BROOKE LANCASTER, and BRUCE ANTHONY, Department of Chemistry/Biochemistry, West Virginia Wesleyan College, Buckhannon, WV, 26201.
Alterations in DNA and RNA profiles from alcohol exposed neuronal stem cells relates a possible mechanism for slowed proliferation and increased apoptosis.

Fetal alcohol spectrum disorders (FASD) are associated with a mother who consumed alcohol while pregnant. FASD symptoms in the fetus include physical effects, behavior abnormalities and learning problems. Phenotypes are associated with alterations in proliferation, differentiation, migration and increased cell death of neural crest stem cells during embryo development. Under normal growth conditions Cyclin D1 expression is transiently increased during late G1 phase and signals the cell to proceed to S-phase. CyclinD1 initiates a cascade of events necessary for transcription of genes required for proper DNA synthesis. It has been shown in mouse embryos that alcohol increases Cyclin D1 protein expression 2-fold, which in turn induces picnosis in 40 % of alcohol exposed neuronal stem cells. We suggest that alcohol has induced premature G1/S phase transition by Cyclin D1 overexpression, which, in turn, alters DNA synthesis and increases apoptosis.

Through the use of flow cytometry we examined both RNA and DNA profiles with gating focuses on G1 and S phases respective. We then examined by antibody/fluorescent staining the overlapping expression of Cyclin D1 levels on these gated populations. Under normal growth conditions, Cyclin D1 expression is limited to the G1/S phase transition. Alcohol alters this expression pattern and suggests a continued expression in S phase cells. Profiles allowed an examination of apoptosis percentage as well. Understanding this mechanistic misregulation may eventually lead to a full understanding and improved treatment options for fetuses exposed to alcohol.