Interaction of physical activity and diet: implications for lipoprotein metabolism

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Abstract

Objective: To consider how physical activity interacts with diet to modify lipoprotein metabolism and comment on implications for human health.

Design: An overview of lipoprotein metabolism is followed by a summary of the main effects of physical activity on lipoprotein metabolism. Interactions with dietary practice and the disposition of dietary lipid are reviewed, with comment on links with body fatness.

Setting: Literature is reviewed in relation to the risk of atherosclerotic disease.

Subjects: Although some data are presented on athletic groups, evidence relating to individuals with normal physical activity habits is mainly discussed.

Results: Physical inactivity is a risk factor for cardiovascular disease and one mechanism may involve changes to lipoprotein metabolism. The consensus is that aerobic activity involving an expenditure of $\geqslant 8\,MJ \cdot week^{-1}$ results in an increase in HDL cholesterol and probably decreases in fasting triacylglycerol. These changes occur despite the spontaneous increase in the proportion of dietary energy from carbohydrate which accompanies increased exercise. For this reason, exercise may be a means of reducing the hypertriglyceridaemic and HDL-lowering effects of low fat (high carbohydrate) diets. Decreases in total and low density lipoprotein cholesterol are sometimes, but not always, reported in sedentary individuals beginning exercise. One mechanism linking all these changes may be alterations to the dynamics of triacylglycerol-rich particles, particularly in the fed state.

Conclusions: The expenditure of considerable amounts of energy through regular, frequent physical activity increases the turnover of lipid substrates, with effects on their transport and disposition which may reduce the progression of atherosclerosis.

Keywords
Exercise
Energy expenditure
Dietary fat and carbohydrate
Lipoprotein lipids

Key messages

- Men and women who are physically active, even at a moderate level of intensity, experience a lower risk of cardiovascular disease.
- This may be in part attributable to their characteristically high concentrations of plasma HDL cholesterol and low concentrations of triglycerides.
- These characteristics may be attributable to improved transport and disposition of dietary fat during the hours following a fat-containing meal.
- Like other effects on metabolic risk factors for cardiovascular disease (lowering of blood pressure, increase in insulin sensitivity), the effects of exercise on postprandial triglyceride metabolism are short-lived.
- Regular, frequent exercise is recommended to modify lipoprotein metabolism. This can be of moderate intensity provided that sufficient energy is expended.

Introduction

Physical inactivity is an important risk factor for cardiovascular disease. Overall, sedentary men experience about twice the risk seen in active men[1] but the greatest difference in risk is seen between those with the lowest levels of activity or fitness and those in the next highest category[2]. Thus a habit of moderate exercise, conferring only a modest level of fitness, appears sufficient to confer a measurable – although not optimal – decrease in risk. Multiple mechanisms are probably involved, with effects potentially on both chronic and acute phases of the disease. Those likely to decrease the progression of atherosclerosis, commonly the underlying pathology, include changes to lipoprotein metabolism.

Energy turnover is enhanced with regular exercise and an example of an association with disease risk is given in Table 1[3].This shows average daily energy intakes in prospective studies of coronary heart disease

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Table 1 Average daily reported intake of energy among men who subsequently became heart attack victims and those who were survivors. Values are kcal · day^{-1} (MJ). Data from Wood[3]

Community studies	Victims	Survivors
English banking and omnibus workers	2 656 (11.12)	2 869 (12.01)
Framingham, MA	2 369 (9.92)	2 622 (10.98)
Puerto Rico	2 223 (9.31)	2 395 (10.03)
Honolulu	2 149 (9.00)	2 319 (9.71)

(CHD); men who subsequently had fatal attacks showed lower levels of energy intake than survivors, an apparent paradox in the light of the increase in CHD risk associated with overweight and obesity. One explanation is that the men with higher energy intakes were more physically active, and that their exercise (or some consequence of this) afforded a level of protection against CHD, compared with more sedentary men who ate less. Thus, the transition from a sedentary to an active state is necessarily associated with a higher energy turnover, with important implications for the transport, storage and utilization of lipid fuels.

