Diabetologia (1997) 40: 125–135

Diabetologia

Review

Exercise and the metabolic syndrome

J. Eriksson[1], **S. Taimela**[2], **V. A. Koivisto**[3]

[1] National Public Health Institute, Helsinki, Finland
[2] Unit for Sports and Exercise Medicine, University of Helsinki, and David Fitness & Medical Ltd, Vantaa, Finland
[3] Helsinki University Hospital, Department of Medicine, Helsinki, Finland

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^2) 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^2 for men and 27.3 kg/m^2 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].

Corresponding author: Dr. J. Eriksson, National Public Health Institute, Mannerheimintie 166, SF-00300 Helsinki, Finland
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 succesful 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 $\dot{V}O_{2max}$. However, exercise induced improvements in insulin sensitivity also occur in the absence of changes in $\dot{V}O_{2max}$ 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 $\dot{V}O_{2max}$ did not influence insulin sensitivity in lean, obese or NIDDM subjects, despite a 27 % increase in $\dot{V}O_{2max}$ (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 $\dot{V}O_{2max}$, 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 $\dot{V}O_{2max}$ (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_2-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_2- and

HDL_3-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. $\dot{V}O_{2max}$ 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 wlth 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	*Aerobic endurance activities* (walking, running, bicycling, swimming, cross-country skiing); *Circuit-type resistance training* (low-intensity, high-volume)
Frequency	*Three to seven sessions* spaced throughout the week Both modalities should be represented
Intensity	*Moderate to strong* on Borg scale [163]
Duration	3–5 min warm-up 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 activites 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.

References

1. Camus JP (1966) Goutte, diabéte, hyperlipémie: un trisyndrome métabolique. Rev Rheumat 33: 10–14
2. Reaven GM (1988) Banting Lecture 1988: Role of insulin resistance in human disease. Diabetes 37: 1595–1607
3. Pyörälä K (1979) Relationship of glucose tolerance and plasma insulin to the incidence of coronary heart disease: results from two population studies in Finland. Diabetes Care 2: 121–141
4. Foster DW (1989) Insulin resistance – a secret killer? N Engl J Med 320: 733–734
5. Black HR (1990) The coronary artery disease paradox: the role of hyperinsulinemia and insulin resistance and implication for therapy. J Cardiovasc Pharmacol 15 [Suppl 5]:S26–S38
6. Landin-Wilhelmsen K (1990) Insulin resistance in obesity and hypertension. Gothenborg, Sweden. Academic Dissertation
7. DeFronzo RA, Ferrannini E (1991) Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, and atherosclerotic cardiovascular disease. Diabetes Care 14: 286–300
8. Ferrannini E, Haffner SM, Mitchell BD, Stern MP (1991) Hyperinsulinaemia: the key feature of a cardiovascular and metabolic syndrome. Diabetologia 34: 416–422
9. Rupp H (1992) Insulin resistance, hyperinsulinemia, and cardiovascular disease. The need for novel dietary prevention strategies. Basis Res Cardiol 87: 99–105
10. Widén EI, Eriksson JG, Groop LC (1992) Metformin normalized nonoxidative glucose metabolism in insulin-resistent normoglycemic first-degree relatives of subjects with NIDDM. Diabetes 41: 354–358
