Maternal Health and Placental Outcomes after Pulmonary Nanoplastic Exposure

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Accumulating plastic waste in the environment has recently led to increasing concerns pertaining to human health. Microplastic concentrations have been measured in the environment, bottled water, food, and human feces. Studies have also demonstrated the ability of plastic to fragment into smaller particles, producing nanoplastics. Though methods of nanoplastic detection and quantification are still in development, the breakdown of micro- to nano-sized particles has been identified. Our lab has shown nanoparticles produced from metallic bulk material can cause adverse pregnancy outcomes after maternal inhalation, including fetal growth restriction. However, maternal-fetal outcomes and placental toxicity after nanoplastic exposure remain unexplored. Here we sought to begin the evaluation of gestational health after pulmonary nanopolystyrene exposure. Sprague-Dawley rats were administered 300 µL of either 20 nm rhodamine-labeled polystyrene (PS) beads (8.8×10^{14} particles/mL) or saline on gestational day 19. Animals were sacrificed 24 hours later and tissues were collected for assessment. Dams who were administered PS had significantly more circulating 17β- estradiol (1104.5 ± 68.1 pg/mL vs 972.0 ± 18.4 pg/mL) and hCGβ (53.4 ± 12.0 pg/mL vs. 6.3 ± 7.5 pg/mL) compared to saline controls. Placental weights in the exposed group were also significantly increased (0.522 ± 0.06 g vs 0.472 ± 0.08 g), with no compensatory increase in fetal weight (2.6 ± 0.7 vs 2.6 ± 0.1). Therefore assessment of placental efficiency, identified as the ratio of placental to fetal mass, was decreased (4.8 ± 0.53 vs 5.5 ± 0.74) in the exposed group. Overall, these studies demonstrate a disruption to maternal endocrine homeostasis during late-stage pregnancy after PS pulmonary exposure. Further, placental mass is elevated within 24 hours of a single maternal pulmonary exposure, which may be due to PS translocation and/or placental inflammation. Increases in hCG production and placental weight without increase in fetal weight indicate placental adaptation to nanoplastic exposure, but with negative effect on placental efficiency. If sustained, these findings suggest maternal compensation to maintain fetal growth and development after PS exposure. *Supported by: NIH-R00-ES024783 (PAS); T32-ES007148 (JND); P30-ES005022.*