Lipoprotein metabolism

A brief outline of the metabolism of lipoproteins helps understand the interactions of diet with exercise and their health implications. These macromolecular complexes comprise a core of triacylglycerol (TAG) and cholesteryl esters, surrounded by a relatively hydrophilic coat comprising phospholipid, free cholesterol and one or more protein molecules known as apolipoproteins. There are four main categories, i.e. chylomicrons, very low density lipoproteins (VLDL), low density lipoproteins (LDL) and high density lipoproteins (HDL).

Chylomicrons carry TAG and cholesterol derived from foods. As they pass through the capillary beds of adipose tissue and muscle their core TAG is hydrolysed by the enzyme lipoprotein lipase (LPL) and the fatty acids released are mostly taken up by the tissues. As they lose TAG, the chylomicrons shrink and cholesterol-rich remnant particles are removed by hepatic receptors. Thus chylomicrons deliver TAG to peripheral tissues and cholesterol to the liver. By contrast, VLDL distribute TAG synthesized in the liver to other tissues. Like chylomicrons, they are a substrate for LPL and become TAG-depleted as they pass through capillary beds. Their remnants are the cholesterol-rich LDL which carry (in ester form) some 70% of the cholesterol in the circulation, delivering it to a variety of tissues according to their needs. Plasma total cholesterol concentration, which is strongly and positively related to the risk of CHD)[4], predominantly reflects LDL cholesterol. HDL provide a means by which cholesterol is routed from peripheral tissues to the liver where it is disposed of safely, mainly via synthesis into bile acids. HDL receive unesterified cholesterol which is released as excess surface material during the degradation of TAG-rich particles, but also incorporate cholesterol from the body's cells when this is present in excess of needs. Serum concentrations of HDL cholesterol are inversely related to the risk of cardiovascular disease[5].

Physical activity and lipoprotein lipids

Well-trained endurance runners, men and women, possess lipoprotein profiles consistent with a low risk of CHD[6]. Differences are most marked for HDL cholesterol that is typically 20–30% higher than in comparable sedentary controls. Plasma TAG concentration is low, particularly when veteran athletes are studied. Total cholesterol concentrations are not consistently reported to be low in endurance-trained people but tend to be when the control group is large and representative of the wider population. By contrast with endurance-trained individuals, athletes trained specifically for strength and power do not differ systematically from sedentary people.

Less athletic, but physically active, people also show favourable lipoprotein profiles. For example, data from the Lipid Clinics Prevalence Study[7] showed that men and women who reported some 'strenuous' physical activity generally had higher HDL cholesterol levels than those who reported none. Differences were independent of age, body mass index, alcohol use and cigarette smoking. Even simple exercise like walking has been linked to elevated HDL levels, with relationships between distance walked per day and the concentration of HDL_2[8], the subfraction that accounts for most of the difference in total HDL cholesterol between athletes and controls. In addition, men and women who habitually walk 12 to 20 km · week^{-1} are only half as likely to possess an unfavourable ratio of total to HDL cholesterol (defined as >5) as a comparable no-exercise group[9]. Thus cross-sectional observations of ordinary men and women, and of everyday activity, provide a basis for proposing that endurance exercise influences lipoprotein metabolism. The findings of longitudinal studies are less consistent but the consensus is that, over months rather than weeks, endurance exercise involving a minimum expenditure of about 8.0 MJ · week^{-1} results in an increase in HDL cholesterol. The majority of studies have employed rather high intensity exercise, most frequently jogging/running, but evidence is gradually becoming available that more accessible, self-governed exercise regimens may also be effective[10]. For example, in previously sedentary middle-aged women who had rather low levels of HDL cholesterol (mean

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1.2 mmol · l^{-1}) at baseline, walking briskly for about 20 km per week over a year resulted in a 27% increase[11]. The pace of walking does not seem to be an important determinant of the increase in HDL cholesterol; this was similar in women walking 4.8 km (3 miles) per day at either 4.8 km · h^{-1} (3 mph), 6.4 km · h^{-1} (4 mph) or 8.0 km · h^{-1} (5 mph)[12].

