The recognition of the cardiovascular risk in a sedentary lifestyle and of the benefits of regular exercise have led to the promotion of sport as a means to improve health and prevent certain diseases. However, the response of the lipid profile to an exercise session or training program is different depending on the type of exercise undertaken, its intensity and frequency, the duration of each session, and the time spent in such a program.1

The lipids stored in the body are an almost limitless source of energy during exercise, and their use increases with its duration. The fatty acids used in muscular metabolism are provided by the adipose tissue, by circulating lipoproteins, and by the triglycerides stored in the muscle cells themselves. Increased sympathetic-adrenal activity and a reduction in insulin concentration are the main stimulants of lipolysis during exercise. Resistance training is associated with increased beta-adrenergic sensitivity of the adipose tissue, and this provokes the enhanced use of fatty acids as an energy source. This adaptive phenomenon reaches its peak four months after starting resistance training. Exercise undertaken at intensities beyond the anaerobic threshold (i.e., when the participation of anaerobic, lactic metabolism increases to a point that it creates a state of imbalance with respect to the body’s buffering systems) leads to an increase in the blood lactate concentration that facilitates the recombination of free fatty acids and glycerol to form triglycerides. This reduces the availability of free fatty acids as an energy source, leaving carbohydrates as the main source of energy during intense exercise. In addition, fatty acids are preferentially oxidized in oxidative muscle fibers (type I fibers), which are recruited and activated during moderate intensity exercise.

Given the above, it is understandable that not all exercise or sporting activity will have the same effect on the lipid profile. Unfortunately, on many occasions the terms “physical exercise” and “sport” are used as synonyms, even though they describe different concepts. Physical exercise should be understood as planned, structured and repetitive exercise with the goal of maintaining or improving one or more components of physical fitness. Sport, though difficult to define, is a physical and intellectual human activity, competitive in nature and governed by institutionalized rules. The competitive character of sport is present in all its forms, though it is more evident in some than in others. Sports can be very different from one another, as can be the factors that limit both them and their specialities.3 This leads to many difficulties when trying to classify sports with the meeting of training needs in mind. According to their bioenergetic characteristics, sports can be classified as: a) aerobic, in which long duration, light-moderate intensity exercise dominates and in which the oxygen supply is essential for obtaining energy (e.g., the marathon, long distance swimming, tour cycling); b) lactic anaerobic, consisting of short, high intensity exercise in which energy is provided by ATP and phosphocreatine (e.g., 50 m and 60 m track events); c) lactic anaerobic, short duration, high intensity exercise (e.g., 400 m track events), and d) mixed, aerobic/anaerobic exercise (e.g., soccer, basketball, volleyball). According to the methodology of sports training, sports are classed as: a) strength and explosive strength sports (weightlifting, jumping, throwing); b) combat sports (boxing, fencing, judo); c) endurance sports (medium and long distance track events, swimming [except for events under 100 m], tour cycling); d) ball sports (soccer, volleyball, basketball), and e) coordination and competitive art sports (gymnastics, synchronized swimming, etc). Comparing the effects of different sports on the lipid profile can therefore be very difficult since, although the same energetic pathways are used, the intrinsic characteristics of training—and especially competition—can vary.

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Correspondence: Dra. A. Boraita.
Centro de Medicina del Deporte, Consejo Superior de Deportes.
El Greco, s/n. 28040 Madrid. España.
E-mail: araceli.boraita@csd.mec.es

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substantially. In individual endurance sports in which exercise is continuous (e.g., long distance running, swimming events over 400 m, cycling), the intensity of exercise can be regulated by the individual competitor, but team games involving a rival team and a state of play (such as soccer or volleyball) demand increases in the intensity of effort and the use of anaerobic energy pathways. Metabolic changes vary according to the predominant pathway used, which might be different when a person is competing or training.\(^4\) When the effects of different sports on the lipid profile are analyzed, the conclusions drawn can be contradictory if the above are not taken into account.

