



# Dietary Changes and the Metabolic Syndrome:

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## Abstract:

The Metabolic syndrome is increasing globally. The clustering of risk factors, leading to cardiovascular disease and T2DM, which are of metabolic origin.

Recently the International Diabetic Federation (IDF) provided worldwide definition for MS which considers central obesity and insulin resistance as important factors. IDF group further highlighted pro-inflammatory state as a (major factor) main initiator of the syndrome. In the context of globalization of food systems, people's diet pattern shifted to more processed food and ready to eat convenient food instead of minimally processed, plant based food. According to the recent studies on processed food suggests that higher intake of these foods are directly associated with Overweight/ Obesity and MS; highlighting the dietary role in modulating the risk of metabolic syndrome risk factors.

Till now, we are treating each condition of metabolic syndrome risk factors separately; instead of implementing therapies that reduce all the risk factors simultaneously. From recent research it is clear that dietary changes approach which target all of the risk factors and targeting the striking underlying causes of obesity and Insulin resistance will be the first step in the management of Metabolic Syndrome.

**(Key words: metabolic syndrome, processed food, central obesity).**

## Introduction:

Metabolic syndrome (MS) is clustering disorder increasing worldwide. MS is defined as a complex of inter connected factors leading to coronary, heart disease, vascular disease and diabetic mellitus or Type-2 diabetes. It's main element are dyslipidemia (Elevated Triglycerides) , ↓HDL, elevated blood pressure, imbalance in glucose homeostasis, central obesity (increased waistline) and insulin resistance are the major manifestation of the MS. According to Grundy and Zimmet (2005) research, “ it is well accepted that the metabolic syndrome increases the risk factor for the development of CVD, T2DM stroke and cancer.

From the past few years, several expert groups have attempted to set forth simple diagnostic criteria to be used for clinical practice to identify MS. These criteria varied in specific elements. Recently, the International diabetic foundation (IDF) published new criteria for MS clinical definition that the presence of abdominal obesity is essential for diagnosis. They considered the abdominal obesity is so highly correlated with insulin resistance and more laborious measures of insulin resistance are unnecessary.

<b>Table :1 IDF(INTERNATIONAL DIABETES FEDERATION) Worldwide definition for Metabolic Syndrome:</b>	
<b>CENTRAL OBESITY:</b>	
Waist Circumference - ethnicity specific.	
Plus any two of the following:	
<b>Raised Triglycerides:</b>	
Greater than or equal to 150mg/dl or on medication.	
<b>Reduced HDL levels:</b>	
Less than 40mg/dl in males	
Less than 50mg/dl in females or on medication.	
<b>Raised Blood Pressure:</b>	
Systolic: greater than 130 mmHg	
Diastolic: greater than 85 mmHg or on medication	
<b>Raised Fasting Plasma Glucose :</b>	
Fasting Plasma Glucose: greater than 100mg/dl or previously diagnosed type 2 Diabetes.	

**Table :2 Ethnic specific cut points for Waist Circumference:**

Ethnic group	Waist circumference(as measured of central obesity)
Euripides	Male $\geq 94$ cm, Female $\geq 80$ cm
South Asians	Male $\geq 90$ cm, Female $\geq 80$ cm
Chinese	Male $\geq 90$ cm, Female $\geq 80$ cm
Japanese	Male $\geq 85$ cm, Female $\geq 90$ cm

Ref: Alberti G, Zimmet et al,2004, IDF,2005

Nesto (2003) and others researched findings also confirms the same as Central Obesity and insulin resistance as important causative factors. Individuals with these factors manifest a prothrombotic state and pro-inflammatory condition. Initially, MS was referred as if a discrete entity with single cause, yet, available research data suggest that it has more than one cause. The main causes according to research are abdominal obesity, insulin resistance, Physical inactivity, aging, hormonal imbalance and atherogenic diet can enhance risk for developing CVD. Although the diet is not listed specifically as an underlying risk factors for the condition. (NCEP(ATP-III)(2002).

Most recent research highlighting the potential role of diet on modulating risk of obesity and Metabolic Syndrome. According to Baker & Friel (2014) "The increasing globalization of food systems, the theory of the nutrition transition suggests that with economic development the population consumption pattern shifted from minimally processed diet rich in plant based staple to diets high in meat, fats and processed foods".

The wide spread availability of highly processed foods, which are rich in fructose than glucose are more contributing factor for damage and MS risk factors. This is strengthened by recent research studies on animals and humans. For a point of view studies by Barnard et al (1992, 1993, 1994) have documented that " diet appears to be a major factor in the metabolic syndrome and have shown that the syndrome can be induced in rats by feeding an high fat- refined sugar diet similar to the typical US-diet and can be controlled in humans by feeding a low fat complex carbohydrates diet" He also observed that within 2 weeks consumption high fat refined sugar diet, the animals were hyper insulinemic and demonstrated skeletal muscle insulin resistance by reduction in glucose transport, but there was no change in triglycerides, blood glucose levels, blood pressure and no indication of obesity. He also noted that " at 2 months , 6 months & 2 years plasma insulin was further increased ( than @ 2 week( abdominal fat cells were enlarged, significantly elevated triglycerides and hyper tension.)