Interactions with diet

Relation with dietary influences on lipoproteins

The high levels of HDL cholesterol and low TAG in endurance athletes and recreational exercisers cannot be explained by their dietary practices. Endurance athletes typically consume a greater proportion of their energy intake from carbohydrate than the population at large; average values from four studies ranged from 49–60% energy intake[13], an intake of around 700 g · day^{-1} [14], compared with some 270 g · day^{-1} for the UK population[15]. Sedentary people taking up physical activity also tend to consume more carbohydrate. An example is shown in Fig. 1 where a group of middle-aged men who took up a modest programme of jogging increased their average daily energy intake by about 1.25 MJ per day over two years (12.5%), the majority of this increase coming from carbohydrate (increase of 70 g · day^{-1} or about 30%)[16].

It has been known for many years that high carbohydrate diets increase fasting TAG and reduce HDL cholesterol. Combined experimental and epidemiological data suggest that every 10% of energy from fat that is replaced by carbohydrates (complex as well as sugars) lowers HDL cholesterol by 0.1 mmol · l^{-1} [17]. Athletes, because of their high energy intake, often have a similar or higher intake of fat in absolute terms than the population at large[13], and the extent to which increased carbohydrate intake without a complementary decrease in fat intake decreases HDL cholesterol is unclear. However the balance of dietary change in free-living people who become more active is probably to increase somewhat the percentage of energy intake derived from carbohydrate. For example in the study referred to above[16] this increased on average from 39% to between 42% and 44% over two years.

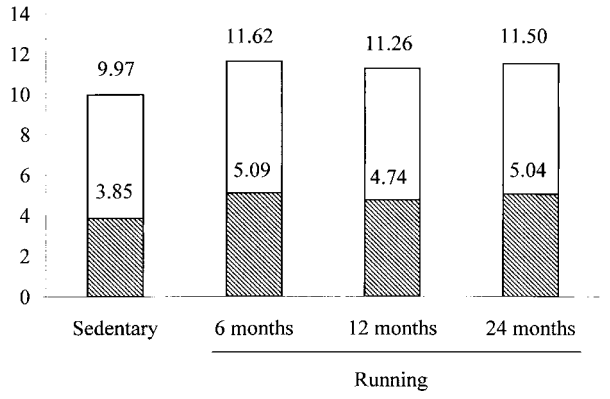

Fig. 1 Average increase over two years in energy intake (MJ) in sedentary men who took up jogging. Hatched area shows energy from carbohydrate[16]

Some studies have found that runners and joggers report a lower consumption of meat[18,19], which may contribute towards lower serum total cholesterol levels where these are reported. This is probably not a major factor however, because there were poor correlations in these studies between the amount of meat and meat products consumed and total cholesterol[18]. It seems, therefore, that physically active people exhibit favourable lipoprotein lipid characteristics despite – rather than because of – their dietary practice. This suggestion is supported by findings that factors related to exercise rather than diet predict changes in HDL cholesterol and TAG levels. For example, Hagan and colleagues studied 45 men who trained by running for 6 months[20]. Dietary practices and changes to this were monitored through food frequency records. The distance run was the best predictor of HDL cholesterol, and the second best predictor of TAG levels. Dietary factors were not important predictors of the distance run.

