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The health benefits of dietary fiber: beyond the usual suspects of type 2 diabetes, cardiovascular disease and colon cancer

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Abstract

Dietary fiber (DF) is deemed to be a key component in healthy eating. DF is not a static collection of undigestible plant materials that pass untouched or unencumbered through the gastrointestinal (GI) tract; instead, DFs are a vast array of complex saccharide-based molecules that can bind potential nutrients and nutrient precursors to prevent their absorption. Some DFs are fermentable, and the GI tract catabolism leads to the generation of various bioactive materials, such as short-chain fatty acids (SCFAs), that can markedly augment the GI tract biomass and change the composition of the GI tract flora. The health benefits of DFs include the prevention and mitigation of type 2 diabetes, cardiovascular disease and colon cancer. By modulating food ingestion, digestion, absorption and metabolism, DFs reduce the risk of hyperlipidemia, hypercholesterolemia and hyperglycemia. Emerging research has begun to investigate the role of DFs in immunomodulation. If substantiated, DFs could facilitate many biologic processes, including infection prevention and the improvement of mood and memory. This review describes the accepted physiologic functions of DFs and explores their new potential immune-based actions.

Introduction

Dietary fiber (DF) decreases the risk for type 2 diabetes (T2D), cardiovascular disease, and colon cancer (1–4) by reducing the digestion and absorption of macronutrients and decreasing the contact time of carcinogens within the intestinal lumen (1, 2, 5, 6). In addition, the United States Food and Drug Administration has approved health claims supporting the role of DF in the prevention of cancer and coronary heart disease (CHD) (7, 8). More recently, and perhaps more interestingly, epidemiological studies have found the

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benefits of DF to extend beyond T2D, CHD, and colon cancer. A 9-year follow-up study of 567,169 American Association of Retired Persons (AARP) members aged 50–71 years old found that in addition to reducing the risk of death, DF intake was inversely related to respiratory and infectious disease mortality (9). In a cross-sectional study of 1,538 pregnant women, a higher DF intake was associated with a reduced risk of preeclampsia (10). DF may also increase positive mood, cognition, and alertness (11, 12). The mechanisms by which DF exerts its protective effects remain to be elucidated, but its ability to physically disrupt macro- and micronutrient absorption has been extensively explored. More recently, the positive impact of DF on human health by altering the intestinal flora has surfaced (13, 14). DF-dependent changes in the bacterial composition of the gut are thought to modify the host's metabolism in a variety of ways, including enhanced bile acid deconjugation (15, 16), production of short chain fatty acids (SCFAs) (17), and modulation of inflammatory bioactive substances. Here we define DF and provide an overview of the impact of DF on metabolism and the gut flora. We also discuss the impact of DF on conditions and diseases not directly linked to diabesity, CHD, and colon cancer.

Defining dietary fiber

DF is a broad category of non-digestible food ingredients that includes non-starch polysaccharides, oligosaccharides, lignin, and analogous polysaccharides with an associated health benefit (3, 6). The physical properties of DF vary, and even a slight variance may influence the physiological effect of the DF. Generally, DF is classified by solubility in water, microbial fermentation in the large intestine, and viscosity. Soluble DFs include pectin, gums, and polysaccharides, whereas insoluble DFs include cellulose, hemicellulose and lignin (3). Previously, it was erroneously believed that soluble DF decreased serum lipids and cholesterol, while insoluble DF solely contributed to fecal bulking. However, the physiologic effects of a particular DF are attributed to its degree of viscosity and fermentation (18). In addition, the definition of DF has expanded to now include oligosaccharides that have properties similar to soluble DFs and resistant starches that escape enzymatic digestion in the small intestine and act as DF in the large intestine (19). Prebiotic DF is not classified in terms of solubility or viscosity but is defined by resistance to digestion and absorption in the small intestine, partial or complete fermentation by microbiota in the large intestine, and the ability to stimulate growth of select bacteria (17). Only two such DFs fit all of the above three criteria and are considered prebiotics: inulin and trans-galactooligosaccharides. In the near future, polydextrose and several other oligosaccharides may be classified as prebiotics due to their ability to meet the above criteria (20).

