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#### Chapter

# Dietary Fiber and Dyslipidemia

I. Gusti Ayu Nyoman Danuyanti and Z.S. Ahmad Fahrurrozi

# Abstract

Fibers are abundantly found in vegetables, fruit, beans, cereals, seeds, and tubers. Beans and seeds, alongside prevailing as both of the fiber sources, are the sources of vegetable protein as well. Whereas tubers are a carbohydrate source, which people deem as a staple food. Fiber intake in diets, particularly soluble fibers, has the ability to produce gel in the intestines, inhibiting glucose and cholesterol absorption. Dietary fibers have the ability to bind bile salts in the digestive tract, and disturbed bile reabsorption will stimulate bile synthesis in the liver. Dyslipidemia has a significant role in systemic responses and inflammation in adipose tissues. Inflammation can increase intestinal permeability and adipose tissues. Dyslipidemic management is carried out by altering lifestyles, intervening in suitable diets to reduce LDL levels, and increasing HDL levels. The degree of compliance with diet interventions is seminal to ensure successful dyslipidemic management.

Keywords: Fiber, Dyslipidemia, Management

#### 1. Introduction

Dyslipidemia as one of the risk factors of metabolic syndrome is the abnormality condition of lipid profile, marked by the increased triglyceride levels (TG), total cholesterol, low density lipoprotein (LDL) cholesterol, and low level of high density lipoprotein (HDL) cholesterol. Dyslipidemia is triggered by lifestyle changes with the tendency of consuming high fat but low fiber food and sweet beverages with high level of fructose alongside with the lack of physical activity [1–4].

High fat dietary with saturated fatty acid and trans fatty acid substance initiates the rising of LDL cholesterol, reduces the level of DHL cholesterol, and arouses oxidative stress on endothelium blood vessel as the result of over production of reactive oxygen species (ROS) that will oxidize extracellular LDL, developing oxidized LDL [5, 6]. On the other hand, high fructose dietary initiates insulin resistance through the reduction of insulin receptor sensitivity [7, 8]. Insulin functions as expression control of sterol regulatory element binding protein (SREBP), roles in the regulation and biosynthetic of fatty acid and cholesterol in liver [1, 9, 10]. As a result, it can increase SREBP expression and appropriately stimulates liver lipogenesis and triglyceride synthesis enhancement in liver.

#### 2. Dietary fiber and dyslipidemia

#### 2.1 Fiber

Fibers are mostly found in food with a low glycemic index [11], so the higher the fiber level contained in the food, the lower the glycemic index. This is because fibers

bring the food bolus into a more viscous condition (gel-formed), thereby slowing down the food digestion process [12–14].

Fibers are abundantly found in vegetables, fruit, beans, cereals, seeds, and tubers. Beans and seeds, alongside prevailing as two fiber sources, are also the sources of vegetable protein, whereas tubers are a carbohydrate source, which people deem as a staple food. An example of fiber sources from tubers is sweet potatoes (*Ipomoea batatas*), which contain fibers of 3–4.2% [15, 16]. Meanwhile, fibers in sweet potato starch are 5.54% [17]. In addition to sweet oranges, yellow pumpkins belong to the vegetable group possessing beta carotenes and considerable high fibers. Previous research attested that yellow pumpkin starch contained fibers by 10–12.24%, while fresh yellow pumpkins contained fibers by 2–3% [15, 18, 19].

Fibers, by definition, are carbohydrate polymers which indigestibled in small intestines but are managed to be fermented by bacteria in the colon [11]. Following their characteristics, fibers are classified into eight, as listed in detail in **Table 1**.

Referring to the Dietary Reference Intake (DRI), fibers are classified into three [12, 21]:

- 1. Dietary fibers are carbohydrates and lignin, which in terms of intrinsic, intact in plants, and indigestible.
- 2. Functional fibers are carbohydrates, which indisgestibled but beneficial for humans.
- 3. Total fibers are a combination of dietary fibers and functional fibers.

