The effect of fiber-rich carbohydrates on features of Syndrome X

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ABSTRACT

There has been much debate among nutritionists and scientists regarding the optimal dietary approach for the treatment of the Insulin Resistance Syndrome, also called Syndrome X. This condition, which may affect as many as 47 million individuals in the United States, significantly increases risk of coronary heart disease and stroke. Major health organizations have historically recommended high-carbohydrate, low-fat (HCLF) diets to reduce chronic disease risk. However, there is evidence that a high intake of carbohydrates may adversely affect one or more of the abnormalities associated with this syndrome. Studies in this area have often had limitations. For example, some studies showing adverse effects of an HCLF diet have not taken into account the dietary fiber content of the diet. This article describes abnormalities often associated with Syndrome X, reviews the beneficial effects of fiber-rich carbohydrates, discusses the effect of fiber-rich carbohydrates on features of this syndrome, and concludes with applications of these findings for those involved in treating individuals with features of this disorder. This review indicates that an HCLF dietary pattern such as that used in the DASH trial, with a level of dietary fiber consistent with the recommendations of the American Dietetic Association (eg, 20-35g/day), containing from 3 to 10 g soluble fiber/day, may be beneficial for treating those with Syndrome X. *J Am Diet Assoc. 2003;103: 86-96.*

A high-carbohydrate, low-fat (HCLF) diet has traditionally been recommended by major health organizations for reducing chronic disease risk. Recently it has been suggested that an HCLF diet is inappropriate for individuals with the Insulin Resistance Syndrome, also referred to as the Metabolic Syndrome, or Syndrome X (1). Two arguments against the use of an HCLF diet in this population are generally cited. First, an increase in carbohydrate intake with a concomitant decrease in fat intake may produce adverse changes in blood lipid and lipoprotein concentrations, eg, an increase in blood triglyceride and small, dense low-density lipoprotein cholesterol (LDL-C) concentrations and a decrease in high-density lipoprotein cholesterol (HDL-C) (2-5). Second, HCLF diets may cause greater postprandial glucose and insulin concentrations in insulin-resistant individuals (1,6), possibly leading to further impairment in insulin action and glucose intolerance. This has led some to conclude that a higher-fat diet in which saturated fat is replaced with monounsaturated and polyunsaturated fat is preferable to an HCLF diet (1).

Although this approach may be effective for some, maintaining a higher fat intake may contribute to "passive overconsumption" (7). In this regard, most researchers in the area of weight management continue to recommend an HCLF diet (8). Importantly, studies showing adverse effects of HCLF diets generally have not taken into account the dietary fiber content of the diet. An HCLF diet that is rich in dietary fiber may not produce the same adverse changes in blood lipids and lipoproteins as an HCLF diet limited in dietary fiber (9). Additionally, the blood lipid and lipoprotein response to an increase in carbohydrate intake may differ depending on the type of fiber-rich carbohydrate consumed, eg, predominantly soluble vs insoluble fiber (10).

The purpose of this review is to discuss the characteristics of Syndrome X and to present findings related to the effect of fiber-rich carbohydrates, particularly those high in soluble fiber, on metabolic abnormalities associated with this syndrome. Practical applications for the utility of an HCLF diet, which includes fiber-rich carbohydrates for individuals with this syndrome, are presented.

DESCRIPTION OF SYNDROME X

Syndrome X refers to a condition in which several specific abnormalities (Figure 1) are clustered together with insulin resistance as the primary defect. Increased intra-abdominal adipose tissue (eg, visceral obesity) may be the initial culprit in the development of this syndrome (11). Elevated stress-related cortisol secretion (12), abnormal uric acid metabolism, and polycystic ovary syndrome (1) have also been associated with this syndrome. Recently, elevated concentrations of C-reactive protein were found to be strongly correlated with waist circumference and visceral fat

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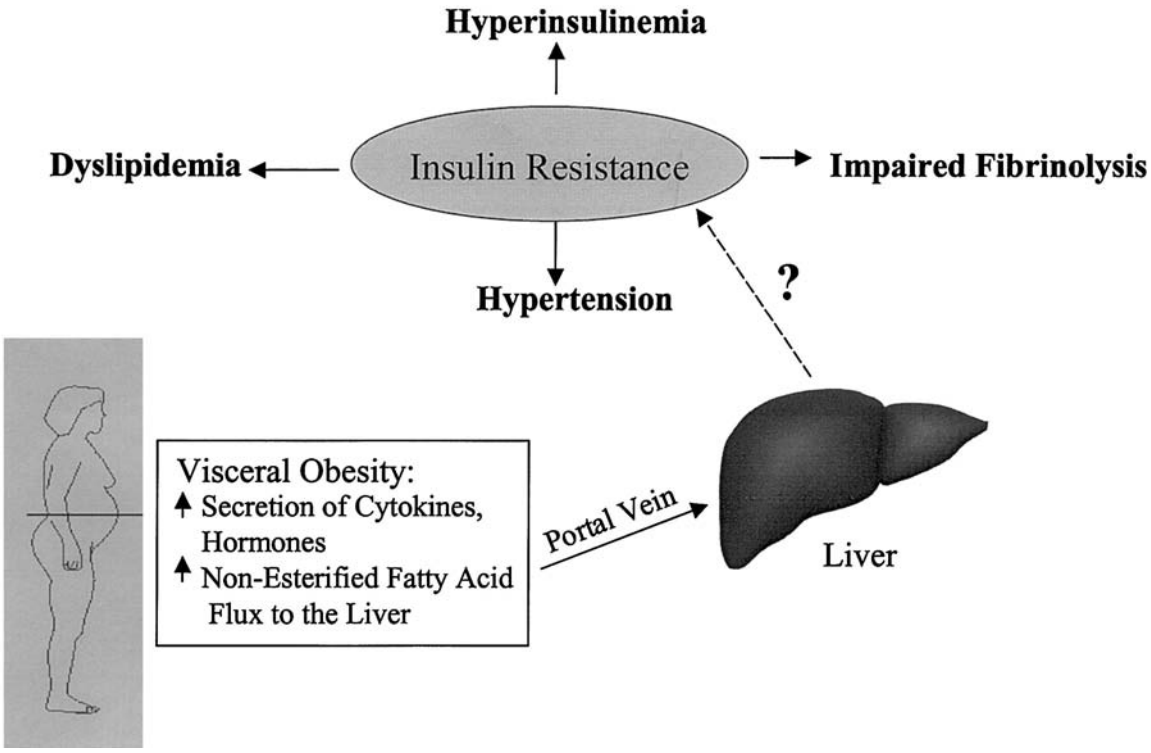

