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REVIEWS IN BASIC AND CLINICAL GASTROENTEROLOGY

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Dietary Fiber Supplements: Effects in Obesity and Metabolic Syndrome and Relationship to Gastrointestinal Functions

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Dietary fiber is a term that reflects a heterogeneous group of natural food sources, processed grains, and commercial supplements. Several forms of dietary fiber have been used as complementary or alternative agents in the management of manifestations of the metabolic syndrome, including obesity. Not surprisingly, there is a great variation in the biological efficacy of dietary fiber in the metabolic syndrome and body weight control. Diverse factors and mechanisms have been reported as mediators of the effects of dietary fiber on the metabolic syndrome and obesity. Among this array of mechanisms, the modulation of gastric sensorimotor influences appears to be crucial for the effects of dietary fiber but also quite variable. This report focuses on the role, mechanism of action, and benefits of different forms of fiber and supplements on obesity and the metabolic syndrome, glycemia, dyslipidemia, and cardiovascular risk and explores the effects of dietary fiber on gastric sensorimotor function and satiety in mediating these actions of dietary fiber.

Obesity is a risk factor for morbidity and mortality from cardiovascular, musculoskeletal, malignant, and metabolic diseases[1] as well as considerable social and financial burdens.[2] Poor compliance with behavior-modifying management programs and frequent regain of weight after the cessation of most medical therapies have led to the use of alternative, conservative approaches based on dietary fiber before considering bariatric surgery.

The potential beneficial effects attributed to dietary fiber[3] were based on earlier epidemiologic studies, indirect evidence,[4] claims of efficacy in a predominantly over-the-counter unregulated domain, and the perception of the public that if a product is natural, it is safe and efficacious. The scientific literature documents several favorable effects of dietary fiber on glucose homeostasis, lipid metabolism, and calorie intake. The gastrointestinal tract plays a role in these functions. The stomach signals satiation in response to a meal and affects the rate of delivery of macronutrients to the small intestine, which is the site for most nutrient and energy absorption. Gastric and small intestinal functions are integrated with glucose-regulatory mechanisms originating in the pancreas (eg, insulin) and the small intestine (specifically incretins, glucose-stimulated insulinotropic peptide [GIP], and glucagon-like peptide 1 [GLP-1]).

This review is written from the gastroenterological perspective and addresses biological properties of dietary fiber or fiber supplements that are relevant to obesity and the metabolic syndrome; efficacy of fiber on weight reduction, glycemic control, atherogenic dyslipidemia, hypertension, and total cardiovascular risk and the proposed mechanisms of these effects; and the role of gastric sensorimotor-modulated functions by dietary fiber.

Properties of Fiber

Dietary Fiber: Definition and Classification

The definition of dietary fiber is "the edible parts of plants or analogous carbohydrates that resist digestion and absorption in the human small intestine, with complete or partial fermentation in the human large intestine. It includes polysaccharides, oligosaccharides, lignin and associated plant substances. Dietary fiber exhibits one or more of either laxation, blood cholesterol attenuation and/or blood glucose attenuation."[5]

Dietary fiber includes several chemical classes: nonstarch polysaccharides (polyglucoses such as cellulose, hemicellulose and β-glucans, polyfructoses [such as inulin], and natural gums and heteropolymers such as pectin), oligosaccharides, lignin (a noncarbohydrate complex of polyphenylpropane units functionally linked to polysac-

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Abbreviations used in this paper: **CVD, cardiovascular disease; GIP, glucose-stimulated insulinotropic peptide; GLP-1, glucagon-like peptide 1.**

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charides, increasing resistance to digestion), fatty acid derivatives (waxes, cutin, and suberin, serving as cross-links between the main constituents), other plant substances (mucilages, storage polysaccharides, and phytates), and analogous polysaccharides (by-products of food production affecting digestibility, or purposefully synthesized compounds).[6,7]

A simpler classification divides dietary fiber into soluble (pectins, gums, and mucilages and storage polysaccharides) and insoluble fiber (cellulose, hemicelluloses, and lignin) on the basis of water solubility. Soluble fiber has favorable effects on glucose and lipid metabolism that are partly attributed to the increased viscosity of luminal contents.[8] Colonic fermentation of soluble fiber yields short-chain fatty acids, which may have beneficial effects on lipid metabolism, cardiovascular disease (CVD) prevention, mucosal differentiation or apoptosis, and mucosal barrier function.[9] Insoluble fiber also has a generally low fermentability, but it possesses passive water-attracting properties promoting fecal bulk, softening, and laxation.

