Ann Res Antioxid. 2018;3:e08 Mini-Review

Annals of Research in Antioxidants http annresantioxidants.com

Diabetic kidney disease and probiotic soy milk

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Received 9 August 2017
Accepted 13 December 2017
ePublished 3 January 2018

Keywords: Diabetic nephropathy, Probiotic soymilk, Reactive oxygen species, Probiotics, Gut microbiota, End-stage renal failure

Citation: Nasri H. Diabetic kidney disease and probiotic soy milk. Ann Res Antioxid. 2018;3:e08.

Abstract

Diabetic nephropathy (DN) or diabetic kidney disease (DKD) is a serious complication of diabetes. Various mechanisms were suggested for the development of DKD. The high production of reactive oxygen species (ROS) is the central and major mediator of diabetes tissue damage. Some studies showed disruption of normal gut microbiota (dysbiosis) associated with systemic and metabolic disease, such as chronic kidney disease. Other studies also demonstrated the correlation between oxidative stress and misbalance of gut bacteria and suggested the consumption of probiotics in diabetic patients. This paper will review the research conducted on the effect of probiotic soy milk on DN.

Introduction

Diabetic nephropathy (DN) or diabetic kidney disease (DKD) is a serious microvascular complication of diabetes. Both types (1 and 2) of diabetes mellitus (DM) can cause nephropathy, but in the second type of DM, a lesser proportion progress to end-stage renal failure. The incidence of DN is increasing worldwide (1). It has been predicted that more than 30% of diabetic patients will get chronic kidney disease (2). DN develops in five stages: Stage I, the size of the kidneys and renal plasma flow is raised, but with no albuminuria or hypertension. Stage II, the thick of basement membrane is increased and mesangial cell is proliferated, but glomerular filtration rate (GFR) is normal; stage III, glomerulus is damaged and microalbuminuria (albumin 30-300 mg/d) is seen; Stage IV, GFR is decreased below 60 mL/min/1.73 m^2 and proteinuria (>300 mg/d) and sustained hypertension is seen; and in Stage V, ESRD with GFR <15 mL/min/1.73 m^2 is revealed (3).

Materials and Methods

For this review, we used a variety of sources including PubMed/Medline, EBSCO, EMBASE, Web of Science, Google Scholar, Scopus and directory of open access journals (DOAJ). The search was conducted by using combinations of the following key words and/or their equivalents; diabetic nephropathy; probiotic soymilk; reactive oxygen species; probiotic; gut microbiota, hypertension, obesity, inflammation, insulin resistance, dyslipidemia, vitamin D deficiency, angiotensin II Endothelin-1, end-stage renal failure, diabetic kidney disease, fibrosis, dysbiosis and chronic kidney disease,

Core tip
Various studies showed disruption of normal gut microbiota (dysbiosis) associated with systemic and metabolic disease, such as chronic kidney disease.

Risk factors for diabetic kidney disease

Risk factors for determining the incidence of DN are hyperglycemia, male sex, hypertension, obesity, inflammation, insulin resistance, dyslipidemia, vitamin D deficiency and genetic factors (2).

Mechanisms for the development of diabetic kidney disease

Mechanisms suggested for the development of DKD were totally divided into hemodynamic and metabolic pathways.

Metabolic pathways of diabetic kidney disease

Hyperglycemia increases the induction of excess superoxide by the electron-transfer chain and then inhibits glyceraldehyde-3-

phosphate dehydrogenase (GADPH). GADPH inhibition stops glycolysis (4). This high production of reactive oxygen species (ROS) is the central and major mediator of diabetes tissue damage, which causes the activation of five pathways involved in the pathogenesis of complications via various mechanisms; the non-enzymatic glycosylation reaction, the hexosamine pathway, and the electron transport chain in mitochondria are the resources of ROS creation in cells of diabetes (5). The mitochondria electron transport chain is a major source of ROS in insulin secretion cells, insulin peripheral sensitive cells and endothelial cells (6). ROS increased in both glomerular mesangial and proximal tubular cells in the kidney because of hyperglycemia. Evidence indicates that ROS may play a main role in the development of diabetic complications such as nephropathy. The main mechanism that hyperglycemia leads to increased ROS and structural changes related with DN are not well recognized, however, it seems nicotinamide adenine dinucleotide phosphate-oxidase (NADPH oxidase) and mitochondrial electron gradients play important roles in hyperglycemia-induced ROS production.