11. Nolan JJ, Ludvik B, Beerdsen P, Joyce M, Olefsky J (1994) Improvement in glucose tolerance and insulin resistance in obese subjects treated with troglitazone. N Engl J Med 331: 1188–1193
12. Paffenbarger RS, Wing AL, Hyde RT, Jung DL (1983) Physical activity and incidence of hypertension in college alumni. Am J Epidemiol 117: 245–257
13. Siscovick DS, LaPorte RE, Newman JM (1985) The disease-specific benefits and risks of physical activity and exercise. Public Health Rep 100: 180–188

14. Caspersen CJ, Christenson GM, Pollard RA (1986) Status of the 1990 Physical Fitness and Exercise Objectives – evidence from NHIS 1985. Publ Health Rep 101: 587–592
15. Tyron WW (1987) Activity as a function of body weight. Am J Clin Nutr 46: 451–455
16. Sallis JF, Patterson TL, Buouno MJ, Nader PR (1988) Relationship of physical fitness and physical activity to cardiovascular risk factors in children and adults. Am J Epidemiol 127: 933–941
17. VanItallie TB (1994) Worldwide epidemiology of obesity. Pharmaco Econ 5 [Suppl 1]:1–7
18. Foreyt J, Goodrick K (1995) The ultimate triumph of obesity. Lancet 346: 134–135
19. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL (1994) Increasing prevalence of overweight among US adults: the National Health and Nutrition Examination Surveys 1960 to 1991. JAMA 272: 205–211
20. Prentice AM, Jebb SA (1995) Obesity in Britain: gluttony or sloth? BMJ 311: 437–439
21. Vague J (1956) Degree of masculine differentiation of obesities: factor determinating predisposition to diabetes, atherosclerosis, gout, and uric calculous disease. Am J Clin Nutr 4: 220–234
22. Pi-Sunyer FX (1993) Medical hazards of obesity. Ann Intern Med 119: 655–660
23. Bray GA (1992) A retrospective view of obesity. Int J Obes 18 [Suppl 3]:S1–S4
24. Bouchard C, Pérusse L, Dériaz O, Després J-P, Tremblay A (1993) Genetic influences on energy expenditure in humans. Crit Rev Food Sci 33: 345–350
25. Keen H, Thomas BJ, Jarrett RJ, Fuller JH (1979) Nutrient intake, adiposity and diabetes. BMJ I:655–658
26. Wilson MA (1990) Treatment of obesity. Am J Med Sci 299: 62–68
27. King AC, Tribble DL (1991) The role of exercise in weight regulation in nonathletes. Sports Med 11: 331–349
28. Shah M, Jeffery RW (1991) Is obesity due to overeating, or to a defective metabolic rate? A review. Ann Behav Med 13: 73–81
29. Pellemounter M, Cullen M, Baker MB et al. (1995) Effects of the obese gene product on body weight regulation in ob/ob mice. Science 269: 540–543
30. Epstein LH, Wing RR, Koeske R, Valoski A (1984). Effects of diet plus exercise on weight change in parents and children. J Consult Clin Psychol 52: 429–437
31. Bielinski R, Schutz Y, Jéquier E (1985) Energy metabolism during the post-exercise recovery period in man. Am J Clin Nutr 42: 69–82
32. Wing RR, Epstein LH, Paternostro-Bayles M, Kriska A, Norwalk MP, Gooding W (1988) Exercise in a behavioural weight control programme for obese patients with type 2 (non-insulin-dependent) diabetes. Diabetologia 31: 902–909
33. van Dale D, Saris WHM, Ten Hoor F (1990) Weight maintenance and resting metabolic rate 18–40 months after a diet/exercise treatment. Int J Obes 14: 347–359
34. Frey-Hewitt B, Vranizan KM, Dreon DM, 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
35. Leibel Rl, Rosenbaum M, Hirsch J (1995) Changes in the energy expenditure resulting from altered body weight. N Engl J Med 332: 621–628
36. Horton ES (1985) Metabolic aspects of exercise and weight reduction. Med Sci Sports Exerc 18: 10–18
