Opinion

The influence of metabolic programming and obesogenic factors on the global rise of obesity

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Obesity, a chronic and recurrent condition, is intrinsically linked to metabolic syndrome-a condition in which factors such as abdominal fat accumulation, elevated glucose levels, high blood pressure, and dyslipidemia increase the risk of type 2 diabetes and cardiovascular diseases.[1] This disorder, influenced by various risk factors, extends beyond physical concerns, manifesting also in mental and emotional impacts known as social stigmas, meaning that the complexity of obesity is revealed in its multifactorial interaction. This disease has become a growing global concern, with incidence rates projected to rise significantly worldwide by 2035, as indicated by the World Obesity Atlas.[2] It is estimated that up to one-third of children and adolescents in Brazil will face the challenge of obesity during this period.

While poor diet and lack of physical activity are recognized as primary determinants of this issue, emerging evidence suggests the potential impact of chemical substances known as "obesogens" related to endocrine disruptors.[3] According to a study published in the journal Obstetrics & Gynecology in 2013,[4] discussing exposure to toxic environmental agents, it was observed that there is a need to identify and minimize the potential harm caused by these substances-a constant concern for healthcare professionals, especially in the context of pregnant women and metabolic programming.

Although it is a multifactorial disease, genetics also plays a crucial role in the development of obesity, influencing both appetite and eating behavior, as evidenced by Francischi and collaborators.[5] Body weight regulation, a delicate and complex interaction between hormones, neuropeptides, and the central nervous system, reveals that mutations in related genes may contribute to obesity, although they cannot fully explain all of its phenomena.[6] Metabolic programming defines a process in which a stimulus or insult during a critical period of life called a critical window-such as during fetal and/or postnatal development-can generate lasting or permanent consequences throughout life and into adulthood in adult animals.[7,8] For example, McCance in 1962,[9] demonstrated through his pioneering studies on metabolic programming in rodents that by adjusting litter size, the amount of food consumed during the postnatal period generated long-term consequences on growth. In this way, hypotheses on early adaptive response, origin, and fetal programming related to adult metabolic diseases are based on the observation that environmental changes during the prenatal and/or postnatal period can alter intrauterine development, leading to obesity and metabolic and histological complications at cardiovascular and hepatic levels, for instance.[10–12] This means that during these critical periods early in life, the body has the ability to respond to environmental situations considered inadequate for normal development.

These responses, carried out through adaptations at cellular, molecular, and biochemical levels, temporarily adjust systems to physiological demands. However, in the long term, such adjustments result in impairments in the functions of different tissues and peripheral organs.[8,13–16] Various experimental studies related to obesity utilize nutritional modification in maternal[17] or offspring[18–20] diets as a methodology to analyze the long-term effects of nutritional changes early in life on the development and consequences of weight gain, diabetes, cardiovascular diseases, liver complications, and obesity in adulthood. Moreover, significant experimental results have been achieved by generating metabolic reprogramming in adult mice through the experimental manipulation of maternal milk availability in the postnatal period by reducing litter size.[8,12–14,21,22] Thus, obesity is a multifaceted condition influenced by genetic, environmental, and metabolic factors that go beyond physical health, as mental and emotional well-being is also directly affected. The relationships between diet, genetic predispositions, and environmental exposures to both natural and artificial endocrine disruptors early in life contribute to the global rise in obesity rates, especially among populations with high consumption of processed and ultra-processed foods.[23] Intervention strategies should consider preventive and corrective actions that address these varied influences.

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Conflicts of interest

The authors declared that there are no conflicts of interest.

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