Excessive consumption of alcohol during pregnancy causes fetal alcohol spectrum disorders (FASD), which is represented through growth deficits of neuronal stem cells contributing to microencephaly and neurological abnormalities. These changes seem linked to reduced proliferation and accelerated apoptosis in neuronal stem cells exposed to alcohol. There is little understanding on how alcohol affects the cell cycle during development. Previous research suggests alterations in stem cell populations at the G1/S phase checkpoint control as a result of misregulated expression of E2F and DP1 transcription factor families. This altered transition causes disrupted DNA synthesis and likely induction of apoptosis. We examined the expression patterns of E2F1 using immunocytochemistry and flow cytometry. We suggest that overexpression of E2F1 may be responsible for the mechanistic changes seen in the cell cycle from alcohol exposure to stem cells.