The human gut flora is dominated by *Bacteroides* and *Firmicutes* (21). Fermentable DF shifts the gut microbial populations by providing substrates for bacterial fermentation. Fructooligiosaccharides and galactooligosaccharides increase the fecal populations of *Bifidobacteria* and *Lactobacillus* in infants (22, 23), whereas inulin increases the *Bifidobacteria* populations in adults (24). DF can be partially or completely fermented to SCFA acetate, propionate, and butyrate (25) (Table 1). The fermentation patterns and the ratio of SCFAs produced depend on DF type. Acetate is the main product of pectin fermentation (26), whereas fermentation of gum arabic and cylcodextrins results primarily in propionate (27, 28). Okara increased cecal butyrate and decreased cecal acetate concentrations in female rats fed 10% okara for 4 weeks, compared to standard rodent chow (29). Acetate and propionate enter the liver via portal circulation and are almost fully metabolized (30); however, butyrate is metabolized by β-oxidation in the colonic enterocytes (31). Finally, the physiologic effects of DFs are partly due to the type and amount of SCFAs produced and the ability of these SCFAs to stimulate intestinal water and sodium absorption as well as influence gut pH and bile salt precipitation (32).

Diabesity

Given the link between an elevated body mass index (BMI), T2D, and CHD (2, 19), the role of DF in weight reduction has been examined in animal and human studies. A strong inverse relationship between DF intake and weight has been established in animal models (28, 29, 33–36) and human epidemiological studies (37–40). A prospective cohort study of 74,091 females over 12 years found women in the highest quintile for whole grain consumption had a 49% lower risk of major weight gain than women in the lowest quintile. Interestingly, DF most significantly prevented weight gain in individuals who were overweight at baseline (39). A prospective cohort study of 894,332 European individuals followed for 6.5 years found that total DF intake was associated with reduced weight and waist circumference (37). In this study, cereal DF was associated with weight, while DF from fruit and vegetables was not. Unfortunately, there is inconsistent evidence regarding the association of increased DF intake to weight loss in human clinical trials (3). These conflicting results in humans may be due to the DF type, dosage, and study population. A meta-analysis of 14 randomized, controlled trials that examined glucomannan and weight loss found that glucomannan supplementation significantly reduced weight (41). In a randomized, placebo-controlled study of 167 overweight individuals, supplementation with 1,240 mg/day of glucomannan with a calorie-restricted diet resulted in a greater weight loss after 5 weeks than a calorie-restricted diet alone (42). In a parallel, double-blind, placebo-controlled clinical trial, weight loss was greater, but not significantly, in obese individuals receiving 3 grams of *Plantago ovarto* (psyllium) + 1 gram of glucomannan 2 or 3 times per day with a calorie-restricted diet versus placebo for 16 weeks (43). While glucomannan seems to show promise for weight reduction, not all clinical trials have demonstrated a weight loss effect. Supplementation with 10 grams of guar gum (glucomannan) 3 times per day for 6 weeks did not result in a weight difference when compared to the control period in a double-blind, placebo-controlled, crossover trial of 25 non-obese men. Overall, diet supplementation with DF appears to be more effective for weight reduction in overweight or obese populations and when used in combination with a calorie-restricted diet. Clinical interventions with other DFs have also produced inconsistent results regarding weight (3). For example, chitosan, a popular DF in over-the-counter weight loss treatments, caused weight loss in a randomized, double-blind, placebo-controlled clinical trial of 230 overweight or obese persons receiving 3 grams of chitosan or a placebo daily for 24 weeks (44). Although weight loss was significantly greater in the DF and placebo groups (0.4% loss in the chitosan group vs. 0.2% gain in the placebo group), it was not clinically relevant in this study. Overall, a diet high in a variety of soluble and insoluble DFs from whole grains, cereals, fruits, and vegetables may be more effective in weight regulation than supplementation with a single DF.