37. Tremblay A, Nadeau A, Fournier G, Bouchard C (1988) Effect of a three-day interruption of exercise training on resting metabolic rate and glucose-induced thermogenesis in trained individuals. Int J Obes 12: 163–168
38. Pavlou KN, Steffee WP, Lerman RH, Burrows BA (1985) Effects of dieting and exercise on lean body mass, oxygen uptake, and strength. Med Sci Sports Exerc 17: 466–471
39. Ravussin E, Lillioja S, Anderson TE, Christin L, Bogardus C (1986) Determinants of 24-hour energy expenditure in man: methods and results using a respiratory chamber. J Clin Invest 78: 1568–1578
40. Wood PD (1993) Impact of experimental manipulation of energy intake and expenditure on body composition. Crit Rev Food Sci 33: 369–373
41. Lichtman SW, Pisarska K, Berman EL et al. (1992) Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. N Engl J Med 327: 1893–1898
42. Keim NL, Barbieri TF, Belko AZ (1990) The effect of exercise on energy intake and body composition in overweight women. Int J Obes 14: 335–346
43. van Dale D, Saris WHM, Schoffelen PFM,TenHoor F (1987) Does exercise give an additional effect in weight reduction regimens? Int J Obes 11: 367–375
44. Hill JO, Sparling PB, Shileds TW, Heller PA (1987) Effects of exercise and food restriction on body composition and metabolic rate in obese women. Am J Clin Nutr 46: 622–630
45. Björntorp P (1989) Sex differences in the regulation of energy balance with exercise. Am J Clin Nutr 49 [Suppl]:958–961
46. Tunstall-Pedoe H, Smith WCS, Crombie IK, Tavendale R (1989) Coronary risk factor and lifestyle variation across Scotland: results from the Scottish Heart Health Study. Scot Med J 34: 556–560
47. Despres JP, Pouliot MC, Moorjani S et al. (1991) Loss of abdominal fat and metabolic response to exercise training in obese women. Am J Physiol 261:E159–E167
48. Colvin RH, Olson SB (1983) A descriptive analysis of men and women who have lost significant weight and are highly successful at maintaining the loss. Addic Behav 8: 287–295
49. Marston AR, Criss J (1984) Maintenance of successful weight loss: incidence and prediction. Int J Obes 8: 435–439
50. American College of Sports Medicine (1990) The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults.Med Sci Sports Exerc 22: 265–274
51. Walberg-Rankin J (1992) Utilizing exercise in the treatment of obesity. Compr Ther 18: 31–34
52. Kaplan NM (1984) Therapy of mild hypertension: an overview. Am J Cardiol 53: 2A–8A
53.Chobanian A (1986) Antihypertensive therapy in evolution. N Engl J Med 314: 1701–1702
54. Blair SN, Goodyear NN, Gibbons LW, Cooper KH (1984) Physical fitness and incidence of hypertension in healthy normotensive men and women. JAMA 252: 487–490
55. Darga LL, Lucas CP, Spafford TR, Schork MA, Illis WR, Holden N (1989) Endurance training in middle-aged male physicians. Physician and Sportsmedicine 17: 85–101
56. Gordon NF, Scott CB, Wilkinson WJ, Duncan JJ, Blair SN (1990) Exercise and mild essential hypertension. Recommendations for adults. Sports Med 10: 390–404
57. Harshfield GA, Dupaul LM, Alpert BS et al. (1990) Aerobic fitness and the diurnal rhythm of blood pressure in adolescents. Hypertension 15: 810–814
58. Kasch FW, Boyer JL, Van Camp SP, Verity LS, Wallace JP (1990) The effects of physical activity and inactivity on

aerobic power in older men (a longitudinal study). Physician and Sportsmedicine 18: 73–83
59. Van Hoof R, Hespel P, Fagard R, Lijnen P, Staessen J, Antoon A (1989) Effects of endurance training on blood pressure at rest, during exercise and during 24 hours in sedentary men. Am J Cardiol 63: 945–949
