

Effects of Inorganic Arsenic on the Epithelial-Mesenchymal Transition, Migration, and Invasion of Placental Cells

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The negative health effects of prenatal exposure to inorganic arsenic (iAs) may result from the direct interaction between the placenta and iAs. The placenta supports proper fetal development by promoting the exchange of nutrients and waste between the fetal and maternal blood supplies. Integral in this function, trophoblasts invade the decidualized endometrium, and migrate towards the maternal spiral arteries, and promote their remodeling to widen the lumen, allow a high rate of blood flow. In a healthy pregnancy, trophoblast invasion and arterial remodeling extends through the decidua and inner myometrium. These invading trophoblasts, termed extravillous trophoblasts (EVTs), undergo an epithelial-mesenchymal transition (EMT). EMT, migration, and invasion have most commonly been studied under the context of iAs-induced carcinogenesis. Studies have yet to examine the effect of iAs on these processes in trophoblast cell lines. To this end, we collected data on the migration of HTR8/SVneo cells and gene expression of EMT markers in placental explants in response to iAs treatments. We observed an increase in migration in response to 0.5 uM and 1.0 uM iAs treatment, with significance ($p < 0.05$) 48 hours after treatment. Gene expression of three EMT markers and two angiogenesis-related genes was significantly altered ($p < 0.05$) in relation to iAs treatment, with all genes showing an increase in expression. These findings support the hypothesis that iAs exposure may negatively impact maternal and fetal health during pregnancy by altering migration of EVT's and disrupting EMT and angiogenesis in the placenta.