RISK FACTORS CONCERNING THE METABOLIC SYNDORME

***Moza Anca Cristina, Popa Loredana Mădălina, Chereji Anca, Popa Amorin Remus**

*****Spitalul Clinic Judeţean de Urgenţă Oradea, Str. Republicii nr.37, Oradea 410167, România, e-mail: anca.moza@yahoo.com

Abstract

Metabolic syndrome has grown in to a major public health problem globally. Somewhat more than one-third (35%) of adults in the USA might be considered as having the metabolic syndrome. This interprets to approximately 84 million US adults suffering by the syndrome (designed from the data provided since 2012 from the US Bureau of Census, with an adult inhabitant population of 239 million).

Key Words: risk factors, environmental, genetic, hormonal, BMI.

INTRODUCTION

Metabolic syndrome is a well-known risk factor for mutually type 2 diabetes as well as cardiovascular illness, and signifies a shared clinical disorder in states with a high occurrence of abdominal obesity. As a consequence, metabolic syndrome might be seen as a sentinel for the harmful effect of harmful diet in vulnerable persons, who might first manifest metabolic concerns or signs (such as visceral obesity, blood pressure, and dyslipidemia), and after an amplified risk of cancer. Agreed the essential complications to achieve long-term interventional nutritional trials for cancer avoidance, it appears doubtful that confirmation, trial-based dietetic recommendations will be obtainable in a little while (Adams S., 2012).

MATERIAL AND METHODS

Articles provided from The US Department of Health and Human Services (HHS), helped determine what the risk factors were for the metabolic syndrome, a number of studies which are present widely in this article and in the References section, gave the information required for the results and impressions presented below. Body conformation by anthropometry, blood pressure, lipid outline, glucose, insulin, food consumption and physical activity level were assessed in the research.

RESULTS AND DISCUSSION

Figure 1 shows a flow diagram of the procedure used to identify the relevant studies. Briefly, we identified the risk percentage potentially relevant articles on MetS. After examining 33 assembled articles, we identified the main elements that confer CVD incidence. No additional studies were identified via cross-referencing.

ENVIRONMENTAL FACTORS

Cigarette smoke and air pollution are the greatest noteworthy external foundations of oxidative stress. Epidemiological researches have confirmed a strong connotation among enlarged air pollution and human deaths and mortality. Making of ROS is the essential instrument which facilitates these damaging effects (Alberti K.G.M.M et al, 2009). Small-term exposure to city air fumes in healthy young adults lead to in augmented oxidative stress which is not limited inside the lungs (Allison D.B. et al, 2003). The central mediators of air pollution-resulting effects are perfumed hydrocarbon and metal-comprising inhalable nanoparticles that can enter the alveolar-septal blockade and therefore produce oxidative stress together via starting of alveolar macrophage and complete vascular oxidases counting the NAD (P) H, mitochondrial and also the xanthine oxidases (Barnett A.H. et al, 1981; Barrett-Connor E., KT. Khaw, 1989). Perfumed hydrocarbons produce ROS from the redox cycling of quinone-centered radicals, by complexion of metals ensuing in amplified electron transference and by reduction of antioxidants by responses amid quinones and thiol-containing combinations. Metals straight sustenance electron transference to produce oxidants as well as lessen ranks of antioxidants (Bergman R. N. et al, 2006). Adding to direct production of ROS, cellular reactions to oxidative stress subsequently nanoparticle contact subsidize to the global damage. Oxidative stress induces stimulation of proapoptotic signal transduction flows and discharge of inflammatory intermediaries, which eventually cause to cell death, mainly of endothelial cells. Endothelial cell injury and death is a crucial occurrence in the expansion and deterioration of CAD and many other vascular pathologies

Unquestionably a disproportion of energy consumption to energy outflow in a more obesogenic setting necessitating fewer physical activity is a proximal factor of the current growth in the metabolic syndrome worldwide. Positive energy inequality outcomes in weight increase; even in the very thin minor rises in weight are related with blood pressure, hyperglycemia, dyslipidemia (Bjorntorp P., 1991). Furthermore, condensed energy consumption and/or enlarged energy production have been revealed to develop metabolic profile in numerous trials (Bouchard C. et al, 1988;

Brody J.E., 2010). Yet, energy inequity is only one side of macro environmental modification to a more obesogenic background determined by socio-economic evolution. With growing wealth there are several other fluctuations which may influence the risk of the metabolic syndrome, such as superior accessibility of and access to an extensive variety of edibles, cigarettes and liquor. Furthermore, all of these modifications take place inside a social setting whose configuration may influence the level of risk and which individuals are at utmost risk.

