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SHOULD MONO-UNSATURATED AND POLY-UNSATURATED FAT PREVENTS THE PROGRESSIVITY PREDIABETES ON WOMAN?

[1]Iche Andriyani Liberty, [2]Ratu Ayu Dewi Sartika

[1]Department of Public Health and Community Medicine, Medical Faculty, Sriwijaya University
[2]Department of Public Nutrition, Public Health Faculty, University of Indonesia

Email: iche.aliberty@gmail.com

ABSTRACT

Most evidence showed that women with diabetes mellitus have a higher risk for cardiovascular disease. Prediabetes is a reversible state that can progressive for future complications or regressive to normoglycemic. The weight loss that prevents progressivity from prediabetes can be achieved by regulating dietary composition. The dietary composition may affect pancreatic β-cell function and insulin sensitivity. Consumption of foods rich in MUFA and PUFA has a positive effect on metabolism, which is associated with an increase in the position of adiponectin, anti-inflammatory cytokines, which increase liver elevation, and reduce atherosclerosis, such as decreased triglycerides, LDL and VLDL and increased HDL. This article explores relevant literature on this subject in order to identify and elaborated the knowledge for further research.

Keywords: MUFA, PUFA, Progressivity, Prediabetes

Prediabetes is a "golden period" in preventing and / or delaying conversion to diabetes, but is very progressive for future complications [1]. People with prediabetes will conversion to diabetes about 4-10 years with a risk of 25% -65% compared to people with normoglycemic which is only 5% [2–4]. This estimated showed that one-third of people with prediabetes will convert to diabetes, one-third regression to normoglycemic and one- third remain in prediabetes status [2]. This period is a time that should be a concern in order to develop optimal prevention efforts.

Women with diabetes mellitus have a higher risk for morbidity and mortality due to cardiovascular disease [5–7].The prevalence of reproductive women with diabetes diagnosed has increased, so that more women enter pregnancies with pre-existing diabetes. According to IDF (2017), currently 1 in 10 women in the world have diabetes and 2 out of 5 of them are at reproductive age [8].

The result of study from Liberty et al (2018) showed that from 371 women with prediabetes status at the beginning of observation, in the 3rd year observation as much as 40.16% (149 subjects) regressive to normoglycemic, 50.67% (188 subjects) did not have conversion, but 9.16% (34 subjects) had progressivity to diabetes. While in the 5th year of observation, 42.05% (156 subjects) had regression to normoglycemic, 36.93% (137 subjects) had no conversion, and 21.02% (78 subjects) had progressivity to diabetes [9].

The weight loss that prevents progressivity from prediabetes can be achieved by regulating dietary composition. The dietary composition may affect insulin sensitivity and pancreatic β-cell function. Low-fat, low-energy carbohydrate diets can lower liver glucose and increase insulin sensitivity in skeletal muscle. Decreased glucose of the liver can be achieved by weight loss of 2%, while increased insulin sensitivity in skeletal muscle can be achieved with a weight loss of 7% [10].

FFA (Free Fatty Acid) and Insulin Resistance

Insulin resistance is closely related to changes in fatty acid / FFA (Free Fatty Acid) patterns in plasma. Fatty acids can be distinguished by their long chains and saturation levels and it has been suggested that the FA composition of diets may differ in the physiological responses of the human body. According to Lyons, Kennedy, & Roche (2016), excess the FFA, in turn, can activate an inflammatory pathway and damage the normal cell signals inside immune cells, adipose tissue, liver, and muscle, leading to cellular dysfunction [11].

As a result, there is a metabolic disorder such as insulin resistance that causes prediabetes that can develop into diabetes. Implications of free fatty acids that promote insulin resistance in major metabolic organs produce lipotoxicity and glucotoxicity. All of these disorders cause insulin signalling disorders and uncontrolled glucose homeostasis. Differential modulation by fatty acids occurs, where saturated fatty acids (SFA) worsen the situation, while monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFAs) reduce the state of this metabolic inflammation.[11]

MUFA and PUFA Reduce Inflammatory

MUFA, also called monounsaturated fat, is an easily digestible fatty acid. The molecule is composed of a series of carbon atoms having one double bond. This double bond causes these fatty molecules to be unsaturated (can still add hydrogen atoms). Monounsaturated fats are usually in the liquid phase at room temperature and will freeze when cooled. Source of monounsaturated fats generally also contain other nutrients such as in some types of vegetable oils. These types of fats are among others found in nuts (almonds, pistachio, macadamia, hazelnut), avocados, and olive oil.