One more study on baboons provides great insight on potential role of diet on metabolic syndrome. In this, four test groups of baboons were fed on different proportions of starch, glucose, fructose and sucrose, supplemented with appropriate amounts of other dietary requirements. All the groups showed elevated triglycerides. Starch & Glucose fed groups showed 37% increase and 65% in fructose fed groups. They also showed the development of fatty streaks in the aorta & this development was greatest in fructose fed group. (Kritchevsky, 1974).

The experimental studies (Steohanie seneff et al,2011,Lopez et al 2015) proves the adverse Metabolic effects of fructose when compared with glucose. Fructose is converted to lipid in the liver leading to (Fatty liver) development of high triglycerides, Low HDL levels, small , dense LDL & insulin resistance. Further studies on refined & processed foods, observed that protein glycation ; which are impaired in function and susceptible to more oxidation damage leading to pro inflammatory condition in the body.(Bray,2007,Vasanti et al,2010)

These glycated proteins accumulates in the blood serum and arterial walls leads to aging , atherosclerosis and issue related to diabetes(Person,1988). More recent study on the consumption of minimally processed , processed and ultra processed foods in a sample of Lebanese adults showed that higher adherence to the minimally processed /Diet pattern decreased odds of hyperglycaemia, triglycerides and MS risk factors( Tavares et al 2012, Louzada et al,2015,Lara,2017). Few more studies on ultra processed food also confirms the same effect of minimally processed foods, which are retaining their nutritional beneficial properties of phytochemicals, antioxidants, fiber and mono unsaturated fats which reduce the oxidative stress , inflammatory response, insulin fluctuations and MS risk factors.( Canella et al,2008-2009; Moubrance et al 2014;Martin et al 2013; Juul and Harmming son ,2015;Anderson et al,2009).

In summary , (Research on ) short- term & long term experiments on animals and human beings it is clear that a diet rich in processed food is the underlying factor responsible for MS risk factors. Even 2 weeks of short time on this diet leading to skeletal muscles insulin resistance. From these findings Dietary changes will be the first approach in the treatment and management of MS rather than treating each condition separately with pharmacological drugs initially.

#### References:

1. Praveen Sharma, Inflammation and the Metabolic Syndrome Ind J. Clinic –Bio Chem(Oct-Dec 2011) , 26 (4) : (317-318).
2. Eckel RH, Grundy SM, Zimmet P. The metabolic syndrome Lancet . 2005;365:1415-28.
3. Nesto RW. The relation of insulin resistance syndrome to risk cardiovascular disease. Rev. Cardio vasc. Med . 2003, 4 (6): S11-8.
4. Hu FB, Meigs JB, Li TY, Rifai N, Manson JE. Inflammatory markers and risk of developing type 2 diabetes in women. Diabetes 2004, 53:693-700.
5. Hankey AJ, Festa A, D' Agostino RB Jr, Wagenknecht LE, Savage PJ, Tracy RP, Saad MF, Haffner SM. Metabolic and inflammation variable clusters and prediction of type-2 diabetes : Factor analysis using directly measured insulin sensitivity. Diabetes .2004,53 : 1773-1781.
6. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes 1988; 37: 1595-1607.
7. National cholesterol Education program(NCEP) expert panel on Detection Evaluation and treatment of high blood cholesterol in adults (Adults treatment Panel III) . Third report of the N C E P expert panel on detection, Evaluation, and treatment of high blood cholesterol in Adults (ATP-III) final report. Circulation , 2002; 106 : 3143-3421.
8. Kikuchi S, Shinpok, Takeuchi M, Yamagishi S, Makita Z, Asaki N, Tashiro K, Glycation – a sweet tempter for neuronal death. Brain Res Brain Res Rev 2003; 41 : 306-23.
9. MC person JD, Shilton BH, Walton DJ. Role of fructose in glycation and cross linking of proteins. Bio chemistry 1988;27;1901-7.
10. Kritchevsky D, Davidson LM, Shapiro 12, et al . Lipid metabolism and experimental atherosclerosis in baboons: influence of cholesterol free, semi –synthetic diets. Am J Clin Nutr 1974; 27:29-50
11. Steohanie seneff, Glyn wain wright, Luea Mascitelli. Is the metabolic syndrome caused by a high fructose And relatively low fat, low cholesterol diet ? Arch Med sci 2011: 1:8-20.
12. Bray GA. How bad is fructose ? Am . J . Clin Nutr 2007; 86:895-896.
13. Vasanti S. Malik, Barry . Popkin, George A. Bray, Jean – Pierre despres, Walter C, Willett , Frank B. Hu Sugar-Sweetened beverages and Risk of Metabolic Syndrome and Type-2 diabetes. Diabetes care, 2010;33(11): 2477-2483.