Food products with 2.5 g of fibers/portion are considered as a good source of fibers, and those with 5 g of fibers/portion are considered an excellent one [20, 22]. Dietary fibers are not correlate with energy building, however, after experiencing fermentation in the colon, the fibers were capable of increasing the volume of feces, enhancing laxative products, softening stool consistency, and forming a short-chain fatty acid (SCFA) contributive to health [11, 23, 24].

#### 2.1.1 Dietary fiber

Dietary fibers are a part of plants, which consumabled and collated from carbohydrates and lignin in plants, resistant to the digestive and absorption in the small intestines and experiencing a partial or simultaneous fermentation in the colon (Brownlee, 2009). The sources of dietary fibers are not only vegetables and fruit but also beans, cereals, seeds, and tubers [24, 25].

The fermentation process, undergone by dietary fibers in the colon, broke down dietary fibers into SCFA, giving the physiological functions, beneficial for health [26]. Primary short-chain fatty acids created by acetate, butyrate, and propionate simultaneously contributing to the mineral absorption process, fat metabolism, and anti-inflammation [26]. The degree of fiber fermentation which creates SCFA products is presented in **Table 2**.

Some factors which affected fiber fermentation were types of substrate (the chemical structure of fibers and solubility), specific microbes (gut microbial activities and population), and transit time in the digestive tract [27, 28].

Butyric acid is needed in maintaining the balance of colon cells by increasing the growth and differentiation of cells and demonstrates higher anti-inflammation than acetate and propionate. Acetate is imperative in increasing ileal motility as well as blood flow to the colon and increasing lipopolysaccharides in relation to Tumor Necoris Factor (TNF), Interleukin-6 (IL-6), and Nuclear

# Dietary Fiber and Dyslipidemia DOI: http://dx.doi.org/10.5772/intechopen.98838

Classification	Types of fiber
1. Dietary fibers	Lignin
	Cellulose
	β-glucan
	Hemicellulose
	Pectin
	Gums
	Resistant starch
2. Functional fibers	Resistant dextrin
	Psyllium
	Fructooligosaccharides
	Polydextrose
	Isolated gums
	Isolated resistant starch
3. Soluble fibers	B-glucan
	Gums
	Wheat dextrin
	Psyllium
	Pectin
	Inulin
4. Insoluble fibers	Cellulose
	Lignin
	Several types of pectin
	Several types of hemicellulose
5. Fermentable fibers	
6. Non-fermentable fibers	Wheat dextrin
	Pectin
	β-glucan
	Guar gum
	Inulin
	Cellulose
	Lignin
7. Viscous fibers	Pectin
	β-glucan
	Guar gum
	Psyllium
8. Non-viscous fiber	Polydextrose
	Inulin

# **Table 1.**Classification by-characteristic of Fibers [20].

Sources of fibers	Fermentation (%)
Mono/oligosaccharides	
Pectin	
Bran	
Cellulosa	
Lignin	

#### Table 2.

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Degree of Fiber fermentation [27, 28].
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Factor Kappa Beta (NF $\kappa\beta$ ) by increasing periphery antibody production. Propionic fatty acid decreased food intake and increased satiety by suppressing leptin activities and activating G protein-coupled receptor-43 and -41 (GPCR-43 and GPCR-41) [22, 24, 26].

Dietary fibers, classified by the solubility properties, were soluble dietary fibers and insoluble dietary fibers [20, 25, 26]. Soluble dietary fibers contributed to forming thick solution (viscous), slowing down gastric emptying and absorption of nutrients including glucose so as to control plasma glucose levels, whereas insoluble dietary fibers functioned to overcome digestive tract disorders, increase the volume of feces, and shorten the transit time of feces in the colon [14, 20].

