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Examination of ku70/80 proteins in non-homologous end-joining repair of double-stranded dna induced by alcohol exposure in neuronal stem cells.

Fetal Alcohol Spectrum Disorders (FASD) demonstrates increased DNA damage and ultimately apoptosis in neural crest stem cells necessary for early development. In adult alcoholism, neuronal stem cells also demonstrate increased DNA damage from chronic and binge drinking.

Although increases in cell death are a hallmarks of stem cell alcohol exposure, there is little understanding of the mechanism that induces DNA damage. Previous comet assay suggest both single and double stained DNA breaks. In this study, neuronal stem cells were treated with 400 mg/dl alcohol over an 8 hour period and examined for double stranded DNA breaks. KU70/80 proteins are part of the repair complex for double stained DNA breaks and bind to non-homologous ends at DNA strand breaks. We suggested that increases in DNA breaks from alcohol my work through a similar repair mechanism to recovery from DNA damage. Examination of this protein was achieved by immunocytochemistry using exposed stem cells. The results in KU expression show only slight increases in expression of either KU70 or KU80 after alcohol exposure. This suggests that KU70/80 protein expression and repair is not the only DNA repair mechanism associated with alcohol exposure and cell death.