FIG 1. Syndrome X: the Insulin Resistance Syndrome.

(13), suggesting that inflammation may be another abnormality associated with this syndrome.

PREVALENCE AND SIGNIFICANCE OF SYNDROME X

Estimates of the prevalence of Syndrome X in the United States vary depending on the source and criteria used to diagnose this condition. Prevalence has been reported to be 16% among white persons (14); 7% in white and African American men and women (15); and 10% and 6% in Native American men and women, respectively (16). In 2002, findings from the Third National Health and Nutrition Examination Survey (17) reported much higher overall prevalence rates (age-adjusted) of 24% for men and 23% for women.

The presence of this clustering of abnormalities has been found to increase risk of coronary heart disease (CHD) and stroke by factors of 1.28 and 1.64, respectively (18). The Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (19) has recognized the disorders of the metabolic syndrome as a secondary target of therapy. The presence of abnormalities associated with Syndrome X increase CHD risk "at any given LDL cholesterol level" (19).

RISK FACTORS FOR SYNDROME X

Genetics Versus Behavior

The development of Syndrome X is likely caused by interactions between and among genetic and behavioral factors, eg, diet and physical activity (20). There is evidence of familial aggregation with respect to features of this syndrome (21). Abdominal obesity is strongly heritable (20), with estimates ranging from 25% to 40%. It has been estimated that 50% of the variability in insulin-mediated glucose disposal in nondiabetic individuals is attributable to genetic factors, with the remaining 20% to 25% attributed to obesity and physical inactivity (22). The atherogenic lipoprotein phenotype (eg, pattern B, characterized by small, dense LDL particles) is under the influence of several genes (3). As an example of an interaction between genetics and behavior, diet composition plays a role in the expression of this trait (2). Heritability estimates for features of this syndrome are 25% to 40% for blood triglyceride concentrations, 50% to 60% for total cholesterol, 30% to 55% for HDL-C, and 50% for hypertension (20). Concordance rates for the presence of all three conditions in monozygotic twin pairs were five times the rates for dizygotic twin pairs, suggesting a strong genetic influence.

Increased Intra-abdominal Fat Accumulation

The physical characteristic commonly associated with Syndrome X is excessive abdominal visceral adipose tissue. Several mechanisms have been suggested regarding why visceral obesity might cause insulin resistance and Syndrome X (11). As depicted in Figure 1, visceral adipose tissue secretes factors (eg, cytokines and hormones) that drain into the portal vein and may alter hepatic metabolism. Second, the visceral adipose depot compared with subcutaneous adipose tissue more readily releases nonesterified fatty acids (NEFA), thereby increasing fatty acid flux to the liver. The increased hepatic NEFA uptake could increase hepatic glucose production while decreasing glucose oxidation, resulting in glucose intolerance (23,24); increase hepatic very low density

Table 1
Clinical identification of the metabolic syndrome[a]

Risk factor	Defining level
Abdominal obesity (waist circumference)	
Men	>102 cm
Women	>88 cm
Fasting triglycerides[b]	≥1.69 mmol/L
HDL-C[c]	
Men	<1.03 mmol/L
Women	<1.29 mmol/L
Blood pressure	≥130/≥85 mm Hg
Fasting glucose[d]	≥6.1 mmol/L

HDL-C=high-density lipoprotein cholesterol.
[a]Reprinted from reference 19.
[b]To convert mmol/L triglycerides to mg/dL, multiply mmol/L×88.6.
[c]To convert mmol/L cholesterol to mg/dL, multiply mmol/L×38.7.
[d]To convert mmol/L glucose to mg/dL, multiply mmol/L×18.0.

lipoprotein (VLDL)-triglyceride secretion, causing hypertriglyceridemia (11); and decrease hepatic insulin removal, leading to hyperinsulinemia (25). Thus, increased visceral fat accumulation may be the initial factor leading to the development of this syndrome.

A number of methods have been developed to determine the magnitude of abdominal visceral adiposity. Methods such as magnetic resonance imaging (MRI) and computed tomography (CT) scans are considered gold standard techniques for the direct quantification of this adipose tissue depot. In clinical practice, waist circumference (>100 cm) and abdominal sagittal diameter (>25 cm) may identify individuals at risk for Syndrome X–related metabolic disorders (26). Criteria developed by the National Cholesterol Education Program (19) for identifying individuals with this syndrome are listed in Table 1. Individuals may be diagnosed with the metabolic syndrome if they have three or more of the specified risk determinants.