A reduced risk for T2D appears to depend on the type and dose of DF and the study population. In animals, soluble DFs decrease T2D related biomarkers. In mice, 10% psyllium and 10% sugar cane fiber decreased the fasting blood glucose and fasting plasma insulin when added to a high fat diet for 12 weeks, compared to the insoluble fiber cellulose (45). β-glucan also improved the glucose tolerance and decreased the serum insulin in mice when added to a high fat diet at a 2 and 4% level (34). In contrast, diabetic dogs fed an insoluble DF had lower maximum and mean blood glucose concentrations and a lower area under the blood glucose curve than dogs fed a soluble DF or a low DF diet (46). In humans, muffins high in β-glucan and resistant starch lowered the postprandial blood glucose and insulin levels more effectively than muffins containing low or medium β-glucan/resistant starch (5). *Plantago ovarta* husk supplementation of 14 grams per day for 8 weeks, when compared to placebo, reduced the serum insulin but not plasma glucose levels (47). Surprisingly, despite the ability of soluble DF to reduce diabetes-associated biomarkers, soluble DF has not been associated with a reduced risk of T2D in population-based studies (48). In a large prospective cohort study of 99,826 women, the intake of total DF, total

insoluble DF, and DF from cereal was inversely related to the risk of diabetes. There was no association found between the soluble DF intake and diabetes risk (48). This finding also highlights the difference in effectiveness of DFs as part of a balanced diet and DFs as standalone supplements.

The protective effect of DF on obesity and T2D has been historically attributed to greater satiety due to an increased mastication, calorie displacement, and decreased absorption of macronutrients (49). This mechanism is believed to be due to the ability of soluble DFs to form viscous solutions that prolong gastric emptying, consequently inhibiting the transport of glucose, triglycerides and cholesterol across the intestine (6, 46, 50–52). *In vitro*, guar gum was shown to reduce glucose absorption by reducing the impact of stimulated intestinal contractions (53). Viscous DFs form gels that decrease the luminal content contact with the pancreatic lipases and bile by forming DF-lipid aggregates that block fat emulsification and micelle formation (51). An increased viscosity of the luminal contents also thickens the unstirred water layer, thereby decreasing the diffusion and uptake of cholesterol and glucose from the intestinal lumen (54). Certain DFs can bind bile acids and micelle components, such as monoglycerides, free fatty acids, and cholesterol, which decrease the absorption and increase the fecal excretion of these entities (55–57).

DF also modifies lipid (Table 2) and carbohydrate (Table 3) metabolism by influencing the expression of key genes and hormones. Acetyl-CoA carboxylase is the rate-limiting enzyme in lipogenesis and is regulated by AMP-activated protein kinase (AMPK). In a 10-week study comparing obese and lean rats, adding 5 grams of *Plantago ovate* to a rat chow increased the phosphorylation of AMPK, consequently inhibiting acetyl-CoA carboxylase. This inhibition of acetyl-CoA carboxylase in obese rats was comparable to the acetyl-CoA carboxylase activity in lean rats fed a control diet (58). Fructooligosaccharide (10 grams/100 grams) also has been shown to decrease the hepatic acetyl-CoA carboxylase expression in rats (59). Increases in SCFAs due to bacterial fermentation may activate hepatic AMPK (60). The fatty acid synthase complex catalyzes the synthesis of fatty acids, primarily palmitate. Reductions in the fatty acid synthase expression have been demonstrated in rodents fed resistant starch, fructans, inulin, β-glucan, *Plantago ovarto* and hydroxylpropylmethycellulose (34, 35, 58, 59). The alteration in the gene expression may be due to a DF-induced modulation of the gut flora and subsequent SCFA production. The conventionalization of germ-free mice resulted in increased hepatic triglycerides, with a corresponding increase in the mRNA expression of hepatic acetyl CoA carboxylase-1 and fatty acid synthase (61). Butyrate activates colonic fatty acid synthesis by contributing carbon atoms for *de novo* lipogenesis (62, 63). Finally, a supplementation of 1.0% gum arabic in the drinking water of female mice over 180 days resulted in an increased expression of triacylglycerol lipase and hormone-sensitive lipase in the paranephric fat; these enzymes are involved in mobilizing fatty acids from adipocytes (28, 64). Gum Arabic also increased the expression of fasting-induced adipose factor, a lipoprotein lipase inhibitor that blocks the incorporation of fatty acids into low-density lipoproteins (28). This increase in fasting-induced adipose factor may be due to the intestinal flora modulation as fasting-induced adipose factor is synthesized in the intestinal epithelium in response to microbiota (61).