Hemodynamic pathways of diabetic kidney disease

ROS activity can increase the levels of angiotensin II which subsequently lead to the limitation of efferent arteriola. Increased levels of angiotensin II are related to high albumin in the urine and nephropathy in patients (7). Endothelin-1(ET-1) is another strong limitator of the efferent arteriole. One of the physiologic functions of ET-1 in the kidneys can be noted to imitating ROS including constriction of the vessels that can cause high blood pressure, impairment of the function of endothelium, inflammation, and fibrosis. Furthermore, high expression of ET-1 can cause the enlargement and multiplication of mesangial cell and production of extracellular matrix. It can also cause albuminuria and development of DN by increasing permeability of glomerulus (8).

Gut microbiota and diabetic kidney disease

The gut microbiota has been appeared to participate in specific metabolic activities. Disruption of normal gut microbiota (dysbiosis) associated with systemic and metabolic disease, such as chronic kidney disease. Recent studies revealed quantitative and qualitative changes in gut microbiota in patients with chronic kidney disease. In addition, dysbiotic intestinal microbiome may participate in progression chronic kidney disease and its complications. The investigation determined dietary factors in short or long term is one of the most important factors that influence the diversity and constitution of the human intestinal microbiota so affecting host metabolism and disorder risk or progress .In addition, elevated interest has created in using probiotics, and prebiotics to decrease the risk of dysbiosis in the intestinal to prevent or cure the human illnesses. The intestinal dysbiosis in chronic kidney disease patient may be due to several factors such as uremia, dietary factor, and iatrogenic reasons (9).

Probiotic

Probiotics, as expressed by the World Health Organization (WHO), are "live microorganisms which when given in suitable quantities confer a health benefit on the host". Probiotics comprise living bacteria, for instance, lactobacilli, bifidobacteria species, and streptococci, which may modify intestinal microbiota and influence health state. The health-promoting effects of probiotics may be associated with their ability to produce anti-bacterial combinations, modulate pH, produce antioxidants, vitamins, and compete with pathogens (10-12). Other studies also demonstrated the correlation between oxidative stress and disbalance of gut bacteria and suggested the consumption of probiotics in diabetic patients (13).

Probiotic soy milk

High intake of probiotics is possible by fortification of foods. Dairy foods have been expanded as fortified products by prebiotics (13). Intake of dietary phosphorus should be limited in diabetic patients with nephropathy. Since the content of phosphorus is the lowest in soy milk compared with dairy foods, adding prebiotics to soy milk might be a good choice for diabetic patients with nephropathy (14). As our knowledge the studies in this field is rare. We reviewed studies on the effect of fortified soy milk with *Lactobacillus plantarum* A7 on the biological markers of oxidative stress including malondialdehyde (MDA), oxidized glutathione (GSSG), reduced glutathione (GSH), 8-iso-prostaglandin F2a (8-iso PGF2a), glutathione peroxidase (GSH-Px), glutathione reductase (GR), and total antioxidant capacity (TAC) among type 2 diabetic patients with nephropathy.

Recently, 48 diabetic individuals (22 males and 26 females) with nephropathy were enrolled for competency by Miraghajani et al in November 2013 until February 2014. Patients were randomly devoted to two equal groups include the control group (soy milk diet) and intervention group (probiotic soy milk diet). The control group and the intervention group received 200 ml/d soy milk and probiotic soymilk respectively for the 8-weeks. All patients received a diet with calculated daily energy, protein, sodium, and potassium. The results of this survey showed GSH were higher in the probiotic soymilk group compared with soymilk group at the end of the study, but GSSG decreased in the probiotic soymilk group. Furthermore, the levels of GPx and GR as antioxidant enzymes increased within the two intervention groups. The activity levels of GPx and GR as antioxidant enzymes increased significantly between two groups. Furthermore, serum levels of 8-iso-PGF2a or MDA did not significantly reduce and TAC concentrations didn't significantly induce within and between two groups (14).

