[ID-P#072] The Neurotoxicity Effects of In- Utero Exposure to Bisphenol A on Learning and Memory Function

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Bisphenol A (BPA) is an endocrine-disrupting compound and is widely known to cause neurotoxicity effects on brain functions. Maternal exposure to BPA is particularly concerning as it can influence the learning and memory function of the offspring. Yet, the underlying mechanism through which these impairments are transmitted from mother to offspring remains uncertain. In this study, the effect of in-utero BPA exposure on learning and memory function was investigated by studying the level of estrogen receptor (ER) in the rat placenta and NMDA receptor subunits, GluN2A and GluN2B, in the male rat hippocampus. Pregnant Sprague Dawley rats were orally exposed to BPA at 5 mg/kg/day from pregnancy day 1 until gestation day 21 (GD21), while the control mothers were without BPA. The mothers were monitored daily until GD21 for cesarean section or spontaneous delivery. The placenta and male fetus hippocampus were dissected at GD21, while the adolescent male hippocampus was dissected at 35 days old (AD35). At GD21 and AD35, the expression of NMDA receptor subunit genes, GRIN2A and GRIN2B was determined by qRT-PCR, while levels of ERα, ERβ, GluN2A and GluN2B by ELISA. The memory retention ability was evaluated using the Morris water maze and step-down passive avoidance test on AD35. The results showed there is a significant increase in the levels of ERα and ERβ in the placenta. In the hippocampus, the expression of GRIN2A and GRIN2B as well as GluN2A and GluN2B were significantly decreased at both ages. Consequently, the AD35 rats showed significant behavioral impairment in both memory tests. In conclusion, the changes in ER level may disrupt the protective roles of the placenta, potentially increasing the fetus's vulnerability to BPA. The persistent impact of in-utero exposure to BPA disrupts the regulation of NMDA receptor subunits, subsequently leading to learning and memory impairment when reaching adolescence.