Maternal low-dose estradiol-17\(\beta\) exposure during pregnancy impairs postnatal progeny weight development and body composition.

Abstract:
Endocrine disrupting chemicals with estrogenic activity play an important role as obesogens. However, studies investigating the most potent natural estrogen, estradiol-17\(\beta\) (E2), at low dose are lacking. We examined endocrine and physiological parameters in gilts receiving distinct concentrations of E2 during pregnancy. We then investigated whether adverse effects prevail in progeny due to a potential endocrine disruption. E2 was orally applied to gilts during the entire period of pregnancy. The concentrations represented a daily consumption at the recommended ADI level (0.05 \(\mu\)g/kg body weight/day), at the NOEL (10 \(\mu\)g/kg body weight/day) and at a high dosage (1000 \(\mu\)g/kg body weight/day). Plasma hormone concentrations were determined using enzyme immuno assays. Offspring body fat was assessed by dual-energy X-ray absorptiometry scanning. In treated gilts receiving 1000\(\mu\)g E2/kg body weight/day we found significantly elevated plasma E2 levels during pregnancy, paralleled by an increased weight gain. While offspring showed similar weight at birth, piglets exhibited a significant reduction in weight at weaning even though their mothers had only received 0.05 \(\mu\)g E2/kg body weight/day. At 8 weeks of age, specifically males showed a significant increase in overall body fat percentage. In conclusion, prenatal exposure to low doses of E2 affected
pig offspring development in terms of body weight and composition. In line with findings from other obesogens, our data suggest a programming effect during pregnancy for E2 causative for the depicted phenotypes. Therefore, E2 exposure may imply a possible contribution to childhood obesity.