and lipoprotein profiles among subjects with the metabolic syndrome. However, it is difficult to distinguish the independent effect of exercise on serum lipids and lipoprotein profile from the effects of other beneficial health habits associated with regular exercise. Obesity, smoking, and excess use of dietary fat are less prevalent among physically active subjects [154, 155].

#### **What kind of exercise should be recommended for individuals with the metabolic syndrome?**

A combination of frequency, intensity, and duration of chronic exercise is responsible for producing a training effect [50]. The interaction of these factors provides the required stimulus. In general, training is specific for its target tissues and the lower the stimulus the lower the training effect and the greater the stimulus the greater the effect. Although age in itself is not a limiting factor for exercise training, a more gradual approach in applying the prescription at older ages seems prudent. Although the adequate dose

of exercise needed to achieve many health benefits is known, the minimum or optimal dose is still unknown for most disorders. Aerobic endurance training of fewer than two sessions a week, at less than 50% of maximum oxygen uptake and for less than 10 min/day, is inadequate for developing and maintaining fitness for healthy adults [50]. However, lower intensities and volumes may be capable of inducing favourable changes in energy expenditure, muscle lipoprotein lipase activity and blood pressure [156, 157].

Research on the effects of resistance training on metabolism is small compared with the same type of research involving aerobic endurance training. However, based on the available studies reviewed we propose that an optimal exercise programme for individuals with the metabolic syndrome should include components that improve cardio-respiratory fitness, muscular strength and endurance, i.e. a combination of aerobic endurance training and circuit-type resistance training [50, 123, 124, 158].

Resistance training can involve high-intensity, low-volume training, or moderate-intensity, medium to high-volume training. The first involves practising with near maximal weights but fewer repetitions (low-volume), the main goal being development of maximal strength and power in order to improve athletic performance. For individuals with characteristics of the metabolic syndrome this kind of resistance training includes a high risk for cardiac and vascular complications, one reason that resistance training was previously seldom applied to this group. Our increased knowledge has proven that moderate intensity, dynamic, high-volume (moderate to high number of repetitions) resistance training can be used effectively and safely [71–74]. However, we recommend that subjects with the metabolic syndrome should have a thorough physical examination performed before starting vigorous exercise in order to reduce the likelihood of complications. A maximal treadmill test should also be considered, as it would give additional information on the appropriate intensity level of the proposed exercise programme.

The previous controversy regarding the beneficial metabolic effects of resistance training is nicely demonstrated by the findings of Hurley et al. [159]. High-intensity, low-volume training was associated with lower HDL- and higher LDL-cholesterol levels than in endurance trained athletes. The more physiologic form of resistance training, i.e. moderate-intensity medium-volume training again was associated with higher HDL- and lower LDL-cholesterol levels, levels comparable to those of long-distance runners. This means that several studies evaluating the metabolic effects of resistance training using groups of strength-trained athletes/weight lifters, with varying training regimens in terms of resistance, repetitions

**Table 1.** Components of an exercise prescription for subjects with the metabolic syndrome

| Component | Suggestion   |
|-----------|--|
| Modality  | <i>Aerobic endurance activities</i> (walking, running, bicycling, swimming, cross-country skiing);<br><i>Circuit-type resistance training</i> (low-intensity, high-volume) |
| Frequency | <i>Three to seven sessions</i> spaced throughout the week<br>Both modalities should be represented   |
| Intensity | <i>Moderate to strong</i> on Borg scale [163]  |
| Duration  | 3–5 min warm-up<br>15–60 min at training intensity   |

and rest intervals as well as lack of control of anabolic steroid use, cannot be considered appropriate for the evaluation of the physiologic effects of resistance training on glucose and lipid metabolism. The type of resistance training programme applied is of utmost importance.

Resistance training certainly stresses the muscle in different ways than does endurance exercise. However, muscle contraction in general seems to elicit an insulin-like effect on glucose uptake [160]. The fact that the prevalence of the metabolic syndrome increases with increasing age and the decline in muscle mass associated with aging is associated with a decline in metabolic function, further supports the hypothesis of the usefulness of circuit-type resistance training [161].

The recommendations (Table 1) should be used in the context of the participants' needs, goals, and initial abilities. The important factor is to design a programme for the individual to provide the proper amount of physical activity to attain maximal benefit at the lowest risk. Emphasis should be placed on factors that result in permanent lifestyle changes and encourage a lifetime of physical activity.

## Conclusion

Population studies suggest that only a few people take adequate exercise. Long-term participation in exercise programmes is usually poor, with a drop-out rate of around 50% from supervised programmes. This means that much emphasis has to be put on the development of the programme. The most important aspect is that exercise should be enjoyable, affordable and accessible. Furthermore, individuals who exercise regularly report improved mood and sense of well-being [162]. This is naturally an important factor contributing to long-term compliance in exercise programmes. In addition to strategies for increasing and maintaining participation, the importance of enhancing levels of routine activities of daily living, such as stair climbing, should be emphasized. An important point is that it does not matter what type of physical

activity is performed; sports, household or yard work, or occupational tasks are all beneficial in increasing energy expenditure.

Regular exercise as a lifestyle behaviour must be emphasized, and therefore exercise probably is most likely to be effective when initiated in young individuals, before the onset of irreversible damage, and when lifestyle changes may be more acceptable. The harmful effects of overeating, smoking, and other physical abuses cannot be corrected by exercise alone. To maximize the benefits of physical training, proper nutrition and healthy habits must be combined to achieve a better quality of life. Since there is a clustering of risk factors in association with the metabolic syndrome the treatment chosen should attempt to aim at as many of the metabolic abnormalities as possible, and this opportunity is provided by regular physical activity.

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