Such precautions are necessary when dealing with the paper published in this issue of the Revista Española de Cardiología by the Department of Medical Physiology of the Faculty of Medicine, University of Granada,\(^5\) which compares the lipid profiles of 28 swimmers, 17 volleyball players, and 23 soccer players. Though all show a lipid profile within the normal reference range, analysis of the interactions between lipoprotein(a) (Lp[a]) and apolipoprotein (apo) B100 and other cardiovascular risk factors (such as low density lipoprotein cholesterol [LDL-C] and the ratio between total cholesterol and high density lipoprotein cholesterol levels [HDL-C]) appears to show the swimmers to have the most favorable profile and the soccer players the least favorable. This might lead one to believe that, with respect to the lipid profile, there are “good sports” and “bad sports,” and that ball sports should not be practiced because of their negative influence on cardiovascular risk. However, it should be taken into account that all the sportmen of this study practiced competition sport, with all its implications in terms of performance demands. Furthermore, not all the subjects trained or competed for the same number of hours each week—a factor which could influence the results.

The paper also compares three very different sports (with some common features). During competition, long distance swimming and soccer make a demand of some 10 MET, whereas volleyball—a very explosive and predominantly anaerobic sport—demands 4 MET. In volleyball and soccer, exercise is intermittent and of high intensity, while swimming is of moderate intensity but continuous. If the dynamic characteristics of training are also taken into account, the swimmers undertook training involving continuous exercise with a 95% aerobic component, whereas the volleyball and soccer players undertook high intensity, intermittent exercise with a 60% and 70% aerobic component respectively. The methodology of training is also different. Swimming is an individual exercise with practitioners introduced at an early age, whereas soccer and volleyball are team sports that require greater sporting maturity. Consequently, though this study does right to analyze the interactions between Lp(a) and apo B100 with other cardiovascular risk factors in competitive sportsmen, its results should not be extrapolated to the practice of sports with the aim of improving cardiac health.

A large amount of scientific evidence is available regarding the positive effects of moderate intensity aerobic exercise on the lipid profile, among which the favorable modifications to lipoprotein metabolism (due to the increase in the cardioprotective HDL-C fraction) stand out. The benefits with respect to blood lipids include reductions in total cholesterol, LDL-C, and triglycerides, and increases in the HDL-C fraction as well as the HDL2-C, HDL3-C, and apo A-I subfractions. Increased lipoproteinlipase activity has also been seen in relation to the above changes in triglyceride and HDL-C levels.\(^6,7\) The majority of researchers report reductions in plasma triglycerides and increases in HDL-C when persons with normal cholesterol levels undertake physical exercise, and although less common, some papers show similar changes in the lipid and lipoprotein levels of hypercholesterolemic patients.\(^8\) These responses, however, might be different depending on whether this exercise is accompanied by changes to the diet and in body composition. Carbayo et al\(^9\) studied the changes that occurred in HDL-C levels caused by the practice of moderate physical exercise and its cessation, and found increased HDL-C levels (at the expense of the HDL2-C and HDL3-C subfractions) when exercise was performed with energetic equilibrium and with no anthropometric modifications. When the energy balance was positive and weight and body mass index increased, HDL-C also increased—although at the expense of HDL3-C alone. This response is related to modifications in the activity of hepatic lipase, an enzyme that favors the conversion of HDL2-C to HDL3-C. It is probable that its action influences the reduction of the HDL2-C subfraction in the presence of body fat.

A number of studies have described transitory changes in the lipid metabolism of sedentary males with normal or increased baseline cholesterol levels following an exercise session. Grandjean et al\(^10\) compared the changes in plasma lipid and lipoprotein levels in sedentary males with normal and high cholesterol levels following a session of aerobic exercise with an intensity equivalent to 70% maximum oxygen consumption. Both groups showed a reduction in total cholesterol and LDL-C immediately afterwards, and both returned to baseline levels 24 h later. However, the reduction in serum triglycerides and the increase in HDL-C and HDL3-C, as well as the increase in lipoproteinlipase activity, lasted longer—at least 48 h. Therefore, in untrained males, and independent of their starting cholesterol levels, there appears to be a lipid response to aerobic exercise which might be partly explained by increased lipoproteinlipase activity. Unfortunately, hardly any studies have recorded the behavior of lipid levels over the course of a training
program. Crouse et al. studied the responses of sedentary males with high cholesterol levels to exercise sessions of different intensity, as well as the adaptations made to a training program over 24 weeks. A single session on a cycloergometer led to transitory changes in plasma lipid and lipoprotein concentrations, independent of exercise intensity. The modifications to total cholesterol, triglyceride, HDL-C, HDL3-C, apo A-I, and apo B levels were statistically significant in samples taken after exercise (from post-effort to 48 h later), independent of exercise intensity or training condition. However, increases in HDL2-C levels were related to exercise intensity.