Daily life variables take in consideration alcohol drinking, cigarette smoking, and physical activity. Alcohol consumption grade was made following strategies from the US Department of Health and Human Services (HHS) (Carey D. G. et al, 1996) with three-level classifications of nondrinkers (i.e., by no means drank alcohol throughout their lifespan or had abandoned alcohol consumption), temperate drinker (i.e., having just only two drinks per day for men and just one drink per day for females), and weighty drinkers (i.e., having over two drinks per day for males and over one drink a day for females). Cigarette smoking was likewise classified into a three-rank variable as antismoker (i.e., those who by no means smoke cigarettes throughout their lifespan or who had abandoned cigarette smoking), light smokers (i.e., people who smoked 1–10 cigarettes everyday), and heavy smokers (i.e., those who smoked over 10 cigarettes per day).

Physical activity was well-defined as a four-rank definite variable established on strategies by HHS (Chuang K. J. et al, 2007) counting inactivity (i.e., no physical activity afar from standard activities of everyday living), small activity (i.e., fewer than 150 minutes of modest concentration physical activity per week, or the corresponding total (75 minutes, or 1 hour and 15 minutes) of dynamic concentration activity), average activity (i.e., reasonable concentration activity of 150 minutes to 300, or 75 to 150 minutes of vital amount physical activity every week), and high activity (i.e., over the correspondent of 300 minutes of modest amount physical activity per week).

Married or not married, earnings, and scholastic ranks were also incorporated as socio-demographic variables by means of accessible informations components from the records.

The metabolic syndrome is better explained in the following figure, by which, we will understand how environmental factors and MetS prevalence in individuals:

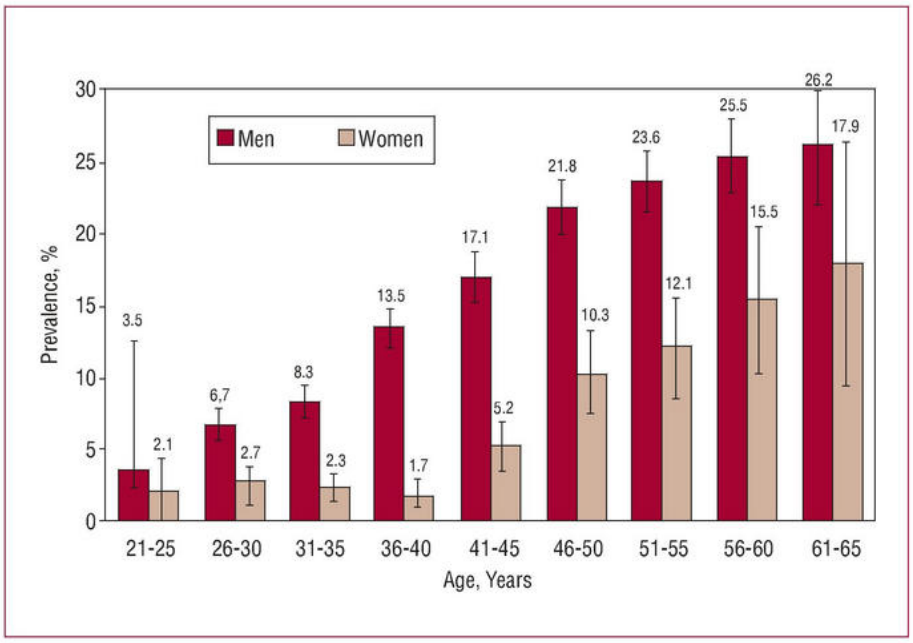

Fig.1. Prevalence of Metabolic Syndrome

GENETIC FACTORS

Heritability is a degree for measuring genetic elements in ailments. It is well-defined as the section of distinction in accountability through the population due to inherited factors. Heritability can be valued via either identical researches, which look for better concordance amongst monozygotic (MZ) twin couples than amongst dizygotic (DZ) alike pairs, or family researches, which inspect the amount of resemblance within families as opposed to between families. Accumulated proof suggests that metabolic syndrome most probable outcomes from the interaction between more than a few genes and a wealthy surroundings. Though the approximation on heritability of metabolic syndrome has not been stated, it is without a doubt that all elements of the syndrome are intensely inherited (Coté M. et al, 2007). Many twin and family researches have revealed noteworthy genetic influences to hypertension. Estimations of inherited variety from 22% to 62% for those with systolic BP and from 38% to 63% for those with diastolic BP. The involvement of genetics to the evolution of diabetes is confirmed by the high occurrence of diabetes amid first-degree blood relatives of type 2 diabetic individuals and the great concordance in matching twins (Després J.P., 2012). It is assessed that the odds that one twin of a MZ pair is diabetic agreed that the other one has diabetes is approximately 50%; the projected chance in DZ duos is of 37%. Family cluster researches have demonstrated that 45% of first-grade blood relatives

