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Prenatal Alcohol Exposure Mediates Adverse Effects on Placental and Fetal development by Impairing Trophoblast Differentiation and Vascular Transformation

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The article, "Dual mechanisms of ethanol-impaired placentation: experimental model", by Drs. Gundogan et al. [1] utilized a robust experimental model of gestation alcohol exposures in rats to examine effects on placentation and fetal development. Drs. Gundogan et al. have pioneered a new field demonstrating the contributions of alcohol-induced impairments in placental growth and development to the pathogenesis of fetal alcohol spectrum disorders. Accumulating evidence indicates that deficits in fetal development and subsequent brain structure and function are linked to impairments in placentation. The authors used state-of-the art molecular approaches as well as high-quality histomorphologic methods to illustrate alcohol-mediated disruption of trophoblastic cell differentiation and vascular transformation; the latter is needed to supply nutrients to, and remove wastes from the fetal environment. The authors correlate structural abnormalities with alterations in the expression of genes that mark the phenotype of placental stem cells, progenitor cells, and invasive trophoblasts. Therefore, this work extends the exciting chapter characterizing the effects of alcohol on the maternal-placental-fetal axis by illustrating that for a given dose of alcohol, timing in relation to the start of pregnancy is critical. Furthermore, the authors showed that the greatest impairments in placental and fetal development occur when alcohol exposure is initiated close to the period of stem cell activation. The relevance of this work to humans is that the adverse effects of prenatal alcohol exposures are likely to be maximum for women who consume alcohol early in gestation, including the interval prior to realization of the gravida state.

Reference

1. Gundogan F, Gilligan J, Ooi JH, Sung J, Qi W, et al. (2013) Dual Mechanisms of Ethanol-Impaired Placentation: Experimental Model. J Clin Exp Pathol 3: 142

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