Dietary Fiber Supplements

Table 1 provides a summary of the properties of commonly used dietary fiber supplements and potential (either established or investigated) effects on the metabolic syndrome.[10–14]

Fiber and Body Weight

Epidemiologic studies suggest an inverse relation of dietary fiber intake and body weight,[15,16] and this is supported by cross-sectional studies (with body mass index[17–19] or body fat mass[20,21]) and large observational studies (body weight gain in women[22] and in men[23]). Body weight gain was inversely correlated with the amount of whole grain ingested[23] in the large-scale study on Coronary Artery Risk Development in Young Adults (CARDIA).[24]

Efficacy of Dietary Fiber and Supplements on Weight Loss in Interventional Studies

A number of interventional human trials have shown weight reduction with diets rich in dietary fiber or dietary fiber supplements;[25–28] however, other studies failed to show any effect.[29,30] Recent meta-analyses of randomized controlled studies suggest only minor effects on weight loss for commonly used dietary fiber supplements. Data are summarized in Table 2[14,28,31–55] and Table 3.[30,56–60]

Proposed Mechanisms for the Effect of Dietary Fiber on Weight Reduction

Body weight and fat mass regulation result from a complex interplay of multiple factors, involving central nervous circuits, peripheral sensation stimuli, mechanical and chemical satiation signals arising in the gastrointestinal tract, afferent vagal input, and adiposity signals from fat tissue and liver.[61] The stomach signals satiation in response to volume and calories of the ingested meal;[62] a lower postprandial volume predicted an increased satiation score and a decreased maximum tolerated volume of a challenge meal test.[63]

In many studies, dietary fiber induced greater satiety compared with digestible polysaccharides and simple sugars.[64,65] Greater satiety may result from several factors: the intrinsic physical properties of dietary fiber (bulking, gel formation, and viscosity change of gastric contents),[66] modulation of gastric motor function, and blunting of postprandial glucose and insulin responses. Postulated effects on gut peptide hormones involved in signaling satiation (such as ghrelin, GLP-1, cholecystokinin, peptide YY, or GIP) remain incompletely resolved.[31,36,61,67–70]

Dietary fiber may also prolong meal duration and result in increased mastication with possible cephalic and peripheral influences on satiety.[71] Dietary fiber–containing meals have a lower energy density[65] and may affect palatability of food, possibly reducing energy intake.[72]

Fiber and Glucose Metabolism

Epidemiology and Mechanisms

Soluble dietary fiber is associated with lower postprandial glucose levels and increased insulin sensitivity in diabetic and healthy subjects, effects that are generally attributed to the viscous and/or gelling properties of soluble fiber.[73–75] Insoluble dietary fiber exerts negligible effects in postprandial glycemia. However, epidemiologic evidence suggests the opposite.[4,76–78] Soluble dietary fiber consumption did not reduce the risk of type 2 diabetes mellitus in observational studies[79,80] or in a meta-analysis including 328,212 subjects.[81] Insoluble fibers demonstrate the strongest associations with decreased risk of diabetes.[77,82] Increased consumption of cereal dietary fiber significantly reduced the risk of diabetes (relative risk, 0.67),[81] and a meta-analysis of 6 prospective studies indicates that a 2-serving-per-day increment in whole grain consumption may reduce the risk of diabetes by 21%.[83]

The mechanisms involved in the favorable effect of dietary fiber on glucose metabolism in humans appear to differ for soluble and insoluble fibers; moreover, additional factors modulate the glycemic effects of natural grain products.

Effects of soluble fiber. Soluble dietary fiber exerts physiologic effects on the stomach and small intestine that modulate postprandial glycemic responses. These include the following:

1. Delayed gastric emptying,[66,84] which accounts for approximately 35% of the variance in peak glucose concentrations after ingestion of oral glucose[85,86]

Table 1. Summary of the Properties of Commonly Used Dietary Fiber Supplements With Established or Investigated Effects in the Metabolic Syndrome