60. Hagberg JM (1990) Exercise, fitness and hypertension. In: Bouchard C (ed) Exercise, fitness and health: a consensus of current knowledge. Human Kinetic Books, Champaign, Illinois
61. Kaplan NM (1992) Exercise for the treatment of hypertension. Help or hype. Am J Hypertension 5: 574–576
62. Gilders RM, Dudley GA (1992) Endurance exercise training and treatment of hypertension. The controversy. Sport Med 13: 71–77
63. Blumenthal JA, Siegel WC, Appelbaum M (1991) Failure of exercise to reduce blood pressure in patents with mild hypertension: results of a randomized controlled trial. JAMA 266: 2098–2104
64. Hagberg JM, Mountain SJ, Martin WH (1987) Blood pressure and hemodynamic responses after exercise in older hypertensive. J Appl Physiol 63: 270–276
65. Kaufman FL, Hughson RL, Schaman JP (1987) Effect of exercise on recovery blood pressure in normotensive and hypertensive subjects. Med Sci Sports Exer 19: 17–20
66. Ferrannini E, Buzzigoli G, Bonadonna R et al. (1987) Insulin resistance in essential hypertension. N Engl J Med 317: 350–357
67. Laakso M, Sarlund H, Mykkänen L (1989) Essential hypertension and insulin resistance in non-insulin-dependent diabetes mellitus. Eur J Clin Invest 19: 518–526
68. Zavaroni I, Bonora E, Pagliaro M et al (1989) Risk factors for coronary artery disease in healthy persons with hyperinsulinemia and normal glucose tolerance. N Engl J Med 320: 702–706
69. Haffner S (1993) Insulin and blood pressure: fact or fantasy? J Clin Endocrinol Metab 76: 541–543
70. Hagberg JM, Goldring D, Ehsani AA et al. (1983) Effect of exercise training on the blood pressure and hemodynamic features of hypertensive adolescents. Am J Cardiol 52: 763–768
71. Harris KA, Holly RG (1987) Physiological responses to curcuit weight training in borderline hypertensive subjects. Med Sci Sports Exer 19: 246–252
72. Hurley BF, Hagberg JM, Goldberg AP et al. (1988) Resistance training can reduce coronary risk factors without altering VO_{2max} or percent body fat. Med Sci Sports Exer 20: 150–154
73. Fleck S (1988) Cardiovascular adaptations to resistance training. Med Sci Sports Exerc 20:S146–S151
74. Effron M (1989) Effects of resistance training on left ventricular function. Med Sci Sports Exer 21: 694–697
75. Hansen HS, Froberg K, Hyldebrandt N, Nielsen JR (1991) A controlled study of eight months of physical training and reduction of blood pressure in children: the Odense schoolchild study. BMJ 303: 682–685
76. Meredith IT, Friberg P, Jennings GL et al. (1991) Exercise training lowers resting renal but not cardiac sympathetic activity in human. Hypertension 18: 575–582
77. Martin JE, Dubbert PM, Cushman WC (1990) Controlled trial of aerobic exercise in hypertension. Circulation 81: 1560–1567
78. Blotner H (1945) Effects of prolonged physical inactivity on tolerance of sugar. Arch Int Med 75: 39–44
79. Holloszy JO, Schultz J, Kusnierkiewicz J, Hagberg JM, Ehsani AA (1986) Effects of exercise on glucose tolerance and insulin resistance. Acta Med Scand [Suppl] 711: 55–65
80. Koivisto VA, DeFronzo RA (1986) Physical training and insulin sensitivity. Diabetes Metab Rev 1: 445–481
81. Schneider SH, Morgado A (1995) Effects of fitness and physical training on carbohydrate metabolism and associated cardiovascular risk factors in patients with diabetes. Diabetes Reviews 3: 378–407
82. Björntorp P, Fahlen M, Grimby G et al. (1972) Carbohydrate and lipid metabolism in middle-aged physically well-trained men. Metabolism 21: 1037–1044