Several factors should be remembered when trying to establish relationships between lipid variables and training, such as the initial levels of serum lipids and lipoproteins, age, the maximum consumption of oxygen, body weight, and the percentage of body fat. In a metaanalysis, Wilmore investigated the influence of age, sex and health on the modifications to plasma triglycerides and HDL-C caused by physical training. Age appeared to have little or no influence on the adaptations made. In females, the HDL-C response was more attenuated than in males, but no difference was seen between the sexes with respect to the triglyceride response. The intensity and the type of training appear to be important, but the papers that report results in this area show marked inconsistency. With respect to results published over the last thirty years, León and Sánchez note increased HDL-C levels to be the most common positive finding regarding the lipid profile following 12 week aerobic training programs of moderate-high intensity. However, no threshold above which benefits are obtained can be set. Crouse et al. observed that men with hypercholesterolemia subjected to aerobic training for 24 weeks at intensities of at least 50%-80% maximum oxygen consumption showed significant changes in their HDL2-C, HDL3-C, apo A-I, and apo B levels independent of the intensity of exercise. However, Allen et al. observed a paradoxical decrease in the concentration of HDL-C in healthy men following a nine week high intensity training program—even though they showed significant increases after moderate training. Exercise would therefore appear to have no effect, or have a negative effect, at the anaerobic threshold. Sgouraki et al. in a study of top class male competitors of four different sports—long distance running, swimming, basketball, and wrestling—found that after a maximum exercise session (100% maximum oxygen consumption) the runners showed the greatest increase in HDL-C (mainly at the expense of HDL2-C), and that this might bear a relationship with their better aerobic adaptation. Other authors have also studied the response to exercise of different intensity and report small differences in the lipid profile; it is therefore unclear to what extent exercise intensity influences lipemia.

The frequency of exercise sessions appears to be more important than exercise intensity for improving these variables. A greater number of sessions per week has been related to a greater concentration of HDL-C and a reduction in the LDL-C/HDL-C and total cholesterol/HDL-C ratios. Since the responses of lipids and apolipoproteins to exercise last longer than 48 h after exercise ends, a frequency of at least one exercise session every 2 days would seem coherent when prescribing an exercise program: this would maintain any responses over time and, over the months of the program, obtain the metabolic adaptations desired. The duration of the sessions and the period over which the sport is practiced appear to be important in obtaining benefits with respect to the lipid profile. The duration of specific exercise sessions, apart from the warm-up and recovery periods, should vary between 30 min and 60 min, depending on the initial level of physical activity. In populations of young people it has been shown that periods of 6-12 months are sufficient for increases in HDL-C to be achieved. From the beginning of regular, moderate exercise programs, adults older than 50 can benefit from an ameliorated physical condition and small improvements in their HDL-C levels. However, the time required to achieve adaptations to lipid metabolism can be longer than that required by younger people. As well as regularity, this older population needs more prolonged exercise programs—at least 2 years—to achieve an improvement in HDL-C levels.

In summary, the intensity and the duration of physical activity necessary to achieve beneficial effects on the lipid profile are difficult to define. Nonetheless, prolonged exercise appears to be necessary to influence HDL-C and LDL-C levels. Results such as those reported by Ruiz et al provide new information on the possible effects of competition level sport on the lipid profile. The interactions between Lp(a) and apo B100 and other cardiovascular risk factors suggest that high intensity mixed sports with a strong dynamic component, eccentric muscle contractions and high muscle and joint impact, such as soccer, can be associated with less favorable lipid profiles. On the other hand, sports with a high dynamic component but with concentric muscular contraction and low joint impact, such as swimming, could be more beneficial. However, much has still to be learned if we are to discern the physiological mechanisms involved in the modification of lipid profiles caused by competition level sport. Further work is necessary comparing different levels of dedication to the same sports. Until then, patients with dyslipidemia should undertake moderate intensity, aerobic sporting activities, but given that the effects of this on lipid metabolism are reversible and can even disappear if training is not continued, physical exercise needs to become part of their regular lifestyle.
REFERENCES


