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The Dietary Exposome on Oxidative Stress in Pregnancy Complications

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Abstract

One in every four to five pregnancies is affected by pregnancy problems, such as gestational diabetes, hypertension, foetal growth restriction, and premature delivery. There is growing evidence that these difficulties are accompanied with an increase in the generation of reactive oxygen species. Although the precise methods by which reactive oxygen species contribute to pregnancy difficulties are not fully known, they may have a causative role in disease pathophysiology given that they are agents that cause cell stress. Pregnancy-related exposures may enhance the creation of reactive oxygen species since several environmental and lifestyle variables and exposures have been shown to affect their production. This review's goal is to give a thorough summary of the endogenous and external exposome components that control reactive species in complex, healthy organism's pregnancies in order to lessen unfavourable pregnancy outcomes; we also describe dietary strategies that attempt to reduce reactive species.

Keywords: Exposome; Pregnancy; Oxidative stress; Inflammation; Diet; Placenta

Introduction

Dietary therapies generally pose little danger during pregnancy and might be viewed as an effective 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]. The "major obstetrical syndromes," which include preeclampsia, foetal growth restriction, and premature labour, have common aetiologies [5]. These 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]. They 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]. The word the term "great obstetrical syndromes" was initially used to refer to conditions that affect 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 significant recurrence risk. GOS is continuously increasing globally [10].

Discussion

The main idea is that these aetiologies are caused by things that happen during foetal development that affect how nutrients, oxygen, waste products, and toxins are exchanged between the mother and the foetus. These 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 define the exposome, all internal, non-genetic elements that affect a person's health during the course of their life, especially during pregnancy. These determinants may be split into three categories: internal (hormones, inflammation, and oxidative stress), specific external (infectious agents, food, and lifestyle), and general external (education, socioeconomic position, and mental load). The growing baby is exposed to a variety of endo- and echo-exposome variables throughout pregnancy, most notably oxidative stress, nutrition, and inflammation. Gestational diabetes mellitus is one example of a pregnancy problem when the exposome is active. GDM is a metabolic condition that manifests as gradually reduced glucose tolerance starting at the end of the second trimester due to the interaction of maternal predisposition and placental factors. to a pregnancy's third trimester Different endo- and ecto-exposome variables that disrupt the maternal-fetal relationship during GDM cause long-lasting postnatal sequelae and adverse effects on the health of both the mother and the foetus. Although certain predisposing variables, such as a pre-pregnancy mother body mass index of at least 30 kg/m2 and genetic susceptibility, are acknowledged, the pathophysiology behind each of the aforementioned pregnancy problems is yet unknown.

Conclusion

The "placental syndrome," which underlies the major obstetrical syndromes, is strongly linked to both inflammation and excessive oxidative stress. This imbalance occurs between the productions of reactive species, such as reactive oxygen species (ROS) and reactive nitrogen species, and the antioxidant defence system. Maternal vascular malperfusion is caused by an inadequately deep placentation since inflammatory responses lead to oxidative stress and oxidative stress feeds an ongoing inflammatory 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 inflammation that shift immune tolerance in the direction of immune effector activation. It is still unknown how exactly oxidative stress and inflammatory processes contribute to the beginning and development of pregnancy problems. However, these mechanisms' contribution to the disease of since inflammatory responses lead to

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Acknowledgement

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Conflict of Interest

None

References

1. Banhidy F, Lowry RB, Czeizel AE (2005) Risk and benefit of drug use during pregnancy. Int J Med Sci 2: 100-106.
2. Deborah E, McCarter, Spaulding MS (2005) Medications in pregnancy and lactation. MCN Am J Matern Child Nurs 30: 10-17.
3. Ward RW (2001) Difficulties in the study of adverse fetal and neonatal effects of drug therapy during pregnancy. Semin Perinatol 25: 191-195.
4. Hansen W, Yankowitz J (2002) Pharmacologic therapy for medical disorders during pregnancy. Clin Obstet Gynaecol 45: 136-152.
5. Loebstein R, Lalkin A, Koren G (1997) Pharmacokinetic changes during pregnancy and their clinical relevance. Clin Pharmacokinet 33: 328-343.
6. Sharma R, Kapoor B, Verma U (2006) Drug utilization pattern during pregnancy in North India. J Med Sci 60: 277-287.
7. Andrade SE, Gurwitz JH, Davis RL, Chan KA, Finkelstein JA, et al. (2004) Prescription drug use in pregnancy. Am J Obstet Gynaecol 191: 398-407.
8. Splinter MY, Sagraves R (1997) Prenatal use of medications by women giving birth at a university hospital. South Med J 90: 498-502.
9. De Jong LT, Van den Berg PB (1990) A study of drug utilization during pregnancy in the light of known risks. Int J Risk Safety Med 1: 91-105.
10. Briggs GG (2002) Drug effects on the fetus and breastfed infants. Clin Obstet Gynaecol 45: 6-21.