The rate-limiting enzyme of cholesterol synthesis, HMG-CoA reductase, converts HMG-CoA to mevalonate. Although β-glucan supplementation in mice did not alter the hepatic expression of HMG-CoA reductase, hydroxylpropylmethycellulose was shown to increase the hepatic expression in hamsters (34, 35). However, a combination of inulin and oligofructose increased the hepatic expression of HMG-CoA reductase in rats (65). Increased HMG-CoA reductase may be due to a depletion of cholesterol pools resulting from an increased excretion of bile cholesterol. Catabolism by microbial populations may

also be important. Bacteria such as *Lactobacillus* and *Bifidobacteria* can exert a hypocholesterolemic effect by enhancing bile acid deconjugation (15, 16). Furthermore, *Lactobacillus* and *Bifidobacteria* remove cholesterol *in vitro* by assimilation and precipitation (25, 66). Fermentation products further affect lipid metabolism. Propionate inhibits the incorporation of acetic acid into fats and sterols, resulting in decreased fatty acid and cholesterol synthesis. Demigne *et al.* demonstrated in rats that propionate inhibited fatty acid and cholesterol synthesis by blocking the integration of C1,4 into fatty acids (62).

Carbohydrate metabolism is influenced by DF intake. Insoluble DF appears to improve insulin sensitivity, but exact mechanisms are unclear (6, 67). Both soluble and insoluble DFs may be involved in the regulation of hormones, such as glucose-dependent insulin tropic polypeptide and glucagon-like peptide-1, that stimulate postprandial insulin release, enhance glucose tolerance, and delay gastric emptying (2, 68). In a study of 14 women, control meals of insoluble DF increased insulin responsiveness and glucose-dependent insulin tropic peptide (67). Proglucagon, a precursor of glucagon-like peptide 1, is produced in the L-cells of the distal ileum and colon and is increased by butyrate production (69). A diet high in fermentable DF, as opposed to cellulose, increased the expression of proglucagon mRNA in the ileum of dogs and rats and in the colon of dogs (36, 69). An increase in postprandial plasma glucagon-like peptide-1 was also observed (6, 69). DFs may increase proglucagon and glucagon-like peptide-1 by augmenting the number of L-cells in the intestine (13).

Shifts in the gut flora affect the manifestation of diabesity. As noted above, the human flora is comprised primarily of *Bifidobacteia* and *Firmacutes* (21). Obesity in both mice and humans is linked to an increase in *Firmacutes* and a decrease in *Bifidobacteria* (21). This microbial misbalance is corrected by weight loss in humans. The obesity-associated microbiom has a heightened capacity for energy harvest (70), which may attenuate weight gain. Certain DFs, such as inulin and oligofructose, increase *Bifidobacteria* populations and may prevent a shift towards an obesity-associated microbiom(71, 72). As evidence, antibiotic-induced changes in the gut flora of obese mice resulte in improved glucose tolerance (73), indicating that the obese state microbiom may also influence diabetes-related parameters.