FEATURES OF SYNDROME X

Insulin Resistance and Abnormal Glucose Metabolism

A primary feature of Syndrome X is insulin resistance. Specifically, the ability of insulin to promote glucose disposal in peripheral tissues such as skeletal muscle is impaired. This can be detected clinically by reduced insulin sensitivity (S_I) (eg, less than normal insulin-mediated glucose disposal), hyperinsulinemia, impaired glucose tolerance, and an elevation in fasting blood glucose concentration (Table 1). Although insulin resistance is usually defined based on impaired glucose transport, other actions of insulin may also be compromised, such as skeletal muscle glycogen synthesis, suppression of adipocyte lipolysis, and insulin-stimulated vasodilation. Importantly, fasting blood glucose concentrations are normal in many insulin-resistant individuals because of compensatory pancreatic insulin hypersecretion. Thus a fasting glucose sample by itself is often insufficient to detect insulin resistance.

A fasting glucose sample by itself is often insufficient to detect insulin resistance

The mechanism by which insulin resistance occurs is complex, but is seems to result from a defect in the phosphatidylinositol-3 (PI-3) kinase insulin signaling pathway. It has been suggested that an increased production of tumor necrosis factor (TNF)-α and/or excessive accumulation of fatty acids in nonadipocytes, phenomena potentially resulting from high levels of abdominal visceral adipose tissue, may contribute to this defect (27). Circulating insulin binds to the insulin receptor at the cell surface, initiating a series of intracellular events that includes PI-3 kinase activation. This signaling pathway (shown graphically in reference 27) eventually results in the translocation of the GLUT4 transporter from intracellular vesicles to the cell membrane, allowing glucose to enter the cell (27). With insulin resistance, there is a defect in one or more step(s) of this pathway so that GLUT4 translocation does not occur.

As S_I worsens, hyperinsulinemia and hyperglycemia may occur because of several factors, including: (a) decreased muscle and liver glucose use, causing increased hepatic glucose production and output (24); (b) insulin hypersecretion; and (c) decreased hepatic insulin removal (28). An increase in blood glucose concentration would stimulate the β cells of the pancreas to increase insulin secretion (eg, compensatory hyperinsulinemia), and elevated concentrations of NEFAs may impair hepatic insulin extraction (25). Increased insulin release combined with a reduction in hepatic insulin clearance would lead to hyperinsulinemia. Impaired glucose intolerance and type 2 diabetes will occur as pancreatic β cells fatigue and can no longer adequately compensate for the insulin resistant state.

The mitogen-activated protein (MAP) kinase insulin signaling pathway does not seem to be impaired in insulin resistant individuals. This pathway is responsible for the mitogenic functions of insulin, eg, cell division and differentiation. With the compensatory hyperinsulinemia resulting from insulin resistance, the signal through the MAP kinase pathway may be amplified, which could have significant implications with respect to cardiovascular disease. Endothelial and vascular smooth muscle cell proliferation may be increased (29) via this pathway, possibly resulting in increased vascular resistance and endothelial dysfunction, a key factor in atherogenesis. The expression of plasminogen activator inhibitor-1 (PAI-1) may be increased (29), causing abnormalities in the fibrinolytic system leading to blood hypercoagulability and thrombosis. Thus, an upregulation of this pathway may contribute to several of the abnormalities associated with the metabolic syndrome.

Dyslipidemia

The dyslipidemia associated with Syndrome X is characterized by elevated blood triglyceride concentrations, low concentrations of HDL-C, and elevated levels of small, dense LDL particles (30). Insulin resistance in the adipocytes results in failure of insulin to suppress lipolysis and as a result, lipolysis is unregulated. The result is increased NEFA flux to the liver. Consequently, hepatic VLDL-triglyceride production and release is increased (4) and clearance of VLDL-triglyceride is decreased (31), leading to significant elevations of VLDL-triglyceride. As shown in Figure 2, exchange of triglyceride and cholesterol ester between VLDL, LDL, and HDL particles results in the formation of triglyceride-enriched LDL and HDL particles, making them a favorable substrate for hepatic triglyceride lipase. Small, dense LDL and HDL particles result. The former are more susceptible to oxidation and are less readily cleared (32), making them atherogenic. The latter are more readily cleared from circulation, resulting in a decrease in HDL-C con-

