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Alcohol exposure suppresses neural crest cells generation and differentiation during early chick embryo

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It is now known that excess alcohol consumption during pregnancy can cause fetal alcohol syndrome (FAS) in which several characteristic craniofacial abnormalities are often visible. However, the molecular mechanisms of how excess ethanol exposure affecting cranial neural crest cells (CNCCs), the progenitor cells of the cranial skeleton, is still not clear. In the study, we investigated the effects of ethanol exposure on CNCCs migration both in early chick embryo and *in vitro* explant culture. First of all, we demonstrated that ethanol treatment caused Alizarin red-stained craniofacial developmental defects including parietal defect. Second, immunofluorescent staining with neural crest special markers indicated that CNCCs generation was inhibited by ethanol exposure and, double immunofluorescent stainings (Ap-2α/PHIS3, HNK1/BrdU and AP-2α/c-caspase3) revealed that ethanol exposure inhibited CNCCs proliferation and increased apoptosis. In addition, it inhibited NCCs production by repressing the expression level of key transcription factors which regulate neural crest development by altering expression of Epithelial-Mesenchymal Transition (EMT)-related adhesion molecules in the developing neural crests. In sum, we have provided experimental evidence that excess ethanol exposure during embryogenesis disrupts CNCCs survival, EMT and migration, which in turn causes defective cranial bone development.

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