#### 2.1.2 Soluble dietary fiber

Inulin, resistant starch, and soluble polysaccharides are soluble dietary fibers. Inulin is a fructooligosaccharide (FOS) class of carbohydrates important to the digestive system, increase the number of intestinal L cells in the proximal part of the colon, elevate SCFA production, decline the pH of the colon so inhibit the growth of pathogenic bacteria, increase the volume of feces, and avert constipation. Inulin is customarily used in food industries as a substitute for fat and sugar, specifically in low-fat food products [14, 29, 30].

Resistant starch is kind of starch or a starch-degraded product undigested in the human small intestine [31] and could be categorized into four [32]:

- 1. Resistant starch 1 (RS1) is starch whose resistance formed after encapsulated in an indigestible matrix.
- 2. Resistant starch 2 (RS2) is starch whose granules are not gelatinized so they were slowly hydrolyzed by  $\alpha$ -amylase.
- 3. Resistant starch 3 (RS3) is starch formed when food containing a cooked and cooled starch.

4. Resistant starch 4 (RS4) is starch which has been chemically modified.

Starch is composed of amylose and amylopectin which could experience gelatinization when being cooked and is easily hydrolyzed by amylase enzymes [22, 31, 33].

Water-soluble polysaccharides (WSP) were water-soluble dietary fibers as a plant's component which could not be enzymatically degraded into a subunit which could be absorbed in the stomach and small intestine. Water-soluble polysaccharides were primarily used by food industries to achieve food quality in regard to viscosity, stability, texture, and appearance [34]. Dioscorea contained glucomannan, including in the WSP group which could develop and thicken in water [14, 34].

## 2.1.3 Insoluble dietary Fiber

Insoluble dietary fibers comprised carbohydrates which contained cellulose, hemicellulose, and non-carbohydrates with lignin [20, 33]. Insoluble dietary fibers were more frequently found in vegetables, wheat, cereals, and beans. They have several functions, e.g., increasing the volume of feces and shortening the transit time of feces in the colon. Because of these functions, they were often exerted to treat digestive tract disorders, such as constipation, diverticular diseases, and irritable bowel syndrome [24, 25, 33].

What is contained by insoluble dietary fibers tremendously determines the physiological effects of the fibers. For instance, cellulose and hemicellulose retained water in feces, enhanced peristalsis of the colon, escalated the colon performances, and reduced the colon intraluminal pressure; whereas lignin is physiologically pivotal in binding minerals, increasing the secretion of bile acids, and acting as an antioxidant [24, 35].

## 2.1.4 Functional fibers

Functional fibers were also insoluble dietary fibers which were indigestible and has beneficial effects as some other dietary fibers has different psychological effects [22, 25]. It is because rich-fiber food also contained bioactive phytochemicals which has additional benefits [33, 36]. Components of dietary fibers considerably determined psychological effects bred. Psychological functions of dietary fibers could bring on the occurrence of some diseases protective effects, e.g. [32]:

- 1. Giving glycemic controls and insulin responses to patients with diabetes mellitus.
- 2. Improving intestinal health through the functions of fibers as prebiotics and protagonist cultures in patients with colon cancer, ulcerative colitis, inflammatory bowel disease (IBD), diverticulitis, and constipation.
- 3. Fixing blood lipid profile in cardiovascular disease and dyslipidemia.
- 4. Increasing satiety and reducing energy intake in obesity.
- 5. Increasing micronutrient absorption in patients with osteoporosis.

## 2.2 Fiber and dyslipidemia

One of the indicators of dyslipidemia is an increase in LDL-cholesterol levels and a decrease in LDL-cholesterol levels so the first target of a dyslipidemic therapy is LDL management. Dyslipidemic management is carried out by altering lifestyles, intervening in suitable diets to reduce LDL levels, and increasing HDL levels [14, 37]. The degree of compliance with diet interventions is seminal to ensure successful dyslipidemic management. Recommended dietary patterns for dyslipidemia are listed in **Table 3** [14, 37].