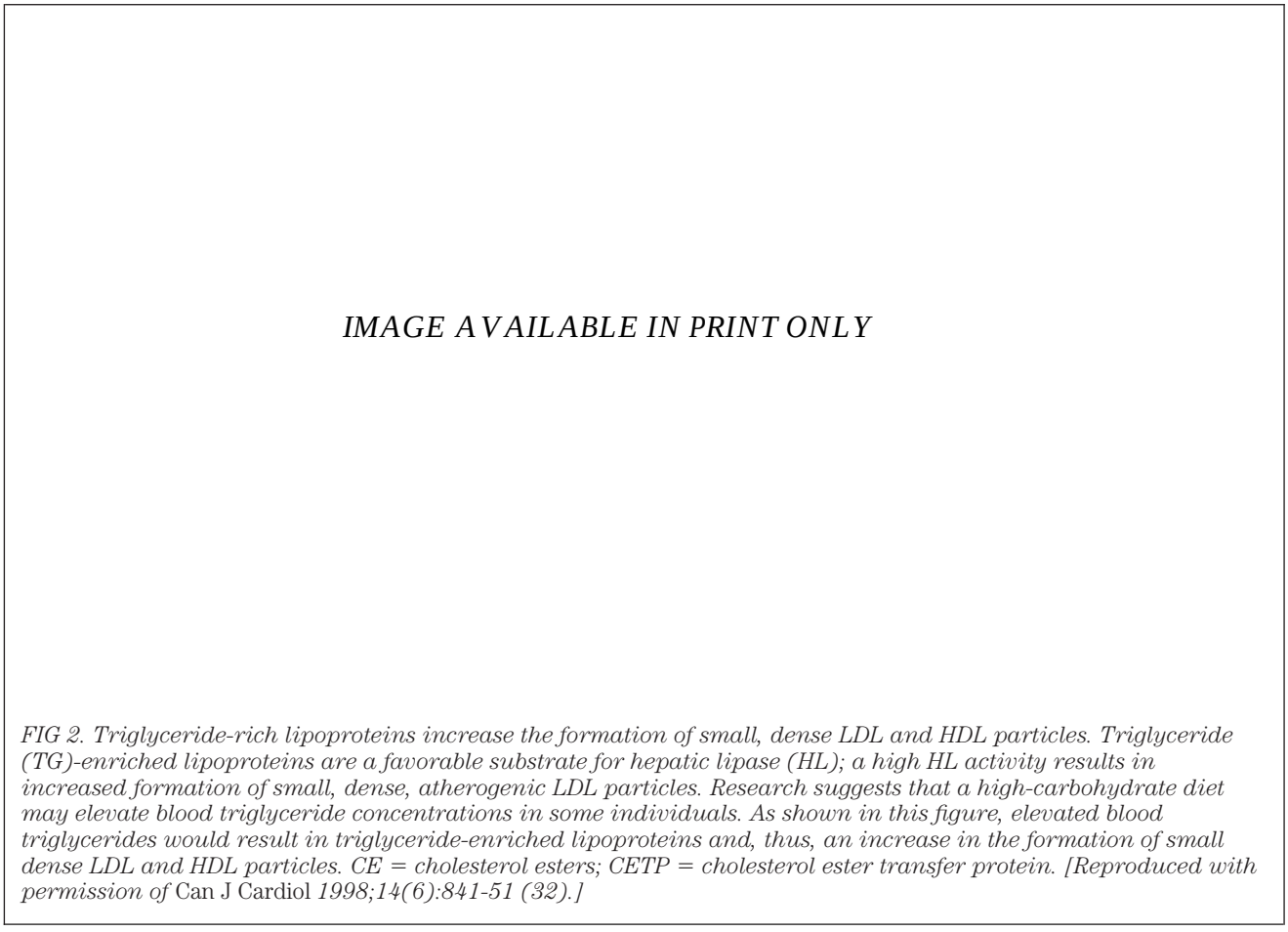

FIG 2. Triglyceride-rich lipoproteins increase the formation of small, dense LDL and HDL particles. Triglyceride (TG)-enriched lipoproteins are a favorable substrate for hepatic lipase (HL); a high HL activity results in increased formation of small, dense, atherogenic LDL particles. Research suggests that a high-carbohydrate diet may elevate blood triglyceride concentrations in some individuals. As shown in this figure, elevated blood triglycerides would result in triglyceride-enriched lipoproteins and, thus, an increase in the formation of small dense LDL and HDL particles. CE = cholesterol esters; CETP = cholesterol ester transfer protein. [Reproduced with permission of Can J Cardiol *1998;14(6):841-51 (32).]*

centrations (32). A predominance of small, dense LDL particles has been termed LDL subclass pattern B, or atherogenic lipoprotein phenotype. Individuals with this phenotype typically have elevated concentrations of blood triglycerides and low concentrations of HDL-C (3).

Under normal circumstances, blood chylomicron concentrations increase as the result of food ingestion; these exogenous triglyceride-rich lipoproteins compete with endogenous triglyceride-rich lipoproteins (eg, VLDL-triglyceride) for clearance by the liver. Chronically elevated levels of VLDL-triglyceride may decrease chylomicron clearance and thus produce postprandial lipemia. If atherogenesis is a postprandial phenomenon (33), this feature of the metabolic syndrome may be particularly detrimental to cardiovascular health.

Hypertension

The exact mechanism by which insulin resistance and/or hyperinsulinemia results in elevated arterial blood pressure is unclear (34), but several possibilities have been suggested. Reaven (1) reports that that blood pressure is elevated with Syndrome X because of one or more of the following: (a) impaired endothelial function with impaired vasodilation as the result of dyslipidemia; (b) increased renal sodium reabsorption, which would increase plasma volume and thus, cardiac output; or (c) increased sympathetic nervous system activation caused by hyperinsulinemia, which may also cause renal vasoconstriction and increased sodium retention (35).

Additionally, under normal circumstances insulin has vasodilatory effects. Because administration of the nitric oxide (NO) synthase inhibitor L-NMMA prevents insulin-induced vasodilation, the vasoactive actions of insulin are likely caused at least in part by an increase in NO-induced endothelial vasodilation (35,36). If peripheral insulin resistance leads to a loss in normal vasodilation (34,36), elevated arterial blood pressure could result.

Hyperinsulinemia may cause hypertrophy of blood vessel walls as the result of enhanced growth factor activity (via increased flux through the MAP kinase insulin signaling pathway), leading to a narrowing of the lumen of resistance vessels (34). Other hypothesized mechanisms have been suggested (34,36).

Finally, it has been suggested that the relationship between insulin resistance and blood pressure may be parallel consequences in that risk of both disorders increases with advancing age, obesity, and physical inactivity (36).

Impaired Fibrinolysis

Insulin resistance is associated with elevated concentrations of PAI-1 (37), a key enzyme involved in the fibrinolytic system. In this system, the plasminogen activators t-PA and u-PA activate

plasminogen, which in turn activate plasmin (38). Plasmin is a proteolytic enzyme that degrades fibrin and other extracellular matrix proteins. PAI-1 inhibits t-PA and u-PA, thus inhibiting the activation of plasminogen and decreasing fibrin degradation. In simple terms, this means that elevated levels of PAI-1 increase the likelihood of clot and plaque formation because of a decreased clot-busting ability.

