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Early-life undernutrition and adult health: Lessons from manmade famines

Alexander Vaiserman
Institute of Gerontology, Ukraine

Early-life malnutrition is important determinant of metabolic disorders and associated cardiovascular disease in later life. To examine whether a link exists between early-life exposure to famine and adult health, we determine the risk of developing type 2 diabetes (T2D) in Ukraine residents born before, during and after the famine of 1933. The sample studied consisted of 28,358 T2D patients born in 1930-1938 and living in Ukraine regions that suffered significant demographic losses due to famine. Reference populations were based on the Ukraine census 2001 (n=2,153,335). It was approximately 1.5-fold increase in the risk of developing T2D in both men and women who were born in the first half of the 1934 year as compared to the individuals who were born in the pre-famine and post-famine cohorts. These differences are highly significant compared to the appropriate reference cohorts born in 1938 [odds ratios are 1, 48 and 1, 52 for men and women respectively]. Remarkably, those individuals who were born in the first half of 1934 and who have higher risk of developing T2D were exposed to the peak of the famine periconceptionally. The findings obtained in our research are similar to those found in studying other famine episodes such as Dutch famine of 1944-45 and suggest that periconceptual exposure to the famine may result in induction of persistent epigenetic changes that predispose to metabolic disorders in the later life.

vaiserman23@gmail.com

Determinants of LDL-C in the South African PURE population

Tertia Van Zyl
North-West University, South Africa

Background: Elevated LDL-C levels promote the development of atherosclerosis and are a major risk factor in the progression of coronary heart disease. Dietary factors, weight, physical activity, age, gender and genetics are important factors that affect plasma LDL-C levels. The aim of the study was to investigate which of these factors best predict the variance in LDL-C levels in a black South African population.

Methods: The PCSK9 and LDLR genes in 1530 volunteers, aged 35 to 60 years, of the South African PURE study population were screened for 52 variants. From these SNPs we determined a genetic risk score and haplotypes. Validated quantified food frequency questionnaires were used to determine the dietary intakes of the volunteers. Spearman's correlations were used to identify which factors correlated best with LDL-C levels. Separate linear regression models were used to determine the predictive value of each of the variables.

Results: The GRS, selected SNPs and haplotypes explained 1.4%, 3.4% and 2.7% of the variance respectively in plasma LDL-C. BMI was the factor with the largest predictive value and could explain 6.6% of the variance in LDL-C levels. There were no significant correlation between LDL-C levels and dietary intakes.

Conclusion: From these results we can conclude that BMI is a factor with a large impact on determining the variance in mean LDL-C levels in this study population. Genetic results showed that rare variants with greater effect on protein function have a better predictive value than the more common variants. The contribution of genetics in predicting LDL-C levels can be strengthened by including more genes as lifestyle factors contributed more towards the prediction of LDL-C levels in this study. The importance of the genetic variants in predicting LDL-C levels was less than the lifestyle factors used in this study.

tertia.vanzyl@nwu.ac.za