Inflammation

Chronic inflammation is inherent to a variety of diseases, including diabesity. DF appears to be anti-inflammatory; decreasing inflammation-associated biomarkers and bioactives, including C-reactive protein (CRP), IL-6, and TNF-α (58, 74, 75). In a case-control study of 88 individuals that examined the relationship between DF intake and plasma cytokine and chemokine concentrations, no association was found between the intake of total, soluble, or insoluble DF to any cytokine or chemokine examined. However, an intake of cereal DF was associated with a reduced inflammatory bioactive array comprised of IL-1β, IL-6, TNF-α, IL-4, IL-5, and IL-13 (76). This immune profile suggests anti-inflammation with the macrophage deactivation (IL-10) phenotype. Conversely, in a cross-sectional study of 1,953 overweight post-menopausal women, the total, soluble, and insoluble DF intakes was inversely associated with plasma IL-6 and TNF-α concentration (75). Several epidemiologic studies have also demonstrated an inverse relationship between DF intake and CRP, a sensitive marker of inflammation and CHD (74, 77, 78). In an examination of NHANES data containing 14,533 adults, the total, insoluble, and soluble DF intakes was linked to lower serum CRP concentrations. In addition, a subgroup of persons with chronic kidney disease who reported a higher DF intake had decreased all-cause mortality (78). Most epidemiologic studies demonstrate an association between DF intake and anti-inflammation, but the cohort and cross-sectional study designs make it difficult to ascertain if DF supplementation would successfully reduce inflammation. To this end, intervention trials have reported inconsistent results that, as mentioned earlier, may reflect the fiber type, dose,

and study population. In a prospective randomized controlled trial of 158 overweight or obese persons receiving 7 or 14 grams per day of psyllium, there was no difference in the serum CRP or IL-6 concentrations, compared to the control group (79). Notably, in a pre-test/post-test study involving 19 elderly nursing home patients, 4 grams of fructooligosaccharides delivered 2 times per day for 3 weeks caused a decrease in granulocyte and monocyte phagocytic activity and IL-6 blood levels. This study additionally demonstrated increased fecal *Bifidobacteria* (72). However, the supplementation of fructooligosaccharides in infant formula appears to afford long-lasting protection against allergens and infection. Galactooligosaccharides and fructooligosaccharides were added to the formula of 134 infants for 6 months, which resulted in a lower incidence of atopic dermatitis, wheezing, upper respiratory tract infections, and fever in the 2 years following the intervention (80). A study of 427 college students given 2.5 or 5 grams of galactooligosaccharide daily supports that galactooligosaccharides prevent infection. Over the 8-week study period, when compared to control subjects, stress-induced gastrointestinal symptoms and the percentage of days of cold and flu were decreased (81). Given that fructooligosaccharides and galactooligosaccharides increase the fecal *Bifidobacteria* and *Lactobacilli* (22, 23, 72), the immunomodulation seen in the above human studies may be the result of DF-induced changes in gut flora populations.

With fermentable DF, gut-generated SCFAs may be responsible for the modulation of appetite, insulin signaling, and inflammation. In adipose tissue, propionate down-regulated the pro-inflammatory cytokine TNF-α (82). *In vitro*, SCFAs suppressed the release of TNF-α from LPS-stimulated human neutrophils while also reducing IL-6 mRNA and proteins in the cultures of human colonocytes (83). SCFAs appear to communicate with the immune system through G-protein-coupled free fatty acid receptors. Free fatty acid receptor 3 (FFA3) is expressed primarily by adipocytes and activated by propionate, butyrate, and acetate. The activation of adipocyte FFA3 by propionate leads to elevated leptin secretion, whereas activation by butyrate initiates adipogenesis (84). FFA2 is expressed in leukocytes and colonic L-cells in addition to adipocytes. FFA2 is activated primarily by propionate (84) and, to a lesser extent, acetate (85). Like FFA3, FFA2 may regulate the differentiation of adipocytes; however, it also appears to lessen the fat cell triglyceride storage capacity and accumulation of body fat deposits (86). How reducing body fat relates to the ability of FFA2 to decrease the plasma free fatty acids (84) and increase the adipocyte insulin sensitivity is unclear (86). Interestingly, DFs may increase the cellular expression of FFA2 as colonic FFA2 was increased by 31% in rats fed 5% nopal (a DF containing a 40:60 ratio of soluble to insoluble fiber) (87). Bypassing FFA2, DF may interact directly with immunoregulatory cells. Mucosal macrophages and dendritic cells have pattern recognition receptors with carbohydrate-binding domains that bind certain DFs, such as β-glucans, and cause a decrease in IL-12 and increase in IL-10 (88), which is consistent with an anti-inflammatory phenotype. Overall, the mechanism by which fermentable DF works may not be principally related to their ability to act as a SCFA source.