Fiber type	Water solubility	Fermentability	Molecular weight (*daltons*)	Chemical composition	Forms	Viscosity/gelation	Derivation	Medical uses
Guar gum	+ (no heating necessary)	High	50,000–8,000,000	Galactose/mannose = 1/2	Powder, added in composite flours[10]	High degree (low shear) under calcium cross-linking abolished by hydrolysis, ultra-high heating	Ground endosperm of *Cyanopsis tetragonolobus*	Hyperglycemia, hypercholesterolemia, obesity
Glucomannan	+ Enhanced by acetylation, in derivatives	High	10,000–1,900,000	Straight chain polymer, D-mannose/D-glucose = 1,6/1 (variable)[11]	Powder, added in composite flours, konjac pasta	Variable: Decrease in acetylation Increase in alkali, heating, mol wt, high glucomannan concentration	Roots of *Amorphophallus konjac*	Hyperglycemia, hypercholesterolemia, obesity (not FDA approved), drug delivery system
Plantago psyllium[12]	+	High	$7–20 \times 10^6$	Highly branched polymer, 22.6% arabinose, 77.4% xylose	Fibrous mucilage	High	Husks of ripe seeds of *Plantago ovata* and *Plantago psyllium* species	Irritable bowel syndrome, constipation, inflammatory bowel disease, obesity, diabetes, hyperglycemia
Pectin	+	High	60,000–130,000	D-galacturonic acid chain, variable L-ramnose substitutions, neutral sugars side chains	Powder, capsules	HM (>60%): hydrogen bonds, heat and pH sensitive LM (20%–40%): Ca^{2+} cross-linking, heat and pH resistant	Cell wall of citrus fruits, apples, and some vegetables	Antidiarrheal, drug delivery system
Alginate[13]	+ (sodium salt)	High	Variable (50–100,000 monomers)	Straight-chain polymer, a-L-guluronic acid, b-D-mannuronic acid	Filaments, granules, powder	Decrease by: increased MG blocks, decreased mol wt High: (greater guar, glucomannan) ionic gelation (calcium cross-linking) Moderate: acid gelation	Cell walls of brown algae	Part of diet in east Asia, antacid
CM3[14]	—	Not reported (low)	Not reported, complex of 10,000 monomers	Highly cross-linked cellulose	Cellulose comprimés in capsule	Low	Cotton wool and bark	Tested in obesity

FDA, Food and Drug Administration; HM, high methoxylated; LM, low methoxylated (percentage denotes degree of esterification); MG, mannuronic-guluronic acid complex.

Table 2. Summary of the Effects of Dietary Fiber on Gastric Emptying, Satiety, Glucose Homeostasis, Intestinal Hormones, and Body Weight Regulation

Type of dietary fiber	Gastric emptying	Satiety	Glucose homeostasis	Intestinal hormones	Body weight-energy regulation
Guar gum	Delayed in most studies; possible threshold at 5 g	Enhanced in most studies; effect is viscosity dependent, abolished by partial hydrolysis of guar, and modulated by meal fat content	Decreased postprandial glucose levels in most studies Gastric emptying delay: main factor Delayed absorption contributes	Decrease in GIP, increase in GLP-1, increase in CCK postprandially[31]	WMD, −0.04 kg; CI, −2.2 to 2.1 Gastrointestinal adverse effects limit guar use for weight loss[28]
Psyllium	Minor effect	Enhanced in most studies; threshold in the range of 5.2–8.5 g	Variable	↔ GLP-1[32]	Body mass index reduction of −2.0 ± 0.3 kg/m^2 at 6 months[33] No effect[34]
Pectin	Delayed with $>$10 g	Enhanced possibly through direct gastric effect	Decreased postprandial glucose level when $>$10 g Possible dose-response relationship	↔ CCK, PP[35] ↔ CCK, GIP[36]	No effect when supplemented to ad libitum diet[37] Reduced energy intake (alginate-pectin combination)[38]
Alginate (limited literature)	Unaffected in healthy normal weight[39] Delayed in stable diabetic patients[40]	Enhanced only by strong-gelling form Independent of gastric emptying	Decrease in correlation to gastric emptying effect[40]	Not reported	Strong-gelling form: 135-kcal (7%) reduction in mean daily energy intake over 7 weeks[41] Reduced energy intake (alginate-pectin combination)[38]
Glucomannan	No effect[42]	Enhanced satiety, combination with psyllium[43]	No effect[42]	↔ GIP[42]	WMD, −0.79; CI, −1.53 to −0.05[44] Weight loss 2.5 kg greater than with placebo at 8 weeks[45] 3.8 ± 0.9 kg weight loss more than with hypocaloric diet alone over 5 weeks in healthy overweight subjects[46]
CM3	No effect[14]	No effect[14]	Not reported	Not reported	3–4 kg weight loss greater than with placebo[47]
Cellulose	Minor effects (unmodified) Delayed (water soluble)	Enhanced (EHEC)[48]	"Second meal effect" in combination with amylopectin/amylose[49]	↔ PP, CCK (EHEC)[48]	No effect (methylcellulose) on ad libitum diet[37]
Wheat fiber	Unaltered in most studies; delayed by undiluted[50] and coarse[51] bran	Enhanced in most studies; inverse correlation with degree of refinement	Variable effects	Increase in GIP, ↔ GLP-1[52]	Modest reductions Interpretation of results difficult because wheat grain was coadministered with other dietary fiber sources in most studies[53–55]