Various scientific bodies have recommended reductions in dietary fat (particularly saturated fat) as a means of reducing serum cholesterol level and hence coronary heart disease. Most recommendations have specified that the fat be replaced by complex carbohydrates. As such changes reduce HDL levels, theoretically they may diminish the anticipated beneficial effects of decreased LDL cholesterol. Exercise was suggested as a way to offset such a diet-related fall in HDL cholesterol. For example, comparison of two different interventions in sedentary overweight men and women, i.e. a low energy, low fat diet alone *v.* the same diet plus exercise (brisk walking and jogging) showed that the addition of exercise to the low fat diet resulted in more favourable changes in HDL cholesterol than diet alone[21]; in men, diet plus exercise provoked a greater rise in HDL cholesterol than did diet only; and in women only the diet-plus-exercise group showed a favourable change in the ratio of LDL cholesterol to HDL cholesterol (Fig. 2)[21]. The same research group recently reported findings from a similar trial in 180 postmenopausal women and 197 men, all of whom had low HDL cholesterol and moderately elevated levels of LDL cholesterol on entry to the study[22]. In this group neither a low fat diet alone (< 30% energy from fat, < 7% energy from saturated fat, < 200 mg cholesterol · day^{-1}) nor exercise alone reduced LDL cholesterol; by contrast, men and women who both followed the diet and exercised experienced reductions relative to controls.[22] This evidence strengthens the case for promoting increased physical activity

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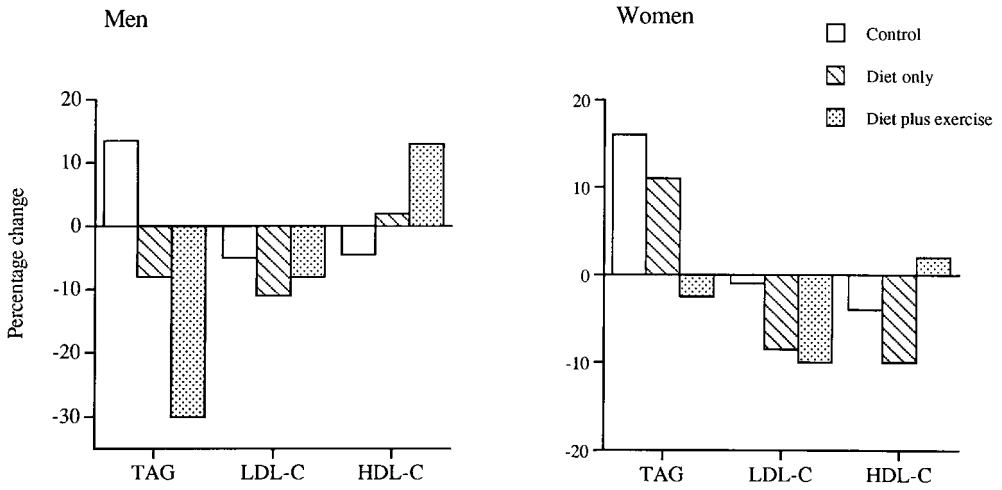

Fig. 2 Percentage changes over one year in overweight men and women who were randomly assigned to either maintain their usual sedentary lifestyle (control) or go on a hypocaloric, low fat, low cholesterol diet (diet only) or follow the same diet and undertake regular moderate exercise (diet plus exercise)[21]

alongside dietary change as a means of reducing the risk of cardiovascular disease.

Changes to the disposition of dietary fat

High concentrations of HDL cholesterol and low concentrations of TAG in active people are linked mechanistically via changes to the transport and disposition of dietary fats. As explained above, chylomicrons and VLDL are removed from the circulation by a common, saturable pathway[23] involving hydrolysis of their core TAG by LPL. This process is linked with HDL cholesterol concentration in two ways. First, nascent (discoidal) HDL particles are converted to mature, spherical particles through the transfer of surface material (including free cholesterol) from TAG-rich lipoproteins after the latter are 'slimmed down' through the action of LPL. This results in the formation of stable, cholesterol ester-rich HDL_2 particles. By this means the rate of HDL_2 synthesis is positively and strongly related to the rate of VLDL degradation. Second, the rate of removal of TAG-rich lipoproteins determines the opportunity for exchange of core lipid with the cholesterol-rich lipoproteins, i.e. HDL and LDL. When the residence time of TAG-rich lipoproteins is short there is little opportunity for this exchange of lipid and the cholesterol content of HDL is preserved, maintaining a good level of reverse cholesterol transport. Thus, HDL cholesterol has been described as an 'integrative marker' for TAG metabolic capacity[24]. The high HDL concentrations in physically active people may therefore be a consequence of the effects of their exercise habit on the disposal of TAG-rich lipoproteins.