Cognition and Memory

Inflammation is an important component of many neurodegenerative diseases. Alzheimer's disease and vascular dementia are both associated with an increased CRP, IL-6, and TNF-α (89). CRP is neurotoxic, and neuron synthesized CRPcan initiate neuro-apoptosis (89). The progression of Alzheimer's disease has been linked to both oxidative stress and AB-induced neurotoxicity. An extract of *Triticum aestivium* L., a type of wheat, has been shown to protect cells from AB cytotoxicity and apoptosis *in vitro*. Additionally, *Triticum aestivium* L. reversed scopolamine-induced spatial memory deficits in rats (90). While research in this area is limited, DF intake may affect cognition and mood due to its role in regulating systemic inflammation, depression, and anxiety (89). A 1.5-g fiber breakfast bar increased

positive mood and performance in a free recall task (12). DF has also been shown to ameliorate pre-depression-associated sickness behaviors in mice because diets containing 10% pectin reduced endotoxin-dependent social withdrawal and fever, compared to diets containing 10% cellulose (91). Furthermore, in a community-based study of 394 post-menopausal women, lignin improved cognitive performance (11). Mechanistically, the microbiom-dependent production of SCFAs is likely key to DF-induced memory and behavioral changes because administering SCFAs illicit alterations in brain functions. Butyrate, possibly via histone hyperacetylation, increased brain-derived neurotrophic factor, a bioactive substance that is critical for neuronal plasticity (92). Administering propionate to rats induced autism-like behavioral changes (92), and diet-induced alterations in the gut flora generated detrimental effects on mouse cognition and behavior (93). Finally, dysregulation within the hypothalamic-pituitary-adrenal (HPA) axis results in depression and anxiety (92), and commensal bacteria have been shown to influence the HPA axis and reduce behavioral abnormalities (92).

Conclusion

The definition and chemical characterizations of DF is well documented and understood, as is the ability of DF to reduce the absorption of macronutrients by increasing the viscosity of the luminal contents and altering the intestinal transit time. The impact of DFs on lipid metabolism is clearly delineated, especially in enhancing the expression of key enzymes of β-oxidation and *de novo* lipogenesis, namely triacylglycerol lipase, hormone-sensitive lipase, acetyl-CoA carboxylase, and fatty acid synthetase. DF is hypocholesterolemic and modulates the expression of HMG-CoA reductase, which decreases cholesterol synthesis and increases the excretion of cholesterol in the bile. The processes by which DF improves glycemic control and insulin sensitivity are less clear. Soluble DF may lower blood glucose by slowing carbohydrate absorption in the gut via increased viscosity. However, epidemiological studies have failed to find a solid relationship between soluble DF intake and the reduced risk of T2D. Finally, emerging research suggests that DF may influence immunity by altering plasma concentrations of key bioactive substances, such as CRP and TNF-α, by modulating the gut flora. Immune system modifications that are likely tied to SCFA and FFA2 suggest a link to the brain-cytokine system, as evidenced by the ability of certain DFs to regulate sickness symptoms and cognitive function. While these exciting new functions for DF are intriguing, much work needs to be performed to determine the mechanisms by which DF works.

