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Nutrition and Cardiovascular Disease

Glycemic index in relation to coronary disease

JC Brand-Miller

Human Nutrition Unit, University of Sydney, NSW, 2006

In cardiovascular disease, dietary fat and blood lipids have attracted the lion's share of attention. But carbohydrate, the macronutrient that increases when fats are restricted, may not be the totally desirable nutrient that we believe. The findings of the Lyon Heart Study, one of the most important nutrition studies ever carried out, emphasise that the 'prudent' high carbohydrate western diet is not the best choice for reducing cardiovascular events. One explanation is the potential to increase postprandial hyperglycemia, an under-recognised risk factor for cardiovascular and total mortality in the non-diabetic population. In the DECODE study and a host of other large prospective cohort studies, high post-challenge blood glucose was associated with 1.8 to 3 times greater relative risk of death. The glycemic potential of carbohydrates is therefore relevant to both prevention and management of coronary disease. Diets based on high glycemic index (GI) carbohydrate foods have been shown to 1) increase day-long blood glucose and insulin levels 2) exacerbate insulin resistance in predisposed individuals 3) adversely affect markers of the metabolic syndrome (triglycerides and HDL-cholesterol) in intervention studies and 4) increase the risk of coronary disease in a healthy population.

How does high blood glucose increase the risk of CVD? Laboratory studies have shown that high glucose levels even within the normal range adversely affect endothelial function via a multitude of mechanisms including oxidative stress, inflammatory factors, protein glycation, LDL oxidation, pro-coagulatory and anti-fibrinolytic activity.¹ In intervention studies of men with hyperlipidemia, Jenkins et al showed that a low GI diet was associated with lower TG and LDL cholesterol levels compared with an otherwise equivalent diet based on high GI carbohydrates.² In women with a family history of CVD following a low GI diet for 4 weeks, Frost et al found increased insulin sensitivity after a glucose challenge and increased glucose uptake in isolated adipocytes.³ Even in lean young adults, a low GI diet reduced muscle triglycerides, a marker of insulin resistance, despite no effect on insulin-stimulated glucose uptake.⁴

Epidemiological studies provide further support. In the Nurses Health Study, those in the highest quintile of GI and glycemic load (GI x carbohydrate) had nearly double the relative risk of coronary infarct, compared to those in the lowest quintile, after adjustment for known risk factors, including fibre.⁵ In several observational studies of healthy men and women, high GI diets have been consistently associated with lower HDL levels.⁶ In post-menopausal women, high GI diets were associated with higher C-reactive protein levels (a marker of low grade chronic inflammation), high triglycerides and lower HDL levels⁷, all of which increase the risk of CVD.

Low GI diets may also reduce visceral fat deposition. In recent studies, we compared 4 weight loss diets of differing glycemic load (GL). Compared to the conventional low fat diet with a high GL, the reduced GL diets produced greater rates of weight loss but only the low GI diet was associated with significant reductions in LDL-cholesterol (unpublished data). Finally, the STOP-NIDDM study using Acarbose (a drug which slows brush border digestion of carbohydrates) provides direct evidence that reducing the rate of carbohydrate absorption *per se* halves the risk of cardiovascular events and hypertension. The use of naturally-occurring 'slow-release' or low glycemic index (GI) carbohydrates to achieve the same end remains controversial.

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