The influence of exercise and training on the handling of TAG-rich lipoproteins can be most clearly seen in the postprandial state, when the entry into the circulation of chylomicrons carrying dietary fat presents a challenge to TAG removal capability. As might be expected from their low TAG concentrations in the fasted state, endurance trained individuals exhibit a lower postprandial TAG response to an oral fat load than their sedentary counterparts. Studies using a fat load between 40–140 g have produced broadly similar findings, i.e. the magnitude of postprandial lipaemia – measured as the area under the plasma TAG concentration *v.* time curve over 8 h – has been found to be 27–59% lower in endurance-trained men than in controls[25–27]. In one of these studies, the magnitude of postprandial lipaemia was 45% lower in athletes despite the fact that their fasting TAG concentrations were similar to those of sedentary controls[26], showing that differences between groups could not be attributed solely to the fasting TAG pool size.

Before attributing the low level of lipaemia in athletes to long-term adaptations to training it should be remembered that athletes invariably train at least once per day. Acute effects of individual sessions of exercise can profoundly influence fasting and postprandial TAG metabolism[28] and measures of fat tolerance in cross-sectional studies have often been made in proximity to a training session[25,26,29–31]. Consequently, although these findings describe accurately the good fat tolerance in trained people in everyday life, they do not permit a conclusion to be drawn on whether long-term adaptations to training exert an influence on postprandial lipaemia beyond the effects of a single session of exercise.

There is little information on the extent to which taking up moderate exercise influences postprandial lipaemia. In a detailed study of six healthy men before and after seven weeks of jogging training Weintraub and co-workers controlled diet carefully to ensure that

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subjects did not lose body weight[32]. Based on the vitamin A-fat loading test, chylomicron retinyl palmitate levels were reduced by 37% with training. There was no relation between chylomicron lipaemia and HDL cholesterol, the authors suggesting that a longer period of training may be needed before this complementary change is evident. A longer study, with 32 and 48 weeks of training, found a 49% improvement in the capability to clear an intravenous fat load alongside a 13% increase in HDL cholesterol[33]. The latter finding, alongside training-induced increases in heparin-releasable LPL activity[32,33], suggests that improved TAG clearance rather than decreased appearance is responsible for the exercise-induced decrease in the plasma TAG response to dietary fat.

In a more recent and randomized controlled study, healthy middle-aged women trained over 12 weeks by brisk walking[34]. Despite clear improvements in endurance fitness there was no change in the plasma TAG response to an oral fat load. One possibility is that the intensity of training, i.e. ~60% $\dot{V}O_{2max}$, was insufficient to stimulate morphological changes in skeletal muscle. However, this study was specifically designed to exclude the effects of recent exercise; subjects were studied 48 h after[34] the last training session and so only a long-lasting adaptive effect would be discerned. In contrast, there are reports of decreased postprandial lipaemia with training from studies where the acute effects of a recent exercise session can be excluded because measurements were made 36 h and 4 days after[32,35]. In the study of Drexel and colleagues, however, a combined intervention of food intake restriction and exercise was employed so the findings are not directly comparable[35].

There are some indications of potential mechanisms; the lower lipaemic response after training appears to reflect mainly differences in chylomicron levels[32,35], which might be expected if training increases LPL activity and chylomicrons are the preferred substrate for LPL[36]. This suggestion is supported by the 'dramatic' effect on chylomicron and chylomicron remnant metabolism when trained men interrupt their training for 2 to 3 weeks[37]; de-training increased the areas under the plasma concentration *v.* time curves by 41% and 37%, respectively.