Synthesis of PAI-1 is increased by cytokines such as interleukin-6 (IL-6) and TNF-α and hormones such as insulin (38). Adipose tissue secretes PAI-1, with more PAI-1 secreted by visceral than subcutaneous adipose tissue (39).

Increased concentrations of fibrinogen and factor VII may also be related to the impaired fibrinolysis associated with this syndrome, but will not be discussed here.

C-Reactive Protein

As a blood marker of chronic inflammation, C-reactive protein may be an indicator of endothelial stress or damage. Excessive abdominal fat may increase cytokine flux (eg, IL-6 and TNF-α), which in turn may increase in hepatic C-reactive protein synthesis (40). C-reactive protein concentrations have been strongly correlated with waist circumference and visceral fat (13).

The body of literature encompassing the abnormalities associated with the Metabolic Syndrome is evolving. There continues to be much debate regarding the best dietary approach for this population. Previous studies have had methodological limitations, making it sometimes difficult to draw clear conclusions about potential dietary treatment approaches. For example, studies showing adverse effects of HCLF diets have generally not taken into account the type or amount of dietary fiber consumed. Specifically, the blood lipid and lipoprotein response to an increase in carbohydrate intake may differ depending on the type of fiber-rich carbohydrate consumed, eg, predominantly soluble vs insoluble fiber (10). This area will be reviewed in the following section.

BENEFICIAL EFFECTS OF FIBER-RICH CARBOHYDRATES

There is a vast body of literature supporting the beneficial effect of fiber-rich carbohydrates for optimal health and disease prevention. It is generally accepted that consumption of fiber-rich carbohydrates is important for gastrointestinal health by decreasing constipation and reducing risk of diverticular disease (41,42). A high intake of fiber-rich carbohydrates is beneficial for weight management (42,43) and for reducing risk of cardiovascular disease (44,45), diabetes (42,46,47), and stroke (48). A high fiber intake may reduce risk of certain types of cancer, specifically colorectal and breast cancer (49-51). However, these relationships are still unclear (52,53).

Because of the different physiologic and metabolic effects of various types and sources of fiber-rich carbohydrates, health benefits differ according to specific type of fiber consumed (eg, soluble vs insoluble). Although studies sometimes do not distinguish between soluble and insoluble fiber (44,48), others have documented the benefit of a specific type of fiber. For example, foods rich in soluble fiber (eg, oats, beans, psyllium) have been shown to improve blood lipid and lipoprotein concentrations (54-57). Studies investigating the effects of wheat bran, rich in insoluble fiber, have had inconsistent effects on blood lipids and lipoproteins (57-59). Anderson and colleagues (42) report that although all fiber-rich foods help prevent constipation and diverticulosis, foods rich in soluble fiber delay gastric emptying and enhance satiety, whereas foods rich in insoluble fiber increase fecal bulk and decrease gastrointestinal transit time.

It is somewhat controversial (60,61) whether or not the glycemic index of the fiber-rich carbohydrate is important with respect to health outcomes (62-65). Thus, it seems that all types of fiber have some health benefit but the specific benefit differs according to type of fiber-rich carbohydrate consumed.

Is there a benefit of increased fiber consumption, particularly for foods rich in soluble fiber, for individuals displaying the features of Syndrome X? There is some evidence that a diet rich in fiber may reduce thrombogenesis (66-68). Literature on the effect of fiber-rich carbohydrates on blood insulin and glucose parameters, blood lipids and lipoproteins, and arterial blood pressure may provide clearer answers.

Effect of Fiber-Rich Carbohydrates on Blood Insulin and Glucose Parameters

It has been suggested that increased consumption of high-carbohydrate, low-fiber foods could increase blood insulin concentrations and reduce insulin action (6,69). Such foods may also increase risk of non–insulin-dependent diabetes mellitus (46,47). Some critics of HCLF diets do not believe that dietary macronutrient composition will alter insulin action in individuals with insulin resistance (1,70) but could affect other abnormalities associated with the insulin resistance syndrome (70).

Specific information related to many of the studies cited in this and the following two sections are provided in Table 2. Supporting the belief that macronutrient composition may not influence fasting blood insulin and glucose parameters are findings from several studies (71-73). Some suggest that an HCLF diet may improve S_I (74-76). With respect to soluble fiber, there is evidence in stroke-prone spontaneously hypertensive rats that a high intake of soluble fiber may reduce fasting blood glucose concentrations and improve S_I by increasing muscle GLUT-4 content (77).

Findings of the effects of fiber-rich carbohydrates on insulin resistance are inconsistent, leading some to question the effectiveness of dietary fiber in reducing insulin resistance (69). Some of the inconsistencies may be caused by methodological differences, including the type of individuals studied, the types of dietary carbohydrates used, and the carbohydrate content of the test diets.

Effect of Fiber-Rich Carbohydrates on Dyslipidemia

The dyslipidemia associated with Syndrome X may be exacerbated by an HCLF diet. This phenomenon, termed carbohydrate-induced hypertriglyceridemia, was reviewed extensively by Parks and Hellerstein (5). Mechanistically, results of studies in this area are inconsistent with some concluding that diet-induced hypertriglyceridemia is the result of reduced VLDL-triglyceride clearance and not increased secretion or de novo lipogenesis (31), and others attributing it to increased VLDL-triglyceride secretion (because of increased hepatic fatty acid availability resulting from increased influx of fatty acids and/or decreased hepatic fatty acid oxidation) (4).