of individuals that suffer of type 2 diabetes are likely to be insulin resistant in comparison with 20% of patients that do not have a family past of diabetes (Eibers J.M. et al, 2003). For abdominal obesity, a solid affiliation has been detected concerning the biologic parents-child couple (Ezzati M., E. Riboli, 2013,) and identical twins (Ghio A.J. et al, 2012) in concernment to BMI. It is likely that genetic elements elucidate almost 40% of the modification in body fat (Going S.B. et al, 2011) and rises to 70% of alteration in abdominal obesity (Goran M.I., 1998). In adding, consistency proportions for glucose intolerance, global obesity, and low HDL-cholesterol are considerably developed amid MZ rather than DZ twins (Ghio A.J. et al, 2012).

Mutual genetic alternatives in an amount of genes could enhance vulnerability to metabolic syndrome. These genetic versions could turn in concern with other gene versions and an amount of environmental elements in illness development. Yet, proof of predisposition genes of metabolic syndrome and their useful variants as well as the allied pathophysiological devices are of paramount significance, for the reason that it might permit researchers to plan anticipatory tactics and targeted managements. Though the noteworthy heritability of the distinct elements of the metabolic syndrome has been already acknowledged and considerable improvement in comprehending the physiology of this syndrome has been accomplished, the fundamental genetic root and the molecular mechanisms continue to be ambiguous.

HORMONAL FACTORS - BMI MEASUREMENT

Body size and consistent Body Mass Index verges problems concerning overweight and abdominal obesity (Groop L. et al, 1996). Regardless of its shared utilization amid clinicians, scientists, and the worldly public there is been constant disapproval of Body Mass Index as a scale of measuring abdominal obesity and as a forecaster of surplus body fat associated with health risk (Groop L., M. Orho-Melander, 2001, Healy M., 2012(1), Healy M., 2012(2)). The common public has come to be attentive of this debate as the general press has exposed these denunciations of Body Mass Index (Hu F.B. et al, 2000, Jaffrin M.Y., 2009, Katzmarzyk P.T. et al., 2010; Meckling K.A, R.A. Sherfey, 2007; Mehta S.R. et al, 2012). Supposed limitations of the BMI measure contain its incapacity to distinguish between thin mass and fat mass and to seize the anatomical circulation of adipose tissue. Researchers have revealed a solid link between high percent body fat and an amplified danger of chronic illnesses such as blood pressure, dyslipidemia, diabetes mellitus, and coronary heart disease (Mills N.L. et al, 2009; Poulsen P. et al, 2001; Romero-Corral A. et al, 2008, Shai I. et al, 2008). Although BMI and the mass of fatness are

extremely linked at high Body Mass Indexes, these processes are not as much associated in standard weight varieties (Sharma A.M., V. T. Chetty, 2005, US Department of Agriculture, Health and Human Services, 2005).

Numerous theories have been offered in the last 2 decades to clarify how visceral fat links to a high risk of CV cases, such as an triggered hypothalamic-pituitary adrenal axis, the contribution of gonadal steroids, stimulus of the endocannabinoid grid and diverse environmental elements (i.e., smoking) and ethnicity (white individuals and the Asians are more probable to have an intensification in visceral adipose tissue) (Sharma A.M., V. T. Chetty, 2005). The solider theory nevertheless is the so-called "lipid excess-ectopic fat pattern"; together amplified in caloric consumption and sedentarism create a high-energy equilibrium.

