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Alterations in cell cycle progression associated with transcription factor E2F1 in FASD.

Phenotypes from alcohol exposure during fetal development, Fetal Alcohol Spectrum Disorders (FASD), include facial dysmorphology, neurological underdevelopment and cognitive loss. Most of the abnormal development is associated with changes in proliferation and cell losses of neural crest stem cells during development. Alterations in proliferation, in FASD, have been associated with loss of checkpoint control primarily at the G1/S phase transition of the cell cycle. Previous studies demonstrated an increase in E2F1 transcription factor expression and expression changes of the E2F regulatory protein Retinoblastoma (RB). However, there is no evidence that alterations in expression effect overall proliferation. We suggest that his mis-regulation may affect not only proliferative differences, but loss of cell numbers and developmental alterations in FASD. To examine the functional significant effect of E2F1/RB expression changes we performed Electro-Mobility Shift Assays and Super-Shift Assays to determine the DNA consensus binding of E2F1 and the changes in repression of transcription associated with RB interactions. We show that alcohol exposure of embryonic neuronal stem cells alters E2F1/DNA binding as well as repression from RB. Understanding these changes may well help define treatment options for fetuses affected by alcohol and drug exposure.