It is generally accepted that elevated triglyceride and small, dense LDL concentrations and low HDL-C concentration are important risk factors for cardiovascular disease (78-83). As depicted in Figure 2, increased blood triglyceride concentrations resulting from an HCLF diet may lead to increased concentrations of small, dense LDL and decreased concentrations

Table 2
Effect of fiber-rich carbohydrates on abnormalities associated with Syndrome X

Author, year (reference)	Study design	Major findings
Insulin resistance/abnormal insulin and glucose metabolism		
Garg and colleagues, 1994 (71)	Multicenter randomized crossover trial, feeding study 42 male and female NIDDM patients Diets[a]: 6 weeks each, HCLF: 55/30/15, 15 g dietary fiber/1,000 kcal vs HF: 40/45 (25% MUFA)/15, 11 g dietary fiber/1,000 kcal	No significant difference noted in fasting plasma glucose, insulin Day-long plasma glucose increased 12% and insulin increased 9% on HCLF vs HF diet
Garg and colleagues, 1992 (72)	Randomized crossover trial, metabolic ward feeding study 8 men with mild NIDDM Diets: 21 days each, HCLF: 60/25/15 vs HF: 35/50/15 (32% MUFA) Diets matched for dietary fiber (25 g/day)	No change in S_I on either diet No change in peripheral insulin-mediated glucose disposal on either diet
Ginsberg, 1996 (73)	Multicenter randomized crossover trial 85 subjects with markers for insulin resistance Diets: 7 weeks each, 55/29/16 (15% MUFA) vs 49/36/15 (21% MUFA) vs 49/36/15 (14% MUFA)	No difference in day-long glucose or insulin concentrations between diets No difference in postprandial glucose and insulin metabolism between diets
Lovejoy and DiGirolamo, 1992 (74)	Cross-sectional study 45 lean and obese subjects Examined associations between habitual dietary intake and insulin sensitivity	S_I was positively correlated with fiber intake but not carbohydrate intake
Fukagawa and colleagues, 1990 (75)	Controlled feeding study 12 healthy subjects Diet: 21-28 days each, HCLF: 68/14/18 (33 g dietary fiber/1,000 kcal) vs control: 43/42/18 (7 g dietary fiber/1,000 kcal)	HCLF diet significantly reduced fasting glucose and insulin concentrations and increased glucose disposal rates HCLF diet enhanced peripheral S_I
Strazinsky and colleagues, 1999 (76)	Randomized crossover design 14 healthy subjects Diet: 2 weeks each, HCLF: 54/25/21 (39 g dietary fiber) vs HF: 36/45/19 (28 g dietary fiber)	S_I was significantly higher on the HCLF as compared with HF diet Fasting glucose was lower and fasting insulin was unchanged on the HCLF vs HF diet
Dyslipidemia		
Chandalia and colleagues, 2000 (84)	Randomized crossover design, feeding study 13 subjects with type II diabetes mellitus Diet: 6 weeks each, MF: 55/30/15 (24 g total dietary fiber, 8 g of which were soluble fiber) vs HF: 55/30/15 (50 g total dietary fiber, of which 25 g were soluble)	HF significantly reduced TC by 6.7%, TG by 10.2% HDL-C and LDL-C were not significantly different on the MF vs HF diet
Brown and colleagues, 1999 (56)	Meta-analysis of 67 controlled trials	Soluble fiber decreases TC by 0.045 mmol/L[b] and LDL-C by 0.057 mmol/L per gram of soluble fiber TG and HDL-C were not significantly changed by soluble fiber
Ripsin and colleagues, 1992 (86)	Meta-analysis of 10 randomized controlled trials using oat products	3 g per day of soluble fiber from oats (~84 g dry oats) reduces TC by 0.13-0.16 mmol/L
Lovegrove and colleagues, 2000 (87)	Double-blind, placebo-controlled randomized design 62 healthy subjects Diet: subjects ate their habitual diet; assigned to consume either 3 g β-glucan (20 g oat bran concentrate) or 20 g wheat bran (control) daily for 8 weeks	β-glucan supplements did not significantly reduce TC or LDL-C HDL-C decreased significantly from 1.5 to 1.4 mmol/L in the β-glucan group from baseline values
Abbasi and colleagues, 2000 (90)	Randomized crossover feeding trial 8 healthy subjects Diet: 14 days each, HCLF: 60/25/15 (13.5 g dietary fiber/1,000 kcal) vs HF: 40/45/15 10.5 g dietary fiber/1,000 kcal), P:S ratio was matched Fasting and postprandial studies performed	HCLF diet resulted in higher fasting plasma TG, RLP cholesterol, RLP TG, and lower HDL-C as compared with the HF diet TC and LDL-C were not significantly different
Gerhard and colleagues, 2000 (91)	Randomized crossover feeding trial 22 healthy subjects Diet: 4 weeks each, HCLF: 65/20/15, 8% MUFA (15 g dietary fiber/1,000 kcal) vs HF: 45/40/15, 14% MUFA (9 g dietary fiber/1,000 kcal) Fasting and postprandial studies performed	Fasting TC and LDL-C were higher on the HF as compared with the HCLF diet Less postprandial lipemia noted after the HCLF than the HF diet
Hypertension		
Kestin and colleagues, 1990 (102)	Double-blind crossover design 24 hypercholesterolemic subjects Diet: 4 weeks each, high-fiber breads containing wheat, rice, and oat bran were provided contributing 12 g dietary fiber/day	No significant effect of any cereal bran on blood pressure was found
Swain and colleagues, 1990 (103)	Double-blind crossover trial 20 healthy subjects Diet: 6 weeks each, habitual diet plus either oat bran (18 g dietary fiber/day) or low-fiber wheat (2 g dietary fiber/day) incorporated into entrees or muffins	Blood pressure did not change during either dietary intervention
Fehily and colleagues, 1986 (104)	Randomized crossover design 201 healthy subjects Diet: 4 weeks each, high-cereal-fiber diet (31 g total dietary fiber) vs low-cereal-fiber diet (19 g total dietary fiber); macronutrient composition did not differ between the two diets	High-cereal-fiber diet did not alter blood pressure

