Increased use of ethanol blends in gasoline suggests a need to assess the potential public health risks of exposure to these fuels. Ethanol consumed during pregnancy is a teratogen. However, little is known about the potential developmental neurotoxicity of ethanol delivered by inhalation, the most likely route of exposure from gasoline-ethanol fuel blends. We evaluated the potential **cognitive** consequences of ethanol inhalation by exposing pregnant Long Evans rats to clean air or ethanol vapor from gestational days 9-20, a critical period of neuronal development. Concentrations of inhaled ethanol (5000, 10,000, or 21,000ppm for 6.5h/day) produced modeled peak blood ethanol concentrations (BECs) in exposed dams of 2.3, 6.8, and 192mg/dL, respectively. In offspring, no dose-related impairments were observed on spatial learning or working memory in the Morris water maze or in operant delayed match-to-position tests. Two measures showed significant effects in female offspring at all ethanol doses: 1) impaired cue learning after trace fear conditioning, and 2) an absence of bias for the correct quadrant after place training during a reference memory probe in the Morris water maze. In choice reaction time tests, male offspring (females were not tested) from the 5000 and 10,000ppm groups showed a transient increase in decision times. Also, male offspring from the 21,000ppm group made more anticipatory responses during a preparatory hold period, suggesting a deficit in response inhibition. The increase in anticipatory responding during the choice reaction time test shows that inhaled ethanol yielding a peak BEC of ~200mg/dL can produce lasting effects in the offspring. The lack of a dose-related decrement in the effects observed in females on cue learning and a reference memory probe may reflect confounding influences in the exposed offspring possibly related to maternal care or altered anxiety levels in females. The surprising lack of more pervasive **cognitive** deficits, as reported by others at BECs in the 200mg/dL range, may reflect route-dependent differences in the kinetics of ethanol. These data show that response inhibition was impaired in the offspring of pregnant rats that inhaled ethanol at concentrations at least 5 orders of magnitude higher than concentrations observed during normal automotive transport and fueling operations, which rarely exceed 100 ppb.