83. Lohmann D, Liebold F, Heilmann W, Stenger J, Pohl A (1987) Diminished insulin response in highly trained athletes. Metabolism 27: 521–524
84. Richter EA, Turcotte L, Hespel P, Kiens B (1992) Metabolic responses to exercise. Effects of endurance training and implications for diabetes. Diabetes Care 15: 1767–1776
85. Ebeling P, Bourey R, Koranyi L et al. (1993) Mechanisms of enhanced insulin sensitivity in athletes: increased blood flow, muscle glucose transport protein (GLUT-4) concentration and glycogen synthase activity. J Clin Invest 92: 1623–1631
86. Nuutila P, Knuuti MJ, Heinonen OJ et al. (1994) Different alterations in the insulin-stimulated glucose uptake in the athlete's heart and skeletal muscle. J Clin Invest 93: 2267–2274
87. Seals DR, Hagberg JM, Allen WK et al. (1984) Glucose tolerance in young and older athletes and sedentary men. J Appl Physiol 56: 1521–1525
88. Rogers MA, King DS, Hagberg JM, Ehsani AA, Holloszy JO (1990) Effect of 10 days of physical inactivity on glucose tolerance in master athletes. J Appl Phyisol 68: 1833–1837
89. Park JH, Brown LR, Park CR, Cohn M, Chance B (1988) Energy metabolism of the untrained muscle of elite runners as observed by ^{31}P magnetic resonance spectroscopy:evidence suggesting a genetic endowment for endurance exercise. Proc Natl Acad Sci USA 85: 8780–8784
90. Bogardus C, Thuillez P, Ravussin E, Vasquez B, Narimiga M, Azhar S (1983) Effect of muscle glycogen depletion on in vivo insulin action in man. J Clin Invest 72: 1605–1610
91. Burstein R, Epstein Y, Shapiro Y, Charuzi I, Karnieli E (1990) Effect of an acute bout of exercise on glucose disposal in human obesity. J Appl Physiol 69: 299–304
92. Devlin JT, Hirshman M, Horton ED, Horton ES (1987) Enhanced peripheral and splanchnic insulin sensitivity in NIDDM men after single bout of exercise. Diabetes 36: 434–439
93. Schneider SH, Khachadurian AK, Amorosa LF, Gavras H, Fineberg E, Ruderman NB (1987) Abnormal glucoregulation during exercise in type II (non-insulin-dependent) diabetes. Metabolism 6: 1161–1166
94. Perseghin G, Price TB, Petersen KF et al. (1996) Increased glucose transport-phosphorylation and muscle glycogen synthesis after exercise training in insulin-resistant subjects. N Eng J Med 335: 1357–1362
95. Richter EA, Garetto LP, Goodman MN, Ruderman NB (1982) Muscle glucose metabolism following exercise in the rat. J Clin Invest 69: 785–793
96. Kirwan JP, Hickner RC, Yarasheski KE, Kohrt WM, Wiethop BV, Holloszy JO (1992) Eccentric exercise induces transient insulin resistance in healthy individuals. J Appl Pysiol 72: 2197–2202
97. Tuominen JA, Ebeling P, Bourey R et al. (1996) Postmarathon paradox: insulin resistance in the face of glycogen depletion. Am J Physiol 270:E336–E343
98. Soman VR, Koivisto VA, Deibert D, Felig P, DeFronzo RA (1979) Increased insulin sensitivity and insulin binding

to monocytes after physical training. N Engl J Med 301: 1200–1204
99. Tonino RP (1989) Effect of physical exercise on the insulin resistance of aging. Am J Physiol 256:E352–E356
100. Kahn SE, Larson VG, Beard JC et al. (1990) Effect of exercise on insulin action, glucose tolerance, and insulin secretion in aging. Am J Physiol 258:E937–E943
101. Kirwan JP, Kohrt WM, Wojta DM, Bourey RE, Holloszy JO (1993) Endurance exercise training reduces glucose-stimulated insulin levels in 60- to 70-year-old men and women. J Gerontol 48:M84–M90