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Table 2 (cont'd)
Effect of fiber-rich carbohydrates on abnormalities associated with Syndrome X

Author, year (reference)	Study design	Major findings
Davy and colleagues, 2002 (105)	Randomized double-blind design 36 men with high-normal to stage I hypertension Diet: 12-week intervention, subjects added either high-fiber oat or wheat cereals (14 g dietary fiber) to their habitual diet	Increased oat or wheat consumption did not alter resting casual or 24-hour ambulatory blood pressure
Strazinsky and colleagues, 1999 (76)	Randomized crossover design 14 healthy subjects Diet: 2 weeks each, HCLF: 54/25/21 (39 g dietary fiber) vs HF: 36/45/19 (28 g dietary fiber)	Resting blood pressure was lower on the HCLF as compared with the HF diet
Saltzman and colleagues, 2001 (107)	Randomized controlled feeding study 43 overweight subjects Diet: 6-week intervention, hypocaloric diet containing either 13 g (3.5 g soluble fiber/day) or 16 g total dietary fiber (containing oats, 7.2 g soluble fiber/day)	Hypocaloric diet containing oats reduced systolic blood pressure, whereas diastolic blood pressure remained unchanged

NIDDM=non–insulin-dependent diabetes mellitus.
HCLF=high-carbohydrate, low-fat.
HF=high-fat.
MUFA=monounsaturated fat.
S_I=insulin sensitivity.
MF=moderate-fat.
TC=total cholesterol.
TG=triglycerides.
HDL-C=high-density lipoprotein cholesterol.
LDL-C=low-density lipoprotein cholesterol.
RLP=remnant lipoprotein.
[a]Macronutrient distribution: carbohydrate/fat/protein.
[b]To convert mmol/L cholesterol to mg/dL, multiply mmol/L×38.7.

of HDL. Critics of HCLF diets often cite this phenomenon as a major reason not to use this diet approach (1).

Because of methodological differences between studies on the effect of HCLF diets on blood lipids and lipoproteins, it is not clear whether all HCLF diets will result in diet-induced hypertriglyceridemia. Some advocates of HCLF diets suggest that adequate amounts of fiber-rich carbohydrates will prevent carbohydrate-induced hypertriglyceridemia. Anderson (9) contends that his group has successfully used high-fiber (eg, 20 g/1,000 kcal) HCLF diets for treating hypertriglyceridemic individuals for 25 years. In support of this are findings from a recent study, which suggest that an HCLF diet with high levels of total and soluble fiber may prevent adverse lipid and lipoprotein changes in individuals with type 2 diabetes (84). Anderson (9) reports "the available literature persuasively documents that moderate to high levels of dietary fiber intake (eg, 10 to 20 g/1,000 kcal) attenuate the carbohydrate-induced hypertriglyceridemia observed with HCLF diets." He maintains that the carbohydrate-induced condition indicates a state of fiber deficiency that can be corrected with the consumption of moderate to large amounts of fiber-rich carbohydrates.

Considerable attention has been focused upon the lipid-lowering properties of soluble viscous fibers, including psyllium, oats, guar, and pectin. The cholesterol-lowering effect of these types of fiber is generally attributed to their ability to decrease bile acid absorption and increase fecal bile acid excretion. As a result, endogenous cholesterol synthesis is diverted from lipoproteins to bile acid synthesis (85), which lowers the hepatic free cholesterol pool. This reduction in hepatic cholesterol concentration upregulates LDL receptors, thus increasing LDL-C clearance. Other mechanisms have been suggested (45). Some have found that increased consumption of soluble fiber (3 to 10 g/day soluble fiber) significantly decreases blood total cholesterol (56,86) and LDL-C concentrations without significantly changing triglyceride and HDL-C concentrations (56). However, not all studies support these findings (87).

Insulin resistance and the pattern B phenotype have been linked to increased postprandial lipemia (88,89). As discussed earlier, chronically elevated levels of VLDL-triglyceride may decrease chylomicron clearance and contribute to postprandial lipemia in individuals with the metabolic syndrome; this may be exacerbated by HCLF diets (90). However, one investigation found that a high-fiber HCLF diet could reduce the lipemic response to a high-fat test meal (91). Thus, the very limited available information suggests that an HCLF diet may not worsen postprandial lipemia if the diet is also high in fiber.

With regard to diet-gene interactions, some data suggests that pattern B individuals may respond favorably to an HCLF diet (eg, reduced concentrations of small, dense LDL particles and LDL-C) (92). In a recent review, however, Krauss (3) reports that the prevalence of the pattern B phenotype increases with an increase in carbohydrate and a decrease in fat consumption. Several studies published on this topic seem to have conflicting results; thus, it is not clear whether an HCLF diet is advisable or detrimental for individuals with the pattern B phenotype (2,93,94).

With respect to fiber-rich carbohydrates, Davy and colleagues (10) found that pattern B individuals have a favorable lipid and lipoprotein response to an increased consumption of oat as compared with wheat cereal. This suggests that the lipid and lipoprotein response to increased carbohydrate consumption may depend on the type of dietary carbohydrate (eg, carbohydrates rich in soluble vs insoluble fiber) as well as LDL subclass phenotype.