No.	Nutrition	Recommended intake
1.	Total fat	25–35% of the total calorie
2.	Saturated fat	Less than 7% of the total calorie
3.	Trans fatty acid	0
4.	Polyunsaturated acid	Maximal 10% of the total calorie
5.	Monounsaturated acid	Maximal 20% of the total calorie
6.	Carbohydrate	50–60% of the total calorie
7	Fiber	25–30 g/day
8.	Protein	±15% of the total calorie
9.	Cholesterol	Less than 200 mg/day
10.	Total calorie (energy)	Energy intake and expenditure should be in balance to maintain the desired body weight and prevent weight gain.

#### Table 3.

Recommended dietary patterns for patients with Dyslipidemia [14, 37].

The recommended dietary patterns should be in control using the following indicators [14, 24, 28]:

- 1. An increase in saturated fat in the diet by 1% would increase LDL by about 2%. A decrease in saturated fat intake would decrease LDL levels by 8%.
- 2. High saturated fat intake correlated with high coronary heart disease within a population.
- 3. Trans fatty acid is able to increase serum LDL levels so it would be better not to consume or consume it at a minimum intake level.
- 4. If monounsaturated, fatty acid changed saturated one, it is able to reduce LDL but not HDL or increase triglycerides.
- 5. If polyunsaturated fatty acid changed saturated one, it is managed to reduce LDL and, at a high dose, HDL too.
- 6. If saturated fat and trans fat intake are minimal, restricting total fat intake is unnecessary.
- 7. Carbohydrate intake should be less than 60% to reduce risks of decreased HDL and increased serum triglycerides.
- 8. Consuming 5–10 g/day of soluble fibers is managed to decrease LDL levels by 5%.

Fiber intake in diets, particularly soluble fibers, has the ability to produce gel in the intestines, inhibiting glucose and cholesterol absorption [38]. Dietary fibers have the ability to bind bile salts in the digestive tract, and disturbed bile reabsorption will stimulate bile synthesis in the liver. Cholesterol is a precursor to bile synthesis, so increased bile synthesis would decrease cholesterol levels in the blood [39]. Short-chain Fatty Acid (SCFA), yielded from fiber fermentation in the colon, is also substantive in inhibiting the activity of hydroxymethyl GLUTaryl-CoA

#### Dietary Fiber and Dyslipidemia DOI: http://dx.doi.org/10.5772/intechopen.98838

reductase (HMG-CoA reductase) enzyme which is needed in forming mevalonates as the primary product for cholesterol synthesis [24, 38, 40].

Dyslipidemia has a significant role in systemic responses and inflammation in adipose tissues. Inflammation can increase intestinal permeability and adipose tissues. A dyslipidemic condition also influences the composition of intestinal microflora and has a direct impact on body weight. Intestinal microbes were the primary sources of several molecules, e.g., lipopolysaccharides and peptidoglycan, as the causes of inflammation in periphery tissues [41]. An increase in Short-chain Fatty Acid (SCFA) in the intestines could reduce the intestinal pH, which indirectly altered the composition of intestinal microflora by cutting the number of harmful/pathogenic bacteria. In terms of the inflammatory process brought about by dyslipidemia, butyrates and propionates suppressed NF $\kappa\beta$  \activation through the inhibitory pathway for the activation of the I $\kappa$ B Kinase (IKK) protein complex, decreasing synthesis and cytokine secretion and proinflammatory adhesion molecules [28, 42, 43].

Previous studies argued that propionates could reduce free-plasm fatty acid levels through a lipolysis inhibition process in adipose tissues, reducing TLR4 receptor expressions to be able to bind with a free fatty acid, which consequently reduced TLR4 expression which could stimulate NF $\kappa\beta$  activation and translocation. The ratio of butyrate effects to inhibition in an inflammatory process is higher than propionates [43, 44].