102. Björntorp P, DeJounge K, Sjöström L, Sullivan L (1970) The effect of physical training on insulin production in obesity. Metabolism 19: 631–638
103. DeFronzo RR, Sherwin RS, Kraemer N (1987) Effect of physical training on insulin action in obesity. Diabetes 36: 1379–1385
104. Pedersen O, Beck-Nielsen H, Heding L (1980) Increased insulin receptors after exercise in patients with insulin-dependent diabetes mellitus. N Engl J Med 302: 886–892
105. Koivisto VA, DeFronzo RA (1984) Exercise in the treatment of type II diabetes. Acta Endocrinol 262 [Suppl]:107–111
106. Reitman J, Vasquez B, Klimes l, Naguesparan M (1984) Improvement of glucose homeostasis after exercise training in non-insulin dependent diabetes mellitus.Diabetes Care 7: 434–441
107. Trovati M, Carta Q, Cavalot F et al. (1984) Influence of physical training on blood glucose control, glucose tolerance, insulin secretion, and insulin action in non-insulin dependent diabetic patients. Diabetes Care 7: 416–420
108. Oshida T, Yamanouchi K, Hayamizu S, Sato Y (1989) Long-term mild jogging increases insulin action despite no influence on body mass index or VO_{2max}. J Appl Physiol 66: 2206–2210
109. Burstein R, Polychronakos C, Toews CJ, MacDougal JD, Guyda HJ, Posner BI (1985) Acute reversal of the enhanced insulin action in trained athletes. Association with insulin receptor changes. Diabetes 34: 756–760
110. King DS, Dalsky GP, Clutter WE et al. (1988) Effects of exercise and lack of exercise on insulin sensitivity and responsiveness. J Appl Physiol 64: 1942–1946
111. King DS, Dalsky GP, Clutter WE et al. (1988) Effects of lack of exercise on insulin secretion and action in trained subjects. Am J Physiol 254:E537–E542
112. Mikinis KJ, Sonne B, Farrell PA, Tronier B, Galbo H (1988) Effect of physical exercise on sensitivity and responsiveness to insulin in humans. Am J Physiol 254:E248–E259
113. Heath GW, Gavin JR, Hinderliter JM, Hagberg JM, Bloomfeld SA, Holloszy JD (1983) Effects of exercise and lack of exercise on glucose tolerance and insulin sensitivity. J Appl Physiol 55: 512–517
114. Lipman RL, Schnure JJ, Bradley EM, Lecocq FR (1970) lmpairment of peripheral glucose utilization in normal subjects by prolonged bed rest. J Lab Clin Med 76: 221–230
115. Segal KR, Edano A, Abalos A et al. (1991) Effect of exercise training on insulin sensitivity and glucose metabolism in lean, obese, and diabetic men. J Appl Physiol 71: 2402–2411
116. Eriksson JG, Groop LC (1994) Resistance training improves insulin sensitivity in children of type 2 diabetic patients. Diabetologia 37 [Suppl 1]:A623 (Abstract)
117. Ruderman NB, Schneider SH, Berchtold P (1981) The metabolically obese, normal-weight individual. Am J Clin Nutr 34: 1617–1621
118. Zavaroni I, Bonini L, Fantuzzi M, Dall'Aglio E, Passeri M, Reaven GM (1994) Hyperinsulinaemia, obesity and syndrome X. J Intern Med 235: 51–56
119. Nyholm B, Mengel A, Nielsen S et al. (1996) Insulin resistance in relatives of NIDDM patients: the role of physical fitness and muscle metabolism. Diabetologia 39: 813–822
120. Rauramaa R (1984) Relationship of physical activity, glucose tolerance, and weight management. Prev Med 13: 37–46
121. Zimmet P, Faaiuso S, Ainuu S, Whitehouse S, Milne B, DeBoer W (1981) The prevalence of diabetes in the rural and urban Polynesian population of Western Samoa. Diabetes 30: 45–51
122. Kemmer FW, Berger M (1992) Exercise. In: Alberti KGMM, DeFronzo RA, Keen H, Zimmet P (eds) International textbook of diabetes mellitus. John Wiley and Sons, pp 725–743