NOTE. The literature is limited for glucomannan, CM3, and cellulose.
WMD, weighted mean difference relative to placebo in meta-analysis; CI, 95% confidence interval; CCK, cholecystokinin; GIP, glucose stimulated insulinotropic peptide; PP, pancreatic polypeptide; EHEC, ethyl hydroxyethyl cellulose ("liquid fiber").

Table 3. Effects of Long-term Fiber Supplementation on End Points of the Metabolic Syndrome and Cardiovascular Risk Factors

Author	Pereira et al[56]	Jenkins et al[57]	Jenkins et al[30]	Esposito et al[58]	Anderson et al[59]	Azadbakht et al[60]
Study design	Randomized, crossover, nonblinded study with two 6-week periods of whole grain or refined grain in 11 OV-OB hyperinsulinemic adults	Parallel RCT in patients with type 2 diabetes, low glycemic index vs high cereal fiber diets, 24 weeks	Randomized crossover study in 23 adult patients with type 2 diabetes mellitus with two 3-month periods of either 19 g or 4 g/day of additional cereal fiber in bread and breakfast cereals	Randomized, single-blind, parallel study in 120 OB women, 3 years, high-fiber (25 g/day) vs low-fiber (16 g/day) diets	Parallel RCT in type 2 diabetic men with hypercholesterolemia, 8 weeks of diet plus (5.1 g psyllium vs cellulose placebo)	6-month RCT with 2 intervention diets (500-kcal restriction [3 servings of whole grain per day], 500-kcal restricted DASH diet [4 servings of whole grain per day]), and one "eat as usual" control
Fasting blood glucose	Insignificant difference	Decrease of 6.8 mg/dL in low glycemic index group compared with high cereal fiber group, $P = .02$	Mean absolute difference of −0.4 in high vs low cereal fiber group, $P = .154$, no significant intragroup change between week 0 and weeks 8–12	7 mg/dL greater difference from baseline at 2 years (intervention minus control group), $P < .001$	−6.1% greater difference from baseline at 2 years (psyllium minus cellulose groups), insignificant	DASH: decrease of −15 and −8 mg/dL (men and women, respectively), $P < .00$
Mean all-day blood glucose					Decrease of 11% in psyllium vs cellulose, $P < .05$	
Fasting insulin	Decrease of 10% in whole grain periods vs refined grain periods	Decrease in high-fiber vs low-fiber groups (−3 μU/mL, $P = .009$)		−3 μU/mL greater difference from baseline at 2 years (intervention minus control group), $P = .009$		
Insulin sensitivity	Decrease in insulin resistance (HOMA) with whole grain(5.4 ± 0.18 vs 6.2 ± 0.18 U, $P < .01$)			Decrease in insulin resistance (HOMA): −0.9, $P = .008$		
Hemoglobin A_{1c}		Relative change of −0.33% in low glycemic index group compared with high cereal fiber group, $P < .001$	Relative absolute change of −0.3% in high vs low cereal fiber group, $P = .263$		−6.3% change from baseline for psyllium, no significant change difference from cellulose group	
Low-density lipoprotein cholesterol		No significant change in high-fiber group compared with low glycemic index group, $P = .14$	Relative absolute difference of 0.01 mg/dL in high vs low cereal fiber group, $P = .798$	−4 mg/dL greater difference from baseline at 2 years for total cholesterol (intervention minus control group), $P = .13$	−4.9% greater change from baseline, no significant change difference from cellulose group	
High-density lipoprotein cholesterol		1.7 mg/dL increase at week 24 compared with baseline (high cereal fiber) vs −0.9 mg/dL decrease (low glycemic index diet), $P = .005$	Relative absolute difference of 0.05 mg/dL in high vs low cereal fiber group, $P = .280$	+4 mg/dL greater increase from baseline at 2 years (intervention minus control group), $P = .02$	−0.9 mmol/L greater change from baseline (psyllium minus cellulose groups), $P < .05$	DASH: increase of 7 and 10 mg/dL (men and women, respectively), $P < .001$
Triglycerides		Insignificant	Relative absolute difference of 0.1 mg/dL in high vs low cereal fiber group, $P = .098$	−12 mg/mL greater difference from baseline at 2 years (intervention minus control group), $P = .04$	−7 mg/mL change from baseline for psyllium, no significant change difference from cellulose group	DASH: Decrease of −18 and −14 (men and women, respectively), $P < .001$ Weight reducing diet: decrease of −13 and −10, $P < .05$
Systolic arterial pressure		Insignificant	Relative absolute difference of −2 mm Hg in high vs low cereal fiber group, $P = .388$	−2 mm Hg greater difference from baseline at 2 years (intervention minus control group), $P = .009$		DASH: Decrease of −12 and −11 mm Hg (men and women, respectively), $P < .001$ Weight-reducing diet: decrease of −6 and −6 mm Hg, $P < .005$
Diastolic arterial pressure		Insignificant	Relative absolute difference of −1 mm Hg in high vs low cereal fiber group, $P = .505$	−1.7 mm Hg greater difference from baseline at 2 years (intervention minus control group), $P < .001$		DASH: decrease of −6 and −7 mm Hg (men and women, respectively), $P < .001$
Waist circumference				−0.06 greater difference in waist/hip ratio from baseline at 2 years (intervention minus control group), $P = .008$		Decrease of 5–7 cm with both interventions vs control, $P < .04$
Body weight/body mass index		−0.9 kg difference in weight reduction (low glycemic index minus high cereal group), $P = .053$	Insignificant difference	−11 kg/−4.2 kg/m^2, both $P < .001$	Psyllium: −0.3 kg, cellulose: +1.5 kg, $P < .05$	DASH: decrease of −16 and −13 kg (men and women, respectively), $P < .001$ Weight reducing diet: decrease of −13 and −12 kg, $P < .05$

