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Review

Physical activity and postprandial lipidemia: Are energy expenditure and lipoprotein lipase activity the real modulators of the positive effect?

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ABSTRACT

Historically, the link between elevated cholesterol and increased risk of cardiovascular disease has been based on fasting measurements. This is appropriate for total, low-density lipoprotein and high-density lipoprotein cholesterol. However, triglyceride concentrations vary considerably throughout the day in response to the regular consumption of food and drink. Recent findings indicate that postprandial triglyceride concentrations independently predict future cardiovascular risk. Potential modulators of postprandial lipidemia include meal composition and physical activity. Early cross sectional studies indicated that physically active individuals had a lower postprandial lipidemic response compared to inactive individuals. However, the effect of physical activity on postprandial lipidemia is an acute phenomenon, which dissipates within 60 h of a single bout of exercise. Total exercise induced energy expenditure, rather than duration or intensity of the physical activity is commonly reported to be a potent modulator of postprandial lipidemia. However, the pooled results of studies in this area suggest that energy expenditure exerts most of its influence on *fasting* triglyceride concentrations rather than on the incremental change in triglyceride concentrations seen following meal consumption. It seems more likely that energy expenditure is one component of a multifactorial list of mediators that may include local muscle contractile activity, and other yet to be elucidated mechanisms.

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1. Introduction

The link between lipid metabolism and cardiovascular disease (CVD) is predominantly based on fasting cholesterol measurements. This is appropriate for total cholesterol, low-density lipoprotein (LDL) and high-density lipoprotein (HDL) cholesterol as these measures vary little in response to feeding. However, triglyceride (TG) concentrations vary considerably in response to the consumption of lipid containing foods, meaning most individuals spend the majority of their day in a fluctuating state of lipidemia. Postprandial lipidemia (elevated TG concentrations in the blood after a meal) was first suggested as an independent risk factor for CVD over 30 years ago by Zilversmit [1]. He proposed that lipid

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accumulating in arteries was not only the result of elevated LDL concentrations, but was also a direct consequence of normal lipid absorption and transport [1]. Since then, chylomicron remnants have been identified trapped within the sub endothelial space [2], confirming a direct link between postprandial lipidemia and development of CVD. In addition, in a high TG environment, cholesterol ester is exchanged for TG in both HDL [3] and LDL [4] particles. Triglyceride enriched HDL is more rapidly catabolized, resulting in a lower HDL concentration [3], while TG enriched LDL is a substrate for both lipoprotein and hepatic lipase [4]. The removal of TG from the TG enriched LDL results in the formation of a more atherogenic small dense LDL particle [4]. Individuals who exhibit larger, and/or prolonged exposure to elevated TG concentrations after a meal, are exposed to an increased risk of CVD. This occurs, not only through the direct accumulation of chylomicrons into arterial walls, but also because the high TG environment promotes the development of a more atherogenic lipid profile [5].

Since the 1970s a multitude of research has been conducted into modifiers of postprandial lipidemia. However, it has only been in the last four years that prospective evidence has confirmed that non-fasting TG concentrations are a risk factor for CVD, independent of other lipid levels and markers of insulin resistance [6,7]. Long-term intervention studies to corroborate that lowering postprandial lipidemia results in reductions in CVD risk may be some time away. However, Zilversmit's hypothesis [1], combined with the role that a higher TG concentration plays in the development of a more atherogenic lipid profile, suggests that any reduction in exposure to high levels of postprandial lipids results in a slowing of the accumulation of lipid in the arteries. Thus improving the lipid profile and, thereby, reducing CVD risk.

For the purposes of this review, postprandial lipidemia is defined as the rise in TG concentration observed after consumption of a meal, beverage or individual food. Commonly, postprandial lipidemia is measured as the area under the TG response curve and can be expressed either as a total response (AUC), or as an incremental response (iAUC), in which measures of TG concentration are adjusted according to baseline values. Generally, postprandial TG response is observed as a slow and gradual increase in TG and a return to baseline over 8–10 h (provided no other food is consumed) [8]. The greater the magnitude and duration of the postprandial rise in TG (both of which will result in a larger iAUC) the greater the exposure of the arterial wall to dietary lipid and the greater the likelihood that TG will replace cholesterol ester in LDL and HDL particles.