PUFA, Polyunsaturated fatty acids were fats containing more than one double bond[12]. The polyunsaturated fatty acid will lose at least 4 hydrogen atoms (H). In a polyunsaturated fatty acid diet generally lowers blood cholesterol as follows: every 1% increase in calories from polyunsaturated fatty acids in the diet results in a cholesterol reduction of approximately 1⁄2 mg/dl. PUFA consists of omega-3 (n-3), omega-6 (n-6), and omega-9 (n-9) fatty acids. PUFA food sources include marine fish, nuts, seeds, legumes, and sesame[11].

Fatty acids play an important role in energy balance, carbohydrate and lipid metabolism, and gene regulation. The profile of fatty acid plasma is influenced by two equally important factors namely the intake of dietary fat and endogenous fatty acid metabolism associated also with desaturase enzyme, which regulates the degree of lipid unsaturation throughout the body. Desaturase delta-9 (D9D) catalyses the conversion of palmitic acid and stearate (C18: 0) to monounsaturated fatty acids (MUFA), palmitoleic acid (C16: 1) and oleic acid (C18: 1, OA) respectively.[12]

It has been observed that delta-9 desaturase activity is high in conditions such as diabetes, atherosclerosis, obesity and metabolic syndrome[12]. Gender differences have been reported in obese subjects for D9D, with higher activity in women than in men. The studies show that replacing saturated fatty acids in the diet with either MUFA or PUFA results in changes in serum fatty acid profiles and improves insulin sensitivity.[13]Thus, many of the dietary guidelines recommend increasing foods rich in monounsaturated fat (MUFA) and reducing saturated fat (SFA).[14]

Research conducted by Krishnan, Steffen, Paton, & Cooper (2017) revealed a high MUFA diet can improve glycaemic control and lower overall insulin levels when compared with taking PUFA, SFA or carbohydrates [15]. MUFA intake of 10-15% of total daily energy is associated with a reduction of up to 10% IFG incidence. Meta-analysis by Imamura et al (2016) revealed that replacing the 5% energy of carbohydrates with SFA had no significant effect on fasting glucose, but lowering fasting insulin. Replacing carbohydrates with MUFA decreases HbA1c, and decreases HOMA-IR. Replacing carbohydrates with PUFA significantly decreases HbA1c and fasting insulin. Replacing SFA with PUFA significantly decreases glucose, HbA1c, C-peptide, and HOMA-IR. Based on the acute insulin response with a gold standard in ten RCTs, PUFA significantly increased the capacity of insulin secretion .[14] Replacement of SFA with PUFA is also associated with greater triglyceride reduction and increased endothelial function than MUFA. The results of this RCT suggest PUFAs rather than MUFAs that are an option for replacement of saturated fatty acid calories in women with Mets who are losing weight[16].

Similar findings were also expressed by Jakobsen et al (2009), that in order to prevent coronary heart disease, the SFA intake should be replaced with PUFA intake rather than MUFA or carbohydrate intake and the substitution effects of carbohydrates may vary depending on the quality of carbohydrates consumed [17]. Compared to PUFA, MUFA comes from a wide variety of foods including red meat, milk, nuts, and vegetable oils; the cardiometabolic effects of these different foods vary greatly: red meat and especially processed meats seem to increase the risk of diabetes; milk, cheese, and yogurt seem relatively neutral or less useful; while certain plant sources of MUFA, such as nuts and olive oil, have cardiometabolic benefits [18].

A logical reason for greater weight loss by PUFA than in MUFA is a greater increase in the angiogenic peptide hormone YY after PUFA intake compared with MUFA or SFA [15]. The other experimental evidence also showed that PUFAs can suppress oxidative stress, hepatic lipogenesis and steatosis, pancreatic lipotoxicity and insulin resistance [18]. The high-SFA diet modulates inflammatory processes with macrophage infiltration and other immunological cells and promotes the production of M2 macrophages with M1 macrophages that cause insulin resistance [19-20].

CONCLUSION

The consumption of food rich in MUFA and PUFA has a positive effect on metabolism, PUFA and or MUFA intake is associated with enhancing the position of adiponectin, an anti- inflammatory cytokine, which increases liver enhancement and reduces atherosclerosis. In order to provide references to obese women with prediabetes, reducing carbohydrate intake by replacing it with foods rich in MUFA and/or PUFAs, will

help to lose weight and correct problems, so progression can be prevented.

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REFERENCES

[1]. Liberty; IA, Kodim N. Assess prediabetes risk, as a golden period for prevention of diabetes. Asian J Pharm Clin Res. 2017;10(6):349-353. doi:http://dx.doi.org/10.22159/ajpcr.2017.v10i6.18215

[2]. Garber AJ, Handelsman Y, Einhorn D, et al. ACE / AACE Consensus Statement Diagnosis and Management of Prediabetes in the Continuum of Hyperglycemia — When Do the Risks of Diabetes Begin ? A Consensus Statement From the American College of Endocrinology and the American Association of Clinical Endo. 2008;14(7):933-946.