RCT, randomized, double-blind, controlled trial; HOMA, Homeostasis Model Assessment.

2. Modification of gastrointestinal myoelectrical activity[87] and delayed small bowel transit[66,88]
3. Reduced glucose diffusion through the unstirred water layer[89]
4. Reduced accessibility of α-amylase to its substrates due to increased viscosity of gut contents.[90,91]

The determining factor in the glycemic effect is the increased viscosity and gel-forming properties of soluble fiber, because the hypoglycemic effect may be reversed by hydrolysis of guar[66] or after ultra-high heating and homogenization.[84]

Additionally, intestinal absorption of carbohydrates may be prolonged by soluble dietary fiber, in part by altering incretin levels[91] (eg, increasing GLP-1 levels).

In experimental clamp studies, soluble dietary fiber also influences peripheral glucose uptake mechanisms,[92] including increased skeletal muscle expression of the insulin-responsive glucose transporter type 4, which enhanced skeletal muscle uptake, augmenting insulin sensitivity and normalizing blood glucose levels.[93] In humans, several fatty acids stimulate expression of peroxisome proliferator-activated receptor γ, which increases levels of adipocyte glucose transporter type 4.[94]

Effects of insoluble fiber. The main effect of insoluble fiber on the risk of diabetes or glycemia involves enhancement of insulin sensitivity.[56] The exact underlying mechanism is still unclear. Alterations in gut microbiota have been implicated, in view of observed microbiota differences between obese and lean subjects, reduced gram-negative bacterial content with diets high in dietary fiber as opposed to high-fat diets,[95] and experimental data showing insulin resistance develops after daily subcutaneous injections of gram-negative bacterial lipopolysaccharides.[96] A trial of whole grain in healthy women showed accelerated GIP and insulin response and improved postprandial glycemia during the following day.[52]

Effects of grains and grain products. Grains rich in soluble β-glucans (oats, rye, barley) improve glucose tolerance more than wheat. Additional factors may also favor the hypoglycemic effects of grains:[97] greater fiber particle size, lower level of processing and refinement, which results in a slower gastric emptying rate,[51] and a high ratio of amylose/amylopectin. The effects on glycemia are also influenced by the amount of ingested grain and individual factors (age, higher body mass index, and more intolerance to glucose).

