Integrative Factors in Hereditary Nutritional Interactions in Single Gene Abnormalities and Polygenic Defects

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Therapy

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The 'genetic variation and dietary response' focuses on genetics and nutrition, and their interaction in the development of chronic diseases, and was composed to consider important advances in molecular biology as they impact genetics, nutrition, and public health. Coronary artery disease, hypertension, diabetes, cancer, and other chronic diseases tend to cluster in families, with relatives being at a much higher risk than the general population. Families share both genes and environments, and similarities can occur as a result of either. Much research has been conducted to define each's contribution and interaction in the development of the individual. Heritability, in broad terms, is the proportion of total variance that can be explained by genes 50% of the variance in plasma cholesterol concentration, 30%-50% of the variance in LDL particle size, and 30%-60% of the variance in blood pressure are genetically determined; 15% to 50% of the variance in fibrinogen, an independent risk factor for coronary heart disease, is genetically determined, while 51% is the figure among the Hawaiian population, indicating significant differences between populations. The variation in bone density is determined by genetics. Heritability calculations are only relevant to the population and environment from which the information is gathered. Heritability may differ between populations if the prevalence of the genes affecting the disease entity under consideration differs. In addition, in vitro studies of atherogenic properties of LDL sub fractions and the effects of lipid-lowering therapies on LDL sub fractions, including diet and exercise, hormonal factors, and pharmacologic agents, should not be copied by populations estimates of LDL particle size heritability range from 30%-50%, indicating the importance of genetic and environmental influences. Aside from age and gender, abdominal adiposity AIDS-related hypertriglyceridemia all have an impact on LDL particle size and density distribution. Dietary fat and carbohydrate intake have a strong influence on the expression of the small, dense LDL phenotype and contribute to differences in LDL particle size distribution observed among individuals and population groups. The plasma lipoprotein profile is characterized by a predominance of small,

dense lipoproteins. LDL particles, specifically LDL3, are linked to a threefold increase in the risk of coronary artery disease.

DESCRIPTION

Mipsin acylation stimulating protein pathway and micro environmental metabolic regulation and to define micro environmental metabolic regulation as the tight linkage of chylomicron triglyceride hydrolysis in the capillary space to adipocyte triglyceride synthesis in the sub endothelial space a variety of clinical disorders of lipoprotein and adipose tissue metabolism result from dysfunction or dysregulation of this pathway. Dysfunction of the pathway results in dyslipoproteinemia, hyperapoB, whereas hyper function is associated with gynoid obesity, but whether this is a primary abnormality or a secondary adaptation is unknown and the pathophysiology of dyslipoproteinemia in android obesity is similar to that of hyperapoB due to a faulty Acylation-Stimulating Protein (ASP) response. Furthermore, they hypothesize that ASP pathway dysfunction may play a role in the pathogenesis of several dyslipoproteinemia associated with coronary heart disease. Hypertriglyceridemia with normal apoB is not linked to an increased risk of cardiovascular disease, whereas hypertriglyceridemia with elevated apoB is. This has implications for treatment because fibrates that lower triglycerides significantly reduce apoB only modestly, whereas HMG CoA reductase inhibitors do the opposite.

CONCLUSION

Methods of controlling lipemic responses to dietary lipids provides a review of the metabolic and molecular mechanisms underlying high and low plasma cholesterol responses to dietary cholesterol and fat in human studies and animal models, as well as an assessment of these studies to see if any markers identified in animal models can be applied to humans. High LDL apoB production is the metabolic variable most frequently associated with a high cholesterolemic response to dietary lipids in humans. Post-translational regulation of LDL apoB secretion

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from the liver is influenced by a variety of factors affecting hepatic cholesterol metabolism.