Abbasi and colleagues also investigated "the impacts of soy milk containing *L. plantarum* A7 on the improvement of kidney function in diabetic patients with nephropathy" among 44 patients with DN. They were randomly assigned to control group whom received 200 ml/d common soymilk and an intervention group that received an equal amount of soymilk comprising probiotic-fortified soy milk (soy milk containing *L. plantarum* A7) for 8 weeks. Results of this survey revealed creatinine, interleukin-18, and sialic acid reduced in the serum of patients taken probiotic soymilk compared with conventional soy milk. Albuminuria also reduced in patients taken probiotic soymilk compared with conventional soy milk. Moreover, the estimated rate of glomerular filtration significantly improved in probiotic-fortified soymilk group compared with the control group (14).

Conclusion

In summary, our review indicates that ROS is the central and major mediator of diabetes tissue damage and there is the correlation between oxidative stress and disbalance of gut bacteria and consumption of probiotic soy milk with *L. plantarum* A7 has a number of health benefits to patients with diabetes, and it is a hopeful strategy for delaying of renal dysfunction.

Authors' contribution

HN is the single author of the manuscript.

Conflicts of interest

The author declared no competing interests.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

Funding/Support

None.

References

1. Martínez-Castelao A, Navarro-González JF, Górriz JL, de Alvaro F. The concept and the epidemiology of diabetic nephropathy have changed in recent years. J Clin Med. 2015;4:1207-16. doi: 10.3390/jcm4061207.
2. Jayakumar RV. Risk factors in diabetic nephropathy. Int J Diabetes Dev Ctries. 2012;32:1-3.
3. Reidy K, Kang HM, Hostetter T, Susztak K. Molecular mechanisms of diabetic kidney disease. J Clin Invest. 2014;124:2333-40. doi: 10.1172/JCI72271.
4. Brownlee M. Biochemistry and molecular cell biology of diabetic complications. Nature. 2001;414:813-20. doi: 10.1038/414813a.
5. Kaneto H, Nakatani Y, Kawamori D, Miyatsuka T, Matsuoka TA. Involvement of oxidative stress and the JNK pathway in glucose toxicity. Rev Diabet Stud. 2004;1:165-74. doi: 10.1900/RDS.2004.1.165.
6. Kassab A, Piwowar A. Cell oxidant stress delivery and cell dysfunction onset in type 2 diabetes. Biochimie. 2012;94:1837-48. doi: 10.1016/j.biochi.2012.01.020.
7. Huang W, Gallois Y, Bouby N, Bruneval P, Heudes D, Belair MF, et al. Genetically increased angiotensin I-converting enzyme level and renal complications in the diabetic mouse. Proc Natl Acad Sci U S A. 2001;98:13330-4. doi: 10.1073/pnas.231476798.
8. Franzén S, Pihl L, Fasching A, Palm F. Internal activation of endothelin type B receptors improves kidney oxygnation in type 1 diabetic rats. Am J Physiol Renal Physiol. 2018;314:F439-44. doi: 10.1152/ajprenal.00498.2017.
9. Kafeshani M. The gut microbiome, diet, and chronic kidney disease. J Prev Epidemiol. 2017;2:e05.
10. Christensen EG, Licht TR, Kristensen M, Bahl MI. Bifidogenic effect of whole-grain wheat during a 12-week energy-restricted dietary intervention in postmenopausal women. Eur J Clin Nutr. 2013;67:1316-21. doi: 10.1038/ejcn.2013.207.
11. Zakostelska Z, Kverka M, Klimesova K, Rossmann P, Mrazek J, Kopecny J, et al. Lysate of probiotic Lactobacillus casei DN-114 001 ameliorates colitis by strengthening the gut barrier function and changing the gut microenvironment. PloS one. 2011;6:e27961. doi: 10.1371/journal.pone.0027961.
12. Miraghajani MS, Najafabadi MM, Surkan PJ, Esmaillzadeh A, Mirlohi M, Azadbakht L. Soy milk consumption and blood pressure among type 2 diabetic patients with nephropathy. J Ren Nutr. 2013;23:277-82.e1. doi: 10.1053/j.jrn.2013.01.017.
13. Miraghajani M, Zaghian N, Mirlohi M, Feizi A, Ghiasvand R. The impact of probiotic soy milk consumption on oxidative stress among type 2 diabetic kidney disease patients: a randomized controlled clinical trial. J Ren Nutr. 2017;27:317-324. doi: 10.1053/j.jrn.2017.04.004.
14. Abbasi B, Ghiasvand R, Mirlohi M. Kidney function improvement by soy milk containing *Lactobacillus plantarum* a7 in type 2 diabetic patients with nephropathy. Iran J Kidney Dis. 2017;11:36-43.