The evidence for wheat bran related to obesity, diabetes and cardiovascular disease

Wheat is a leading human cereal crop. Wheat bran (WB) is a by-product of conventional milling of wheat grains and is a concentrated source of dietary fiber. Interest in WB as a source of dietary fiber and functional components has increased in recent years. There is considerable evidence indicating inverse correlations between wheat or WB consumption and cardiovascular disease (CVD) risk. In light of this evidence, WB has been increasingly utilized in food industry and the international dietary guidelines have endorsed increased consumption of whole grains and WB-rich products. Multiple roles have been claimed to explain the link between regular WB consumption and CVD risk including improvement in lipid profile, insulin sensitivity, body weight, blood pressure and lessening inflammation, although the exact mechanism is not yet fully elucidated.

Over years, CVD studies have ignored phospholipids as a biomarker for the disease risk prediction. Phospholipids are known to affect a number of cellular functions such as cell bioenergetics, growth, viability, proliferation, recognition, signal transduction and apoptosis. Altered phospholipid metabolism has been reported in many disorders, particularly obesity and diabetes. Early investigations have considered increased serum phospholipids, cholesterol and triglycerides as important predictors of CVD events.

Diets high in sugars like sucrose or fructose have been found to cause a 3-fold increased risk of death due to CVD. Impaired metabolism of cholesterol, triglycerides, phospholipids, glucose and uric acid, and insulin and leptin resistance are among the many CVD-related abnormalities associated with high sugar intake. In spite of these facts, controlled long-term studies that link the consumption of WB and sucrose with serum glucose and phospholipids in particular are generally lacking.

We investigated whether the consumption of high-sucrose diets with varying amounts of WB (0, 5, 10 or 20% w/w) had any effect on serum glucose, total cholesterol, triglycerides, low- and high-density lipoprotein cholesterol, phospholipids and total lipids in rats fed such a dietary regimen for periods of 4, 8, 12 and 16 weeks. Different feeding periods were included so that possible adaptation of the animals for the bulking effects of WB could be evaluated.

We demonstrated that in rats, the addition of varying amounts of WB to sucrose-based fiber-free diet induces time dependent changes in serum glucose, total cholesterol, and high-density lipoprotein cholesterol and food intake. The different WB diets had only apparently random effects on these variables. Both WB and fiber-free diets had similar effects on body weight, low-density lipoprotein cholesterol and triglycerides. Unlike the other lipid fractions, total lipids and phospholipids decreased progressively in response to dietary WB level irrespective to feeding duration.

It is important to note that the recorded effect for WB on glucose and lipids was reinforced by significant linear trend responses. With the exception of total lipids and phospholipids, these responses were seen to be time dependent. Thus, it is conceivable that a sort of adaptation in the mechanisms of glycemic and lipidemic control may have occurred as a result of prolonged feeding of dietary WB. These effects can also be associated with action of WB on food intake and body weight.

In summary, when incorporated into high-sucrose diets, varying amounts of WB appear to exert a profound reducing effect on glucose and lipids particularly the phospholipids in rats, in an interaction that is likely to have clinical implications in cardiometabolic conditions in humans**.** It would be of great importance to explore the mechanisms by which WB and sucrose interact and modify phospholipid assimilation and metabolism under sucrose diet conditions. This could be useful to lessen the debate surrounding the claim that consumption of whole grains including WB can reduce the risk of obesity, diabetes and CVD.

Mousa Numan Ahmad
Department of Nutrition and Food Technology, Human Nutrition and Dietetics
The University of Jordan
Amman, Jordan.

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