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11TH OBESITY AND ENDOCRINOLOGY CONGRESS

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Nutritional genetics and epigenetics of obesity and type-2 diabetes

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Obesity and sequel Type-2 Diabetes are complex disorders that constitute major public health problems. The evidence for familial aggregation of both T2D and obesity is substantial. To date, more than 150 genetic loci are associated with the development of monogenic, syndromic or multifactorial forms of T2D or obesity, many within lipid and carbohydrate metabolism pathways. SNPs located in or near FTO, MC4R, MC3R, POMC, LEP, LEPR, PLIN1, APOA5, LIPC, FABP2, INSIG2, IRS1, GIPR, ADBR2, ADRB3, UCP1, RETN, ADIPOQ, IL6, PPARG, TCF7L2 and CLOCK, among others, are implicated in both diabetes and obesity gene networks, pleomorphic with nutritional and metabolic traits. A personalized nutritional approach based not only on phenotypic traits but also on genetic make-up, may help to control body weight, sugar metabolism and obesity. Recent advances in nutrigenetics, bioinformatics and genome-wide association metabolomics studies are set to unleash a revolution in personalized nutrition. In this symposium, we discuss the evidence concerning the genetic and epigenetic contribution to individual risk of T2D and obesity and explore the potential role of nutritional and environmental mechanisms for precision treatment. We also explain how genetics, epigenetics and environment are likely to interact to define the individual risk of disease, through analyzing the results of a number of recent human clinical trial studies that use genetics to personalize treatment plans for obesity, metabolic syndrome and diabetes management.

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