123. Campaigne BN, Lampman RM (1994) Exercise in the clinical management of diabetes. Human Kinetics. Champaign, Illinois
124. Ruderman N, Devlin JT(1995)The health professional's guide to diabetes and exercise. ADA, Alexandria, Virginia
125. Wahren J, Felig P, Ahlborg G, Jorfeldt L (1971) Glucose metabolism during leg exercise in man. J Clin Invest 50: 2715–2725
126. National Institutes of Health (1987) Consensus development conference on diet and exercise in non-insulin-dependent diabetes mellitus. Diabetes Care 10: 639–644
127. Lehmann R, Vokac A, Niedermann K, Agosti K, Spinas GA (1995) Loss of abdominal fat and improvement of the cardiovascular risk profile by regular moderate exercise training in patients with NIDDM. Diabetologia 38: 1313–1319
128. Hickson RC, Rosenkoetter MA, Brown MM (1980) Strength training effects on aerobic power and short-term endurance. Med Sci Sports Exerc 12: 336–339
129. Tesch P (1988) Skeletal muscle adaptations to long-term heavy resistance exercise. Med Sci Sports Exerc 20 [Suppl 5]:S132–S134
130. Helmrich SP, Ragland DR, Leung RW, Paffenbarger RS (1991) Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. N Engl J Med 325: 147–152
131. Manson JE, Rimm EB, Stampfer MJ et al. (1991) Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. Lancet 338: 774–778
132. Manson JE, Nathan DM, Krolewski AS, Stampfer MJ, Willett WC, Hennekens CH (1992) A prospective study of exercise and incidence of diabetes among US male physicians. JAMA 268: 63–67
133. Jarrett RJ (1989) Epidemiology and public health aspects of non-insulin-dependent diabetes mellitus. Epidemiol Rev 11: 151–171
134. Zimmet P, Dowse G, Finch C, Serjeantson S, King H (1990) The epidemiology and natural history of NIDDM: lessons from the South Pacific. Diabetes Metab Rev 6: 91–124
135. Eriksson K-F, Lindgärde F (1991) Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise. The 6-year Malmo feasibility study. Diabetologia 34: 891–898
136. Manson JE, Spelsberg A (1994) Primary prevention of non-insulin-dependent diabetes mellitus. Am J Prev Med 10: 172–184
137. Reaven GM, Lerner LR, Stern MP, Farquhar JW (1967) Role of insulin in endogenous hypertrigyceridemia. J Clin Invest 46: 1756–1767

138. Tobey TA, Greenfield M, Kraemer F, Reaven GM (1981) Relationship between insulin resistance, insulin secretion, very low density lipoprotein kinetics, and plasma triglyceride levels in normotriglyceridemic men. Metabolism 30: 164–171
139. Blomhoff JP (1992) Lipoproteins, lipases, and the metabolic cardiovascular syndrome. J Cardiovasc Pharmacol 20 [Suppl 8]:S22–S25
140. Haskell WL (1984) The influence of exercise on the concentrations of triglyceride and cholesterol in human plasma. Exerc Sport Sci Rev 12: 205–244
141. Durstine JL, Haskell WL (1994) Effects of exercise on plasma lipids and lipoproteins. Exerc Sport Sci Rev 22: 477–521
142. Stefanick ML, Wood PD (1994) Physical activity, lipid and lipoprotein metabolism, and lipid transport. In: Bouchard C, Shephard RJ, Stephens T (eds) Physical activity, fitness and health. International proceedings and consensus statement. Human Kinetics, Champaign, Ill., pp 417–531
143. Paffenbarger RS, Hyde RT, Wing AL, Steinmetz CH (1984) A natural history of athleticism and cardiovascular health. JAMA 252: 491–495
144. Leon AS, Connett J, Jacobs DR, Rauramaa R (1987) Leisure-time physical activity levels and risk of coronary heart disease and death: The Multiple Risk Factor Intervention Trial. JAMA 258: 2388–2395