In circumstances of insulinresistance or hereditary predisposition to visceral obesity, the dysfunctional hypodermic adipose tissue will be incapable to stock the energy excess. The afflicted free fatty acid and the disrupted deliverance of adipokines create a surplus of triglycerides that will be kept in non-predictable organs, like the liver, heart, pancreas, kidney, skeletal muscle and mainly visceral adipose tissue, an occurrence defined as ectopic fat storage (US Department of Agriculture, Health and Human Services, 2005). This condition will create all the alterations that describe the cardiometabolic syndrome, with the consequential enlarged CV risk profile as described before. Moreover the visceral fat, ectopic liver fat is believed to be the most significant ectopic organ where the fat can be stored in a disagreeable way. This could decrease the hepatic removal of insulin, creating a raised hepatic glucose production, glucose intolerance and quite too much atherogenic lipoproteins, conducting it by itself to an extraordinary cardiometabolic risk profile. Lately, as all the ectopic fat storages are correlated, it has been proposed that they can be separated into two unalike groups: those with mainly systemic results (visceral adipose tissue, liver fat and skeletal muscle intracellular lipids) and those with local results

CONCLUSION

Obesity and metabolic syndrome are characterized by high cardiovascular risk and increased prevalence of new-onset diabetes. Lifestyle habit changes based on a multidisciplinary approach are required for effective and persistent weight loss and should include a meaningful reduction of calorie intake and increase in physical activity.

However, control of body weight is difficult to achieve only with an adequate lifestyle. Thus, pharmacological treatment is helpful, and new drugs are welcomed to improve and maintain body weight

Yet, action must be taken to avoid and/or regulate the epidemic of metabolic syndrome. Dietary approaches that decrease the occurrence of metabolic syndrome have the potential to diminish the forthcoming risk of cancer. For example, adherence to Mediterranean nutritional patterns is related with lower occurrence and evolution of metabolic syndrome.

REFERENCES

1. Adams S., 2012, "Forget BMI, just measure your waist and height" say scientists, The Telegraph.
2. Alberti K.G.M.M, R.H. Eckel, S.M. Grundy, P.Z. Zimmet, J.I. Cleeman, K.A. Donato, J.C. Fruchart, W.P. James, C.M. Loria, S.C. Smith Jr., 2009, International Diabetes Federation Task Force on Epidemiology and Prevention, National Heart, Lung, and Blood Institute, American Heart Association, World Heart Federation, International Atherosclerosis Society, International Association for the Study of Obesity, Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention, National Heart, Lung, and Blood Institute, American Heart Association, World Heart Federation, International Atherosclerosis Society, International Association for the Study of Obesity, Circulation 120, 1640–1645.
3. Allison D.B., S.K. Zhu, M. Plankey, MS. Faith, M. Heo, 2002, Differential associations of body mass index and adiposity with allcause mortality among men in the first and second National Health and Nutrition Examination Surveys, NHANES I and NHANES II Journal, follow-up studies. Int J Obes Relat Metab Disord, 26(3):410—6. Mar.
4. Barnett A.H., C. Eff, R. D. Leslie, DA. Pyke, 1981, Diabetes in identical twins. A study of 200 pairs, Diabetologia, 20:87–93.
5. Barrett-Connor E., KT. Khaw, 1989, Cigarette smoking and increased central adiposity, Ann Intern Med Publish, 111:783–7.
6. Bergman R. N, S. P. Kim, K. J. Catalano, IR. Hsu, JD. Chiu, M. Kabir, et al., 2006, Why visceral fat is bad: mechanisms of the metabolic syndrome, Obesity (Silver Spring); 14 suppl 1:16S 9S.
7. Bjorntorp P., 1991, Metabolic implications of body fat distribution, Diabetes Care; 14:1132–43.
8. Bouchard C., L. Perusse, C. Leblanc, 1988, Inheritance of the amount and distribution of human body fat., Int J Obes ; 12:205–15.
9. Brody J.E., 2010, Weight index doesn't tell the whole truth, The New York Times
10. Carey D. G., T.V. Nguyen, L. V. Campbell, 1996, Genetic influences on central abdominal fat: a twin study, Int J Obes Relat Metab Disord; 20:722–6.
11. Chuang K. J., C. C. Chan, T. C. Su, C.T. Lee, C.S. Tang, 2007, The effect of urban air pollution on inflammation, oxidative stress, coagulation, and autonomic dysfunction in young adults, American Journal of Respiratory and Critical Care Medicine ; 176(4):370–376.
12. Coté M., I. Matias, I. Lemieux, S. Petrosino, N. Alméras, J. P. Després J.P., 2007, Circulating endocannabinoid levels, abdominal adiposity and related cardiometabolic risk factors in obese men. Int J Obes (Lond); 31:692–9.
13. Després J.P., 2012, Body fat distribution and risk of cardiovascular disease: an update. Circulation; 126:1301–13. Updated review of the critical importance of the body fat distribution and its relationship with cardiovascular risk.