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Abbreviations

AARP	American Association of Retired Persons
AMP	adenosine monophosphate
AMPK	AMP-activated protein kinase
CHD	coronary heart disease
CRP	C-reactive protein
DF	dietary fiber
FFA	free fatty acid

HMG-CoA	3-hydroxy-3-methylglutaryl-coenzyme A
IL	interleukin
SCFA	short chain fatty acid
T2D	type 2 diabetes
TNF	tumor necrosis factor

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Table 1

Summary of the effect of dietary fiber on cecal short chain fatty acid production and lipid excretion.

Fiber	Species	Propionate	Acetate	Butyrate	Total SCFA	Fecal Lipid	Fecal Bile	References
α-cyclodextrin	rodent					+		13
β-cyclodextrin	rodent	+	+	−	+			49
Cassia tora Linn.	rodent					+	+	53
Hydroxlpropyl methylcellulose	rodent						+	16
Gum arabic	rodent	+	−	−	−			29
Okara	rodent	+	+		+−	+		15
Pectin	rodent	+	+	+	+			41,67
Passion Fruit	rodent					+	+	36
Resistant Starch	rodent					+		38

“+”indicates dietary fiber affect this parameter positively or negatively.

“−” indicates dietary fiber did not have an effect on this parameter.

Table 2

Summary of the effects of fiber on biomarkers related to dyslipidemia and hypercholesterolemia

Fiber	Species	Serum Lipids	Hepatic Lipids	Free Fatty Acids	Plasma TG	Hepatic TG	Total Plasma Cholesterol	Hepatic Cholesterol	References
α-cyclodextrin	rodent		−		+/−		−		13
β-cyclodextrin	rodent	+			+		+		49
β-glucan (Barley)	rodent			+/−	+/−		+/−		14,40
	human				−		+		6
Blueberry peel extract	rodent	−	−		−	−	+	+	59
Cassia tora Linn.	rodent			+	+		+		53
Hydroxlpropyl methylcellulose	rodent	+	+		+	+/−	+	+	16,92
	human						+		9
Gum arabic	rodent			+	−		+		29
Glucomannan (guar gum)	human				+		+		23
Inulin (oligofructose)	rodent	+			+	+	+/−	+	35,88
	human			−	+		−		10
Okara	rodent		+		+/−	+/−	+/−	+/−	15,51
Passion Fruit	rodent				+		+		36
PolyGlycopleX (PGX)	rodent				+				17
Psyllium	rodent			+	+	+	+		55,87
	human				+		+		1,8,52
Resistant Starch	rodent	+			−	+	+	+	38

“+” indicates dietary fiber affect this parameter positively or negatively.

“−” indicates dietary fiber did not have an effect on this parameter.

TG= triglycerides

Table 3

Summary of the effects of fiber on weight and biomarkers related to type 2 diabetes

Fiber	Species	Weight	Plasma Glucose	GTT	Plasma Insulin	ITT	References
α-cyclodextrin	rodent	+	−		−		13
β-cyclodextrin	rodent	−					49
β-glucan or Barley	rodent	+/−	+/−	+	+/−		14
	human		−	+	−	+	6
Blueberry peel extract	rodent	−					59
Cassia tora Linn.	rodent	−					53
Hydroxlpropyl methylcellulose	rodent	+/−	+/−		−		16,92
Gum arabic	rodent	+					29
Galactomannan (guar)	human	−	+		+		23
Inulin (oligiofructose)	rodent	−	+		+		35
	human		−		−		10
Okara	rodent	−	−				15,51
Pectin	rodent	+/−					41,67
	human	−					46
Passion Fruit	rodent	−					36
PolyGlycopleX (PGX)	rodent	+	−		−		17
Psyllium	rodent	+/−	+/−		+		12, 55, 90
	human	−	+/−	−	+/−		1, 7, 52
Resistant starch	rodent	−					38

“+” indicates dietary fiber affect this parameter positively or negatively.

“–” indicates dietary fiber did not have an effect on this parameter.

GTT= glucose tolerance test ITT= insulin tolerance test