Effect of Fiber-Rich Carbohydrates on Arterial Blood Pressure

Epidemiological studies consistently report that increased dietary fiber and/or whole grain consumption is associated with a reduced risk of CHD (44,95-97). Additionally, some have reported a significant inverse relationship with CHD risk and soluble fiber (97,98) and oat consumption (97), but this finding has not been universal (44,96).

An inverse association between dietary fiber intake and arterial blood pressure has been reported (99,100). This relationship has also been noted specifically with oat intake (101). However, very few clinical trials have been done that support a direct effect of fiber on blood pressure without weight loss. As shown in Table 2, a comparison of the consumption of three sources of fiber-rich carbohydrates on arterial blood pressure found no significant effect of any fiber source on blood pressure (102). Consistent with this are findings from several studies (103-105).

In contrast, a few published intervention studies report that a fiber-rich HCLF diet can lower arterial blood pressure (76,106,107). Results from the Dietary Approaches to Stop Hypertension (DASH) trial (106) suggest that an HCLF diet rich in fruits, vegetables, low-fat dairy products, and whole grains is effective in reducing blood pressure. Thus, the available literature suggests that either weight loss or a whole-food HCLF diet containing multiple sources of fiber-rich carbohydrates (fruits, vegetables, and whole grains), and not just the addition of a single source of fiber (eg, oats), may be necessary to significantly reduce arterial blood pressure.

CONCLUSIONS

Benefits of Fiber-Rich Carbohydrates for Weight Management

Weight loss, specifically the reduction of body fat, may be the most effective strategy for improving abnormalities associated with Syndrome X in overweight individuals (1,20). The degree of insulin resistance and/or hyperinsulinemia does not alter weight loss response to a hypocaloric diet (108). A moderate weight loss of ~10% can significantly enhance insulin clearance, thereby reducing hyperinsulinemia, although greater degrees of weight loss may be required to reduce insulin secretion and improve insulin resistance (28).

Ad libitum HCLF diets generally result in weight loss and would thus be preferable to a diet high in monounsaturated or polyunsaturated fat for insulin-resistant individuals (109). Fiber-rich diets may be less energy-dense and may promote greater feelings of satiety (41). Regarding weight maintenance, weight management experts recommend an HCLF diet for most individuals (8). Fiber-rich carbohydrates, particularly those high in soluble fiber, should be included as part of the maintenance HCLF diet to prevent adverse lipid and lipoprotein changes in individuals with this syndrome.

How Much Dietary Fiber?

The ADA recommends a daily fiber intake of 20 to 35 g/day for adults (41). In contrast to these recommendations, the mean dietary fiber intake of Americans is 15.1 g per day, and 18.2 g in males over age 50 (110). Recommended intakes for amounts of specific fiber types are generally not made, although it has been recognized that foods rich in soluble fiber have beneficial properties (19,41,111). The replacement of dietary fat with carbohydrate my have adverse effects on blood lipids and lipoproteins unless carbohydrates rich in soluble fiber are consumed (10). An intake ranging 3 g (86) to 8 to 10 g/day (10,56) may be advisable. Because individuals with the metabolic syndrome are characterized by the presence of dyslipidemia, these individuals would benefit from an HCLF diet that includes foods rich in soluble fiber such as oats.

Dietary Fiber Versus Other Components of Fiber-Rich Foods

It is not clear whether the health benefits of fiber-rich foods are directly attributable to their dietary fiber content alone or whether there are other components or properties of the foods (eg, essential fatty acids, micronutrients, antioxidants, and phytochemicals) that are responsible for their beneficial health effects (85). In support of this are findings of epidemiological studies, which have found that even after adjusting for fiber intake, consumption of whole grains is independently associated with a reduced risk of CHD (44), total mortality (112) and ischemic heart disease (113). Therefore, it seems likely that the beneficial effects of fiber-rich carbohydrates are attributable not only to their fiber content, but also to other properties of the food. Thus, the emphasis of dietary recommendations should be placed on the consumption of foods rather than nutrients.

Dietary Fiber and Fiber-Rich Foods Versus a High-Fiber Pattern of Intake

In recent years, major health organizations such as the American Heart Association and the United States Department of Agriculture have made dietary recommendations that are more food- and dietary pattern-based and less nutrient-based. This approach is more consumer friendly, allowing us to focus on aspects of our diet other than grams or percentages of nutrients.

The strongest evidence in support of this approach is findings from the DASH trial (106,114,115). The primary objective of this trial was to assess the effects of dietary patterns on blood pressure. The DASH dietary pattern was high in fruits (~5 servings/day), vegetables (~4 servings/day), grains (~8 servings/day), and low-fat dairy products (~2 servings/day); emphasized fish and chicken rather than red meat; and was low in total fat (~26%), saturated fat (~7%), cholesterol (~150 mg), sugar, and refined carbohydrates. Sodium intake (~3 g/day) and body weight were kept constant. The fiber content of the diet was approximately 30 g/day. After 8 weeks, the DASH diet significantly reduced resting systolic and diastolic blood pressure (106), 24-hour ambulatory blood pressure (114), blood total cholesterol, and LDL-C concentrations (115) as compared with a control diet (eg, typical American diet). Despite the relatively high carbohydrate content of the diet (~57% of total energy), blood triglyceride concentrations did not increase. A small but significant decrease in HDL-C concentration (0.09 mmol/L) was detected (115).

Because of the improvements in arterial blood pressure and blood lipid and lipoprotein concentrations (with the exception of the reduction in HDL-C), this type of dietary pattern may be beneficial for individuals with Syndrome X. The limited effectiveness of long-term weight loss maintenance might suggest that it would be worthwhile for future studies to investigate the effect of the DASH dietary pattern (without weight loss) on abnormalities associated with the metabolic syndrome.

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