Fiber and Dyslipidemia, Hypertension, and Cardiovascular Risk

Effects of Fiber on Dyslipidemia

Soluble fibers. Recent clinical trials[98–100] and meta-analyses[101,102] support the cholesterol-lowering properties of soluble dietary fiber (pectin, guar gum, psyllium, and oat β-glucan). Low-density lipoprotein reductions of 6%–15% but no alterations in high-density lipoprotein or triglyceride levels have been consistently reported. Only a single study in patients with type 2 diabetes mellitus reported a 10% decrease in serum triglyceride levels after 6 weeks of a high-fiber diet particularly rich in soluble fiber.[103] Animal studies have elucidated that the main mechanistic effects of soluble fiber are related to fecal loss of bile acids.[104] This results in the reduction in hepatic cholesterol pools, modification of the activity of enzymes regulating cholesterol homeostasis,[105] up-regulation of hepatic low-density lipoprotein receptors,[106] and increased plasma low-density lipoprotein removal.[107] A fiber-induced decrease of food glycemic index may also enhance the beneficiary effects on dyslipidemia.[108]

Insoluble fibers. These exhibit small cholesterol-lowering properties without inducing significant bile acid loss, and effects are mainly attributed to its satiation and satiety influences.[109]

Fiber and Hypertension

Several trials and observational studies have shown a beneficial effect of increased fiber intake (both soluble and insoluble) on the control[110,111] and possibly prevention[112] of hypertension. The antihypertensive effects of fiber were confirmed in a meta-analysis of randomized trials in hypertensive subjects.[113] The postulated mechanisms include improvement of hyperinsulinemia and insulin resistance[114] and a reduction of body weight.[115]

Fiber Consumption and Risk of CVD

Three large-scale population studies reported an inverse association of high fiber intake[24] or whole grain consumption[116,117] with risk of CVD. The first study did not examine specific effects of different dietary fiber sources; thus, its effects may be attributable in part to other biologically active compounds present in high-fiber diets (antioxidants, phytochemicals).[24] In the 2 other studies, the lower CVD risk was not fully explained by the intake of whole grain fiber and antioxidants, suggesting that other constituents of a natural fiber diet contribute to the effect. In a study of 68,782 women, only cereal fiber, among different dietary fiber sources, was associated with a reduced risk of CVD.[116] In an observational study in 11,260 men and women, lower dietary fiber and antioxidant intake was associated with a greater number of CVD cases and non-CVD deaths in both men and women.

In summary, large observational studies support an inverse association of dietary fiber intake from natural food sources and CVD risk. The association persisted after adjustment for confounders (body mass index, age, smoking, and vitamin supplementation). This effect appears mostly related to consumption of cereal and whole grain.

Fiber and Gastric Sensorimotor Functions Related to the Metabolic Syndrome

Given that influences of dietary fiber on metabolic and cardiovascular outcomes are in part related to gastrointestinal functions, it is relevant to review the known effects of dietary fiber on gastrointestinal functions and mechanisms of satiation, which are summarized in Table 2.

Conclusions

There are several studies showing that the general population and diabetic patients in the United States do not meet adequate mean daily fiber intake in their diets.[118–120] On the other hand, there are clear and multiple benefits from the dietary incorporation of fiber supplements and natural foods and grains on the metabolic syndrome and CVD risk and, possibly, their prevention. The gastrointestinal tract is a crucial intermediary in these benefits through fiber modulation of gastric and small bowel motility, intestinal absorption, hormonal milieu, colonic microbiota, and fermentation. These interrelated influences also trigger diverse hepatopancreatic and peripheral alterations (such as glucose utilization and uptake), which further benefit the metabolic syndrome. Ongoing research on the gastrointestinal and metabolic effects of dietary fiber will provide valuable insight into the undefined mechanisms and may lead to new strategies to derive the greatest benefit from rational use of dietary fiber. We believe that future guidelines from influential professional organizations (such as in the fields of diabetes, obesity, cardiology, and gastroenterology) may help incorporate the results of research in grain products, recommend the best dietary sources, and refine methods and doses to benefit diabetic patients, patients with impaired glucose tolerance, and the public. It is also conceivable that combination supplemental formulas of different forms of dietary fiber could optimize viscosity, dose, preparation method, and palatability profiles to maximize patient compliance and metabolic benefits.

Although health effects of fiber have been postulated for centuries, they have been systematically investigated for only 30 years. The integration of current knowledge regarding dietary fiber in the context of the metabolic syndrome suggests dietary fiber still plays a pivotal role in the metabolic syndrome and its consequences.

Supplementary Data

Note: The first 50 references associated with this article are available below in print. The remaining references accompanying this article are available online only with the electronic version of the article. Visit the online version of *Gastroenterology* at www.gastrojournal.org, and at doi:10.1053/j.gastro.2009.11.045.

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Conflicts of interest

The authors disclose no conflicts.

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