#### 2.3 Fiber, dyslipidemia and its relationship with autoimun disease

Inflammation is one of the immune response which if it does excessive can cause autoimmune diseases. Inflammation is often followed by an increase in ESR, CRP, cytokines and complement activation which is able to result in tissue damage. Dyslipidemia is a potential trigger for chronic inflammation. Thereby, dyslipidemia is associated with autoimmune diseases. Although it is not clear whether dyslipidemia is a predisposing factor or an outcome of autoimmune disease. In line with this, an obesity is managed to increase the incidence and severity of several autoimmune diseases, such as psoriasis and rheumatoid arthritis. Cytokines in the form of IL-17a are known to be pathogenic in psoriasis and RA. For this reason, IL-17a blocking is used as the treatment of autoimmune diseases [45].

Furthermore, the autoimmune condition plays a role in the development of dyslipidemia and atherosclerotic plaque formation in patient with autoimmune rheumatoid disease (ARD), such as RA. This is related to the formation of autoantibodies and chronic inflammation which occured. As in SLE, ox-LDL is also frequently find in synovial biopsy specimens of RA patients. The product of ox-LDL is able to be recognized by the scavenger receptor and influence the action of macrophages. Meanwhile, the results of ox-LDL digestion by macrophages can be toxicable to endothelial cells, chemotaxis of inflammatory cells and cause changes in smooth muscle function. In the state of dyslipidemia, especially hyperlipidemia, serum LDL levels are elevated [46].

Additionaly, increasing endogenous butyrate production is managed to be a valuable strategy in the prevention of obesity and related metabolic diseases. However, in the other side, this also can increase exogenous intake through butyrate supplements. Most likely, the causative lack of randomized controlled trials proving the efficacy of butyrate in these metabolic disorders is mainly due to the poor palatability of the actual butyrate preparations available on the market. Nevertheless, there is an urgent need for products that mask the unpleasant organoleptic properties of butyrate, in oder to facilitate clinical studies in children and in adult patients [47]. Increasing interest in the effect of dietary fiber, on lowering the blood lipid concentration. There are various mechanisms by which serum and hepatic lipids are reduced by dietary fiber: binding to bile, viscosity, and bucking in the small intestine caused the suppression of glucose and lipid absorption, increased production of SCFAs, and modulation of lipid metabolism-related genes. In addition, dietary fibers, classified as the seventh nutrients, are generally considered safe, but overconsumption could cause intestinal discomfort. From the above evidences, dietary fibers could be used as alternative supplements to exert health benefits, including lipid-lowering effects on humans. However, more clinical evidence is needed to strengthen this proposal and its fully underlying mechanism still requires more investigation. Only if we fully understand the mechanism and dose relationship of each kind of DFs we are able to apply them in the intervention of hyperlipidemic patients [48].

In populations who habitually consume diets rich in plant foods, great adherence to three types of plant-based diets were differentially associated with risk of incident dyslipidemia. Study result strongly supports considering the quality of plant foods for dyslipidemia prevention. Prospective studies are needed to confirm the relationship between a plant-based diet and dyslipidemia in diverse populations with different dietary habits [49].

## 3. Conclusions

Dyslipidemia has an important role in systemic and inflammatory responses in adipose tissue. Inflammation can increase the permeability of the intestines and adipose tissue. One indicator of dyslipidemia is an increase in LDL cholesterol levels and a decrease in LDL cholesterol levels so that the first target of dyslipidemia therapy is LDL management. Dyslipidemic management is carried out by changing the lifestyle, intervening in an appropriate diet to reduce LDL levels, and increasing HDL levels. The level of adherence to dietary interventions is critical to ensure successful dyslipidemic management. The recommended dietary pattern should be controlled using the following indicators. Increasing saturated fat in food by 1% will increase LDL by about 2%. Decreasing saturated fat intake will reduce LDL levels by 8%.

#### Acknowledgements

This chapter is supported by Politeknik Kesehatan Mataram and Badan Pengembangan dan Pemberdayaan Sumberdaya Manusia, Ministry of Health Republic of Indonesia.

#### Notes/thanks/other declarations

Thank you for using this reference as input for the management of dyslipidemia.

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