14. Eibers J.M., E.J. Giltay, T. Teerlink, PG. Scheffer, H. Asscheman, J.C. Seidell, 2003, Effects of sex steroids on components of the insulin resistance syndrome in transsexual subjects, Clin Endocrinol (Oxf); 58:562–71.
15. Ezzati M., E. Riboli, 2013, Behavioral and dietary risk factors for noncommunicable diseases. N. Engl. J. Med. 369, 954–964.
16. Ghio A.J., M.S. Carraway, M.C. Madden, 2012, Composition of air pollution particles and oxidative stress in cells, tissues, and living systems, Journal of Toxicology and Environmental Health Part B; 15(1):1–21.
17. Going S.B., T.G. Lohman, E.C. Cussler, D.P. Williams, J.A. Morrison, P. S. Horn, 2011, Percent body fat and chronic disease risk factors in U.S. children and youth, Am J Prev Med;41(4 (Suppl. 2)):S77—86.
18. Goran M.I., 1998, Measurement issues related to studies of childhood obesity: assessment of body composition, body fat distribution, physical activity, and food intake, Pediatrics; 101 (3 Pt 2):505—18.
19. Groop L., C. Forsblom, M. Lehtovirta, 1996, Metabolic consequences of a family history of NIDDM (the Botnia study): evidence for sex-specific parental effects, Diabetes Publish; 45:1585–93.
20. Groop L., M. Orho-Melander, 2001, The dysmetabolic syndrome, J Intern Med; 250:105–20.
21. Healy M., 2012, Move over BMI: There's a new way to measure fat, Los Angeles Times.
22. Healy M., 2012, We may be fatter than we think, researchers report. Los Angeles Times.
23. Hu F.B., B. Wang, C. Chen , Y. Jin, J. Yang, M.J. Stampfer, 2000, Body mass index and cardiovascular risk factors in a rural Chinese population, Am J Epidemiolgy; 151(1): 88-97.
24. Jaffrin M.Y., 2009, Body composition determination by bioimpedance: an update", Curr Opin Clin Nutr Metab Care; 12(19494768):482—6.
25. Katzmarzyk P.T., G.A. Bray, F. L Greenway, W. D. Johnson, R. L. Jr. Newton, E. Ravussin, 2010, Racial differences in abdominal depotspecific adiposity in white and African American adults. Am J Clin Nutr. ; 91:7–15.
26. Meckling K.A, R.A. Sherfey, 2007, A randomized trial of a hypocaloric high-protein diet, with and without exercise, on weight loss, fitness, and markers of the Metabolic Syndrome in overweight and obese women, Appl Physiol Nutr Metab; 32(4): 743-752.
27. Mehta S.R., I. F. Godsland, E. L. Thomas, D.V. Pavitt, S. X. Morin, J. D. Bell, 2012, Intrahepatic insulin exposure, intrahepatocellular lipid and regional body fat in nonalcoholic fatty liver disease, J Clin Endocrinol Metab.; 97:2151–9.
28. Mills N.L., K. Donaldson, P. W. Hadoke, 2009, Adverse cardiovascular effects of air pollution, Nature Clinical Practice Cardiovascular Medicine. ; 6(1):36–44.
29. Poulsen P., A. Vaag, K. Kyvik, H. Beck-Nielsen, 2001, Genetic versus environmental aetiology of the metabolic syndrome among male and female twins, Diabetologia; 44:537–43.
30. Romero-Corral A, V. K. Somers, J. Sierra-Johnson, 2008, Accuracy of body mass index in diagnosing obesity in the adult general population, Int J Obes (Lond); 32(18283284):959—66.
31. Shai I, D. Schwarzfuchs, Y. Henkin, D. R. Shahar, S. Witkow, Greenberg et al. 2008, Weight loss with a low-carbohydrate, Mediterranean, or low-fat diet, N Engl J Med ; 359(3): 229-241.

32. Sharma A.M., V. T. Chetty, 2005, Obesity, hypertension and insulin resistance.” Acta Diabetol; 42(Suppl. 1):S3—8.
33. US Department of Agriculture, United States Department of Health, and Human Services, 2005, Alcoholic beverages, Dietary Guidelines for Americans, chapter 9, pp. 43–46, Government Printing Office, Washington, DC, USA.