[3]. Geiss LS, James C, Gregg EW, Albright A, Williamson DF, Cowie CC. Diabetes Risk Reduction Behaviors Among U.S. Adults with Prediabetes. AMEPRE. 2010;38(4):403-409. doi:10.1016/j.amepre.2009.12.029

[4]. Tuso P. Prediabetes and Lifestyle Modification: Time to Prevent a Preventable Disease. Perm J. 2014;18(3):88-93. doi:10.7812/TPP/14-002

[5]. Kautzky-Willer A, Kamyar MR, Gerhat D, et al. Sex-Specific differences in metabolic control, cardiovascular risk, and interventions in patients with type 2 diabetes mellitus. Gend Med. 2010;7(6):571-583. doi:10.1016/j.genm.2010.12.001

[6]. Guo VYW, Yu EYT, Wong CKH, et al. Ovarian aging in women with diabetes: An overview. Women's Heal Issues. 2017;25(4):732-738. doi:10.1016/j.whi.2015.06.012

[7]. De Abreu L, Holloway KL, Kotowicz MA, Pasco JA. Dysglycaemia and other predictors for progression or regression from impaired fasting glucose to diabetes or normoglycaemia. J Diabetes Res. 2015;2015. doi:10.1155/2015/373762

[8]. IDF. Diabetes Voice, Global Perspektives on Diabetes. 2017;(2 July).

[9]. Iche Andriyani Liberty. Predictive Value of Triglyceride Glucose Index as A Acurate Marker of Prediabetes Conversion Among Women Reproductive Age. DIssertation of Epidemiology Doctoral Program. Universitas Indonesia. 2018.

[10]. Qian F, Korat AA, Malik V, Hu FB. Metabolic effects of monounsaturated fatty acid- enriched diets compared with carbohydrate or polyunsaturated fatty acid-enriched diets in patients with type 2 diabetes: A systematic review and meta-analysis of randomized controlled trials. Diabetes Care. 2016;39(8):1448-1457. doi:10.2337/dc16-0513

[11]. Lyons CL, Kennedy EB, Roche HM. Metabolic inflammation-differential modulation by dietary constituents. Nutrients. 2016;8(5). doi:10.3390/nu8050247

[12]. Rondanelli M, Klersy C, Perna S, et al. Effects of two-months balanced diet in metabolically healthy obesity: lipid correlations with gender and BMI-related differences. Lipids Health Dis. 2015;14(1):1-11. doi:10.1186/s12944-015-0131-1

[13]. Alsharari ZD, Risï¿½rus U, Leander K, et al. Serum fatty acids, desaturase activities and abdominal obesity - A population-based study

of 60-year old men and women. PLoS One. 2017;12(1):1-15. doi:10.1371/journal.pone.0170684

[14]. Imamura F, Micha R, Wu JHY, et al. Effects of Saturated Fat, Polyunsaturated Fat, Monounsaturated Fat, and Carbohydrate on Glucose-Insulin Homeostasis: A Systematic Review and Meta-analysis of Randomised Controlled Feeding Trials. PLoS Med. 2016;13(7):1-18. doi:10.1371/journal.pmed.1002087

[15]. Krishnan S, Steffen LM, Paton CM, Cooper JA. Impact of dietary fat composition on prediabetes: a 12-year follow-up study. 2017;20(9): 1617-1626. doi:10.1017/ S1368980016003669

[16]. MillerM,SorkinJD,MastellaL,etal.managementINtheMetabolicSyndrome :The MUFFIN Study. 2017;10(4):996-1003. doi:10.1016/j.jacl.2016.04.011.Poly

[17]. Jakobsen MU, Reilly EJO, Heitmann BL, et al. Major types of dietary fat and risk of coronary heart disease : a pooled analysis of 11 cohort studies 1 – 3. Am J Clin Nutr. 2009;89:1425-1433. doi:10.3945/ajcn.2008.27124.Am

[18]. Martínez-González MA. Benefits of the Mediterranean diet beyond the Mediterranean Sea and beyond food patterns. BMC Med. 2016;14(1):1-3. doi:10.1186/s12916-016-0714-3

[19]. Garcia-ArellanoA,RamallalR,Ruiz-CanelaM,etal.Dietary inflammatory index and incidence of cardiovascular disease in the PREDIMED study. Nutrients. 2015;7(6):4124-4138. doi:10.3390/nu7064124

[20]. Figueiredo PS, Inada AC, Marcelino G, et al. Fatty acids consumption: The role metabolic aspects involved in obesity and its associated disorders. Nutrients. 2017;9(10):1-32. doi:10.3390/nu9101158