Risk factors associated with increased postprandial lipidemia include genetics, sex and age. For a review of the effects of genetic polymorphisms on postprandial lipidemia see [9]. Men tend to have greater postprandial TG concentrations than women [10], although there is some indication that high postprandial TG concentrations are associated with greater risk in women [7]. Not unexpectedly, older individuals tend to have higher postprandial responses than younger individuals [10–12]. This review, however, is focused on lifestyle factors that can be modified to decrease prolonged and elevated exposure to postprandial lipids, with specific emphasis on physical activity. In particular, the effects of exercise and meal timing on postprandial lipidemia will be discussed, as well as how the intensity, duration, and energy expenditure of exercise may or may not influence postprandial lipidemia. Literature on this topic was acquired by conducting comprehensive searches in SciVerse Scopus and PubMed using the search terms *postprandial*, *lipidemia*, *lipidaemia*, *lipemia*, *lipaemia*, *physical activity*, *exercise*, and *resistance exercise*. All searches were limited to peer reviewed journals published in the English language prior to June 2011. In addition, reference lists of articles identified in the searches were surveyed to identify further literature matches. This review builds on that of Gill and Hardman [13] to provide an up to date summary of the literature available on lifestyle factors that can be modified to reduce postprandial lipidemia.

2. The effect of meal composition on postprandial lipidemia

In studies performed to investigate the effects of different amounts of dietary fat it has been found that intakes of 15 g or less do not significantly increase postprandial TG concentrations. Suggesting that $\leqslant$15 g of dietary fat does not challenge the TG clearance capacity of healthy participants [14,15]. Amounts of fat between 30 and 50 g, however, seem to increase postprandial TG in a dose-dependent manner [14,16–18] while amounts above 80 g result in an even greater postprandial TG response, which is no longer dose dependent [16,18].

The type of fat can also affect the postprandial TG response. Short and medium chain fatty acids enter circulation through the portal route, rather than through chylomicron secretion, meaning they have a minimal effect on the postprandial TG response. It is, therefore, likely that in studies that have used dairy fat as the predominant fat source, a lower postprandial response will have been observed than if other, longer chain fatty acids sources, were used [19]. Studies using fat sources that provide long chain fatty acids indicate that saturated (SFA), monounsaturated (MUFA) and n–6 polyunsaturated fatty acids (PUFA) do not have differential effects on the postprandial TG response [20,21]. However, the use of butterfat has produced both increased [22], and decreased [23] postprandial TG responses when compared to olive oil. This discrepancy could, at least in part, be due to the differing amounts of fat used in these studies, or that the fatty acid composition of butter can vary depending on the diet of the cow producing the cream for butter creation. Consuming $n-3$ fatty acids, however, can lower the postprandial TG response when compared to a fat source containing a mixture of fatty acids [9].

The effect of long-term modification of the fatty acid composition of the diet has also been investigated. Replacing SFA with MUFA (olive oil) for 8 weeks has been shown to result in an alteration of the shape of the postprandial TG response curve. The MUFA diet produced a higher initial peak, with a very slow decline over time, while the SFA diet produced a lower initial peak, followed by a plateau, and a second higher peak much later in the postprandial period [24]. However, this change in shape did not necessarily result in a difference in the calculated AUC response [25]. Consumption of MUFAs resulted in a faster appearance of lipid into circulation when compared to the consumption of SFA [24], as well as the production of fewer, larger chylomicrons [25]. Regular consumption of $n-3$ fatty acids has resulted in lower TG responses compared to $n-6$, SFA and MUFA [26–29]. Mechanisms proposed to explain this effect include a decreased production of VLDL, which in itself reduces TG concentrations while also decreasing the competition for lipoprotein lipase (LPL) [28], a slowing of entry of chylomicrons into circulation [18], and/or an up-regulation of LPL activity [30].

The nature of the carbohydrate added to a meal can also affect postprandial lipidemia. The addition of glucose (50, 100 g) to a fatty meal decreases the TG response, in a dose dependent manner [31], delaying the chylomicron response and suppressing the VLDL response [32]. Interestingly, a recent study reported this decrease in response occurs only in women and not men [33]. The addition of sucrose or fructose seems to have the opposite effect, increasing postprandial lipidemia, by reducing clearance of TG from circulation [34,35]. Even 50 g of fructose added to 5 g of fat, which by itself would not be enough fat to facilitate a significant change in TG in the circulation, results in a significant increase in the concentration of postprandial TG in circulation [15]. In healthy participants fed high, moderate or low glycemic index (GI) foods, there was