The importance of effects of individual exercise sessions was mentioned. This is underlined by a report that endurance athletes refraining from exercise for six days experienced a profound increase in the lipaemic response to a standard high-fat meal, most of this increase occurring in the first 60 h[38]. It was known since the early 1960s that a session of exercise diminishes postprandial lipaemia. For example, postprandial TAG concentrations at 4, 6 and 8 h after consuming a high-fat breakfast were higher when men ($n=6$) had exercised at 75% of peak $\dot{V}O_2$ for 30 min., starting 1 h after the meal than in a rested control trial[39]. A similar approach, i.e. a high-fat meal followed by exercise or rest in a repeated measures design, was employed to study the effects of cycling[40] and treadmill walking[41]. In both studies subjects exercised at 40% $\dot{V}O_{2max}$ for 90 min., starting 1.5 h after the test meal. Postprandial lipaemia was lower in the exercise trial by 34% and 24%, respectively[40,41]. However, most or all of this difference was evident during the period of recovery after exercise. Exercise-induced changes in LPL activity – the putative mechanism – appear to be delayed[42]. For this reason studies examining fat tolerance some hours after a session of exercise provide a clearer picture of exercise-induced changes in postprandial lipid metabolism.

For example, young adults walked for two hours at about 40% of $\dot{V}O_{2max}$ one afternoon and their fat tolerance was tested the following morning[43]. Postprandial lipaemia was nearly one third lower than on a no-exercise control trial. The energy expended during exercise seems to be a determinant of its effects on lipaemia, as shown in Fig. 3; sessions of moderate or low intensity exercise of equivalent energy expenditure (1.5 h at 60% $\dot{V}O_{2max}$ *v.* 3 h at 30% $\dot{V}O_{2max}$) resulted in identical decreases in lipaemia[44].

These changes to the disposition of dietary fat probably contribute to exercise-induced increases in HDL cholesterol and decreases in TAG levels. It is not known, however, how these changes to the handling of the fat in a standard meal might be affected by the spontaneous dietary adjustments described above, i.e. increased intake of energy and carbohydrate.

Health benefit from enhanced metabolic capacity for TAG-rich lipoproteins

It is 20 years since it was first proposed that chylomicron remnants are atherogenic[45]. Current thinking is that, although these particles may not be implicated directly, repeated episodes of exaggerated or perturbed postprandial lipaemia result in multiple disturbances of lipoprotein which are, in turn, related to premature atherosclerosis[46]. This view is supported by the findings of case-control studies that postprandial TAG concentrations, particularly when measured 6–8 h after an oral fat load, are important discriminators of individuals with known coronary artery disease[24]. People spend most of their lives in the postprandial state so, speculatively, the effects of regular, frequent exercise could attenuate this component of the atherogenesis.

There may be related benefits for weight regulation, as shown by a recent study of middle-aged women. The women ate a high-fat breakfast on two occasions, once the morning after a long walk and once after a day with minimal activity[47]. Fasting and postprandial fat

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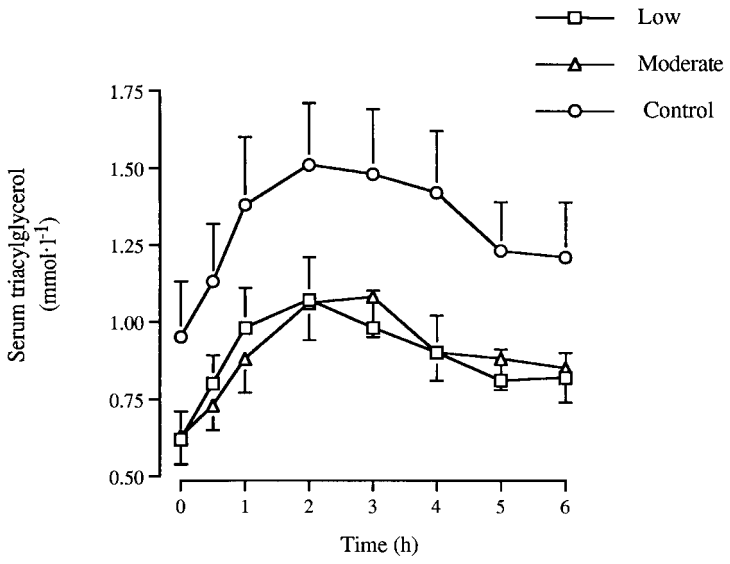

