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Introduction

Dietary therapies generally pose little danger during pregnancy and might be viewed as an e ective therapeutic strategy [1]. Every person's lifelong health depends critically on the perinatal period [2]. Unfavourable prenatal conditions include elements that increase the likelihood of later-life chronic illness [3]. Placental dysfunction and poor prenatal outcomes have all been associated with, for example, diabetes, hypertension, stroke, and coronary artery disease [4]. e "major obstetrical syndromes," which include preeclampsia, foetal growth restriction, and premature labour, have common aetiologies [5]. ese disorders all have a protracted subclinical phase that only manifests when pregnancy reaches the point when the body's defence mechanisms are no longer able to support them [6]. ey are all highly correlated with the growth and functional development of the placenta [7]. A window of opportunity for potential actions to prevent the later emergence of overt symptoms exists during this subclinical stage [8].

e word the term "great obstetrical syndromes" was initially used to refer to conditions that a ect pregnancy and include a placental component to their pathogenesis [9]. Preterm labour and premature membrane rupture, preeclampsia, spontaneous miscarriage, stillbirth, and abnormal foetal growth are all referred to as GOS. Around 15% of all pregnancies are complicated by GOS, most of them with a signi cant recurrence risk. GOS is continuously increasing globally [10].

Discussion

e main idea is that these aetiologies are caused by things that happen during foetal development that a ect how nutrients, oxygen, waste products, and toxins are exchanged between the mother and the foetus. ese things start subclinical pathology that develops into clinical manifestation over the course of pregnancy. As a result of several exposome variables, these occurrences involve exposure to endogenous metabolites and exogenous nutrients. In order to de ne the exposome, all internal, non-genetic elements that a ect a person's health during the course of their life, especially during pregnancy. ese determinants may be split into three categories: internal (hormones, in ammation, and oxidative stress), speci c external (infectious agents, food, and lifestyle), and general external (education, socioeconomic position, and mental load). e growing baby is exposed to a variety of endo- and echo-exposome variables throughout pregnancy, most notably oxidative stress, nutrition, and in ammation. Gestational diabetes mellitus is one example of a pregnancy problem when the exposome is active. GDM is a metabolic condition that manifests as

oxidative stress and oxidative stress feeds an ongoing in ammatory response; the two concepts are tightly connected. Increased oxidative stress and a greater risk of unfavourable pregnancy outcomes may be the results of immunological responses triggered by in ammation that shi immune tolerance in the direction of immune e ector activation. It is still unknown how exactly oxidative stress and in ammatory processes contribute to the beginning and development of pregnancy problems. However, these mechanisms' contribution to the disease of It is important to consider oxidative stress as an endogenous component that is a part of endo-exposome oxidative stress. In general, pregnancy is a physiological condition of protracted, modestly enhanced oxidative stress because of the foetal growth and development's high oxygen metabolic requirements. e physiological in ammatory phenomena of pregnancy that is necessary for the angiogenic processes required to increase the placental vascular bed have led to this modestly heightened oxidative stress. Superoxide anion hydrogen peroxide, hydroxyl radical NO, and Peroxynitrite are examples of common ROS and RNS products that are produced from oxygen. In cellular metabolism, cell signalling cascades, and gene expression under physiological circumstances, ROS and RNS play critical roles. During pregnancy, oxidative stress is brought on by an increase in the placenta's mitochondria's metabolic activity and an excess of ROS.

Acknowledgement

None

Conflict of Interest

None

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