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Editorial Open Access

Metabolic Syndrome: The Genetic Aspect

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Editorial

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We have seen increased interest in recent years in the study of metabolic syndrome. Metabolic syndrome is the fastest growing health problem worldwide. Metabolic syndrome is a name given for a group of risk factors that collectively increase the risk for atherosclerotic cardiovascular disease (ASCVD), stroke, and type 2 diabetes [3-5]. It is also known as syndrome X, the deadly quartet, and the insulin resistance syndrome. According to a recent estimate it affects one in five people in the United States and its incidence increases with advancing age. About 25% of the population of US suffers from metabolic syndrome [6]. Two most important risk factors for metabolic syndrome are obesity and insulin resistance. As a result of insulin resistance sugar and fat levels rise in the affected individual. Other risk factors include genetics, fluctuating hormonal levels, and lack of physical activity. The most widely recognized of the metabolic risk factors are atherogenic dyslipidemia (AD), elevated blood pressure, and elevated plasma glucose. AD is characterized by three lipid abnormalities: elevated serum triglyceride, small low density lipoprotein (LDL) particles, and reduced high density lipoprotein (HDL) cholesterol levels [7]. This lipid trinity is commonly seen in persons with premature coronary heart disease [8]. Individuals with AD are obese, insulin resistant, and physically inactive.

Metabolic syndrome is determined by the interaction of various genetic and environmental factors. Recent research suggests that metabolic syndrome has an element of heritability, indicating a genetic basis to it [9]. The incidence of metabolic syndrome differs among various ethnic groups, with the highest rate in Mexican-American women [10]. Although African Americans have a higher prevalence of obesity and hypertension compared to whites, they still have a lower incidence of metabolic syndrome. These ethnic differences strongly indicate a genetic component in the pathogenesis of metabolic syndrome. High clustering of factors such as hypertension, diabetes, and obesity in family and twin studies also underscores the role of genetics in metabolic syndrome [11].

Individuals with certain rare single-gene disorders show group of abnormalities commonly seen in the metabolic syndrome. Studies indicate that development of metabolic syndrome is connected to common genetic variants, although the associations were weak and replications poor. It was suggested that thrifty genes, which maintain optimal levels of energy during periods of fasting, could be responsible for metabolic syndrome [12]. Common variants in a number of candidate genes influencing fat and glucose metabolism together with various environmental factors can increase susceptibility to the syndrome. Among these, the genes for β3-adrenergic receptor, hormone-sensitive lipase, lipoprotein lipase, IRS-1, PC-1, skeletal muscle glycogen synthase, etc. appear to increase the risk of the metabolic syndrome. Candidate gene studies have identified linkage between metabolic syndrome and a number of genes, such as PPARγ, adiponectin, CD36, and β receptors [13]. More research efforts are needed to establish the genetic basis of metabolic syndrome. Progress in understanding the genetic basis of metabolic syndrome should occur as soon as a consensus is reached on the true nature of metabolic syndrome, its components and diagnostic criteria.

There exists a correlation between obesity and inflammatory markers, such as C-reactive protein (CRP), which has been associated with increased risk of cardiovascular disease (CVD) [14]. Levels of CRP are influenced by different factors such as obesity, smoking, alcohol, physical activity, and genetics [15]. CRP through systemic inflammation could well be the connecting link between obesity, insulin resistance, and CVD. Future research should be directed to answer whether CRP plays any major biological role in metabolic syndrome or is just a marker. Focus should be on systems-based approaches that combine genomic, molecular and physiological data to understand the complexity of metabolic syndrome. Some studies have suggested that the disruption of the circadian rhythm (chronodisruption) may lead to metabolic syndrome [16]. Animal models such as mice have revealed that disruption of genes regulating circadian clock results in a phenotype similar to metabolic syndrome. Interestingly, studies in humans have shown that genes influencing circadian rhythm are expressed in adipose tissue, and that their expression levels and genetic variants correlate with different components of the metabolic syndrome. It would be intriguing to study the relationship between polymorphisms in different circadian genes and traits associated with metabolic syndrome. I am sure the journal "Endocrinology and Metabolic Syndrome" from OMICS Group will make a positive impact in this direction.

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