Fig. 3 Serum triacylglycerol responses to a high fat mixed meal consumed in the fasted state in nine young adults (5 men). Three trials: after a day of minimal physical activity (control), after a 3 h walk at 30% VO_{2max} (low) and after a 1.5 h walk at 60% VO_{2max}[44]

oxidation was significantly greater after walking. This likely reflects increased uptake of fatty acids into muscle and fits well with the findings of a study of LPL when endurance athletes refrained from exercise for 2 weeks; LPL activity in muscle decreased markedly, with a complementary increase in adipose tissue[48]. These findings are also concordant with the reported association between low skeletal muscle LPL activity and increased systemic respiratory quotient (hence reduced fat oxidation)[49]. It is becoming clear that exercise with the body's large muscles has the potential to influence fat balance, and hence (in the long term) body composition through changes to disposition of dietary lipids.

Interactions with changes to amount and distrbution of body fat

Discussion of the interactions of diet and physical activity is incomplete without mention of the relations with body fatness. Obesity is associated with dyslipidaemia[50], in particular with elevated plasma concentrations of TAG, low concentrations of HDL cholesterol, high levels of apolipoprotein B, a preponderance of small dense LDL and high levels of postprandial lipaemia. Derangements are exacerbated for a given level of obesity when the intra-abdominal fat mass is enlarged[50].

People who take up regular exercise tend to lose a small amount of body weight or fat[51] and this may contribute towards associated changes in lipoprotein lipids[52]. Direct comparison in a randomized trial of the effects on lipoproteins of restricting food intake with a comparable energy deficit achieved through increased exercise makes the point clearly[53]. Weight loss (3.5–7.5 kg over a 7-month period), whether through exercise or dieting, resulted in similar decreases in TAG and increases in HDL cholesterol.

There is evidence that changes in lipoprotein lipids are associated with loss of weight and/or fat, particularly when abdominal fat is lost[54]. For example, exercise-induced fat loss was significantly associated with increases in HDL cholesterol[55], changes in the LDL cholesterol to HDL cholesterol ratio[56], fasting TAG concentrations[56,57] and with changes in the chemical composition of LDL (more cholesterol-enriched and protein-poor)[57].

Changes in the amount and distribution of body fat with exercise thus probably contribute towards the effects on lipoprotein metabolism attributable to exercise. This conclusion is confirmed by the findings of meta-analysis of 95 studies of exercise training[58]. Although significant changes in HDL cholesterol and in TAG were found for studies where body weight did not change, for both these variables the effect of exercise was heightened when body weight loss accompanied training[58].The increase in HDL cholesterol averaged 3.3% (0.04 mmol $\cdot l^{-1}$) in 33 studies where body weight did not change, compared with 4.9% (0.06 mmol $\cdot l^{-1}$) when subjects lost weight.

Summary

Regular physical activity increases the flux of lipid substrates, in relation to the energy expended. This results in an enhanced metabolic capacity for TAG, a reduction in fasting and postprandial levels of TAG and an increase in the cholesterol carried in HDL. Although there is no clear threshold of benefit, it has been

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suggested that most changes to HDL cholesterol occur at distances of 11.3–22.5 km (7–14 miles) per week[59]. Such exercise levels are easily met by a slow jog or a brisk walk of about 30 min. on most days of the week. As there appears to be close links with energy expended in exercise[12,44] it is not surprising that more exercise appears to exert a progressively greater effect on both HDL cholesterol and TAG[19]. These changes occur despite the increased intake of energy and carbohydrate which usually accompanies higher levels of physical activity, showing that exercise can offset the hypertriglyceridaemic and HDL-lowering effects of high carbohydrate diets. There is some evidence, particularly for individuals with low HDL at outset, that only a combination of dietary change and increased activity is effective in modifying concentrations of plasma lipoprotein lipids.

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