Evidence for epigenetic changes prenatal as a cause of clinical obesity later in life

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The global prevalence of obesity is predicted to increase between 30 and 80% by 2030, with the greatest disease burden in developing counties such as Iran. Obesity is routinely caused by lifestyle choices in which a poor quality yet high calorie diet and low levels of activity play major roles. The role of genetic variation in determining such differential susceptibility is unclear. As with other complex disease traits, while some candidate genes have been reported, the contribution of individual polymorphisms to pathogenesis may be small. There is increasing evidence that the prenatal environment acts through developmental plasticity which involves induced changes in the epigenetic regulation of non-imprinted genes and underlies differential risk of obesity and associated conditions including cardiometabolic disease and some cancers. This presentation will focus on the role of altered epigenetic processes in non-imprinted genes in humans and in animal models in determining differential susceptibility to obesity. There is substantial evidence in animal models that the early environment determines future disease risk and that induced changes in the epigenome are part of the causal process. However, research to apply the findings of animal studies to understand the role of epigenetics in human obesity and related co-morbidities is still in its early stages. Nevertheless, identification of periods of epigenetic plasticity after birth and demonstration that the epigenome can be manipulated by relatively simple interventions strongly support the feasibility of future therapeutic strategies to reverse the adverse effects of prenatal constraint on risk of disease throughout the life course.