14. Barnard R.J.D. J. Faria, J.E Menges, and D.A Martin Effects of a high –fat, Sucrose diet on Serum insulin and related atherosclerotic Risk factors in Rats. *Atherosclerosis* 100:229-236, 1993.

15. Barnard R.J, D.A Martin, E.J.Ugianskis, and S.B Inkeles. Role of diet and exercise in the management of hyper insulinemia and associated atherosclerotic risk factors. *Am J. Cardiol.* 69 :440-444, 1992.

16. Barnard, R.J and S.J new Exercise and diet in the prevention and control of the Metabolic Syndrome *sports Med.* 18;218-228, 1994.

17. R.James Barnard, Christion K. Roberts, shira M. Varon and Joshna J. Berger. Diet induced insulin resistance precedes other aspects of the Metabolic Syndrome. *J. Applphysiol* 84:1311-1315, 1998.

18. David son MB and D. Garvey studies on mechanism of hepatic insulin resistance in cafeteria-fed rats. *Am J Physiol.* 264. (Endocrinol Metab. 27) : E18-E23, 1993.

19. Grimditch G.K , R.J Barnard, E. Sterh licht, R.H Whitson and S.A Kaplan. Effect of diet on insulin binding and glucose transport in rat sarcolemmal vesicles. *Am J. Physiol.* 252 (Endocrinol. Metab. 15) E420-E425, 1987.

20. Sterlien L.H, D.A pan, A.P kriketos, and L.A BAur, High fat diet induced insulin resistance *Ann. Ny Acad Sci* 688:82-90, 1993.

21. Vrana A, L. Kazdova. Z. Dobesova. J. Kunes, V.Kren V. Bila, P.Stobla and I.Klines. Triglyceridemia, glucose regulation , and blood pressure in various rat strains. Effects of dietary carbohydrates *Ann. NY Acad Sci* 683:57-58, 1993.

22. Baker P & Friel S. Processed foods and the nutrition transition : evidence from Asia. *Obes Rev.* 15 564-577, 2014.

23. Tavares LF, Fonseca SC, Rosa MLG et al. Relationship between ultra processed food and Metabolic syndrome in adolescents from a Brazilian family Doctor program. *Public Health Nutr.* 15; 82-87, 2012

24. Da costa Louzada ML, Baraldi LG, Steele EM et al. Consumption of ultra processed food and obesity in Brazilian adolescents and adults *prev Med* 81, 9-15, 2015.

25. Canella Ds, Levy RB, Martins Ap B et al. Ultra processed food products and obesity in Brazilian house holds *plos one* 9, e 92752, (2008-2009).

26. Moubrance J-C, Betal M. Mactia Ap B, et al , processed and ultra processed food products : consumption trends In Canada from 1938 to 2011. *can JDiet Pract res* 75, 15-21,2014.

27. Martins Ap B, Levy RB., Claro RM et al . Increased contribution of ultra-processed food products in the Brazilian diet (1987-2009) , *Re V saude publica* 47, 656-665. 2013

28. Juul F & Hemmings son E; Trends in consumption of ultra processed foods and obesity in Sweden between 1960 & 2010. *Public health Nutr* 18, 3096-3107. 2015.

29. Moubrance J-C, Martins Ap B, Claro RM et al consumption of ultra- processed food and likely impact on Human health. Evidence from Canada. *Public Health Nutri* 16, 2240-2248.

30. Esmaillzadeh A, Kimiagar M, Mehratri yet al. Fruit and vegetable intakes, C-reactive protein ,and the metabolic syndrome. *Am J clin Nutr* 84, 1489-1497.

31. Sleeth ML. Thompson EL, Ford HE et al, Free fatty acid receptor 2 and nutrient sensing; a proposed role for fibre , fermentable carbohydrates and short chain fatty acids in a appetite regulation, *Nutr Res Rev* 23, 135-145 (2010).

32. Anderson Jw. Barid P, Davis RH et al . Health benefits of dietary fiber. *Nutr Rev* 67, 188-205, 2009.

33. Fernandez ML & west KL. Mechanism by which dietary fatty acids modulate plasma lipids. *J.Nutr* 135, 2075-2078, 2005.

34. Lopez S. Bermudez B, Ortega A et al. Effects of meals in a rich in either mono unsaturated or saturated fat on lipid concentrations and on insulin secretion and action in subjects with high fasting triglyceride concentrations. *AM J. Clin Nutr* 93, 494-499 . 2011.

35. Lara. NAsreddine et al. A minimally processed dietary pattern is associated with lower odds of metabolic syndrome among Lebanese adults. *Public Health Nutr* : 21(1), 160-171, 2017.

36. Scott M. Grundy , Does a diagnosis of metabolic syndrome have value in clinical practice? *Am. J. Clin Nutr.* 83;1248-51, 2006.