145. Ekelund LG, Haskell WL, Johnson JL, Whaley FS, Criqui MH, Sheps DS (1988) Physical fitness as predictor of cardiovascular mortality in asymptomatic North American men: The Lipid Research Clinics Mortality Follow-Up Study. New Engl J Med 319: 1379–1384
146. Nikkilä EA, Taskinen M, Rehunen S, Härkönen M (1978) Lipoprotein lipase activity in adipose tissue and skeletal muscle of runners: relation to serum lipoproteins. Metabolism 27: 1661–1671
147. Pollare T, Vessby B, Lithell H (1991) Lipoprotein lipase activity in skeletal muscle is related to insulin sensitivity. Arterioscler Thromb 11: 1192–1203
148. Gudat U, Berger M, Lefèbvre PJ (1994) Physical activity, fitness, and non-insulin-dependent (type II) diabetes mellitus. In: Bouchard C, Shephard RJ, Stephens T (eds) Physical activity, fitness and health. International proceedings and concensus statement. Human Kinetics, Champaign, Illinois, pp 669–683
149. Wood PD, Stefanick ML, Dreon DM et al. (1988) Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. N Engl J Med 319: 1173–1179
150. Tremblay A, Despres JP, Maheux J et al. (1991) Normalization of the metabolic profile in obese women by exercise and a low fat diet. Med Sci Sports Exerc 23: 1326–1331
151. Lampman RM, Schteingart DE (1991) Effects of exercise training on glucose control, lipid metabolism, and insulin sensitivity in hypertriglyceridemia and non-insulin dependent diabetes mellitus. Med Sci Sports Exerc 23: 703–712
152. Lamarche B, Despres JP, Pouliot MC et al. (1992) Is body fat loss a determinant factor in the improvement of carbohydrate and lipid metabolism following aerobic exercise training in obese women? Metabolism 41: 1249–1256
153. Gordon NF, Cooper KH (1988) Controlling cholesterol levels through exercise. Compr Therapy 14: 52–57
154. Blair SN, Jacobs DR, Powell KE (1985) Relationships between exercise or physical activity and other health behaviors. Public Health Rep 100: 172–180
155. Escobedo LG, Marcus SE, Holtzman D, Giovino GA (1993) Sports participation, age at smoking initiation, and the risk of smoking among US high school students. JAMA 269: 1391–1395
156. Jennings G, Nelson L, Nestel P et al. (1986) The effects of changes in physical activity on major cardiovascular risk factors, hemodynamics, sympathetic function, and glucose utilization in man: a controlled study of four levels of activity. Circulation 73: 30–40
157. Haskell WL (1994) Dose-response issues from a biological perspective. In: Bouchard C, Shephard R, Stephens T (eds) Physical activity, fitness, and health. International proceedings and consensus statement. Human Kinetics, Champaign, Illinois, pp1030–1039
158. Soukup JT, Kovaleski JE (1993) A review of the effects of resistance training for individuals with diabetes mellitus. Diabetes Educ 19: 307–312
159. Hurley BF, Seals DR, Hagberg JM (1984) High-density-lipoprotein cholesterol in bodybuilders vs powerlifters: negative effects of androgen use. JAMA 252: 507–513
160. Holloszy JO, Constable SH, Young DA (1986) Activation of glucose transport in muscle by exercise. Diabetes Metab Rev 1: 409–423
161. Tzankoff SP, Norris AH (1977) Effect of muscle mass decrease on age-related BMR changes. J Appl Physiol 43: 1001–1006
162. McAuley E (1994) Physical activity and psychosocial outcomes. In: Bouchard C, Shepard RG, Stephens T (eds) Physical activity, fitness, and health: international proceedings and consensus statement. Human Kinetics, Champaign, Illinois, pp 551–568
163. Borg GA (1973) Perceived exertion: a note on history and methods. Med Sci Sports Exerc 5: 90–93