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Metabesity-journey starts before birth?

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INTRODUCTION

Metabesity directly means metabolic syndrome along with obesity, diabetes, cardiovascular diseases and neurodegenerative disorders, and accelerated aging with high risk of cancers.[1] Prolonged inactivity, sedentary lifestyle and unbalanced nutrition predispose to weight gain and obesity, also termed as metabesity (metabolic obesity).

The pathogenesis of metabesity occurs due to inflammatory and oxidative damage, insensitivity to regulators such as insulin or leptin, leading to cell death.[2] Inflammatory responses rising from diabetes and obesity affects the astrocytes and the regulation of metabolic hormones. Etiology lies in complex relationships among genes and the obesogenic environment.[3]

Among the studies regarding how metabolic syndrome occurs over the last few decades in different populations Barker hypothesis[4] proposed that the fetal environment may have an effect on the development of noncommunicable disease in adulthood and also maternal undernutrition during pregnancy resulted in fetal programming that altered the structure, function, homeostatic pathways, and/or metabolism of the developing offspring, predisposing them to an increased risk of disease later in life. Dutch Hunger Winter have state that time during gestation maternal nutrition has differing and lasting impacts on later life disease in their offspring.[5]

When a woman who is obese becomes pregnant, the changes in the in utero environment increase the risk of offspring developing childhood obesity[6]and metabolic syndrome in adulthood. Because of the reduced insulin sensitivity in obese compared with lean women, there is an increase in the insulin response in early pregnancy which affects early placental growth and gene expression leading to release of placental factors that decrease insulin sensitivity in maternal tissue (skeletal muscle, liver, and adipose tissue), thereby resulting in increased nutrient availability for feto-placental growth. Increased availability of nutrients such as glucose and lipids thereby contributes to fetal adiposity, which becomes manifest only in late gestation.

Drugs that decrease body weight are potentially of use as these can help to reduce the levels of adipose tissue macrophages which have a key role in obesity-related inflammation eg; metformin decreases the serum level of the pro-inflammatory cytokines like TNF-alpha and IL-6 and also lowers expression of the M1 macrophage markers CD11c and MCP-1 in adipose tissues. Alterations in macrophage polarization improve obesity-related inflammation.Combat metabesity by altering the composition of the gut microbiome.[3]

So metabesity is now considered to be one of the major public health problems worldwide with emphasis shifting on a more holistic approach of management. Future research into programmed disease risk should clearly establish affirmative interventions that have the capacity to break the cycle of obesity

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References

1. Alberti KGM, Zimmet P, Shaw J, IDF Epidemiology Task Force Consensus Group. The metabolic syndrome—a new worldwide definition. Lancet. 2005;366:1059-62.
2. Third Report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report. Circulation. 2002;106:3143-421.
3. Raza SA, Ali SS, Ali KB, Ali CA et al. Metabesity: expert panel recommendation for taking up the challenge by a multidisciplinary approach. JPMA. 2020 Mar 28;2020.
4. Fall C, Osmond C. Commentary: The Developmental Origins of Health and Disease: An Appreciation of the Life and Work of Professor David J.P. Barker, 1938-2013. Int J Epidemiol. Oxford University Press; 2013;42:1231-2.
5. Roseboom T, de Rooij S, Painter R. The Dutch famine and its long-term consequences for adult health. Early Hum Dev. 2006;82:485-91.
6. Perng W, Oken E, Dabelea D. Developmental overnutrition and obesity and type 2 diabetes in offspring. Diabetologia. 2019 Oct.