

Maternal Bisphenol S exposure impairs testicular function in adult *Wistar* rat offspring via thyroid axis disruption

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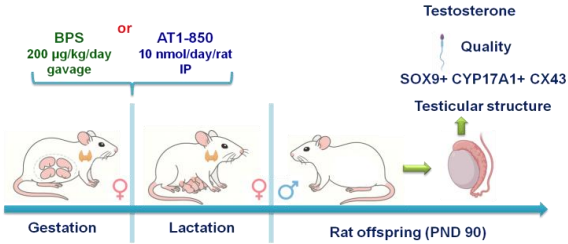
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INTRODUCTION

- Emerging evidence indicates that BPS acts as an endocrine disruptor.
- Thyroid hormones regulate testicular development, steroidogenesis, and spermatogenesis.
- The role of thyroid hormone receptor (THR) disruption in BPS-induced reproductive toxicity remains unclear.
- To determine whether maternal exposure to BPS during gestation and lactation impairs testicular function in adult male offspring through disruption of THR signaling.

METHODOLOGY



RESULTS

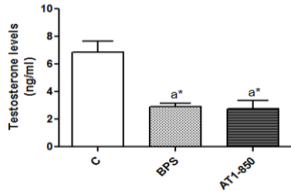
1- Reproduction outcomes

Table 1: Body weight, anogenital distance, Relative weight of reproductive organs and Sperm parameters of adult male offspring (PND90)

Findings	C	BPS	AT 1-850
% of sperm motility	59.62 ±6.05	47.32 ±2.72a	46.26 ±5.68a
% of sperm viability	94.52 ±0.95	90.93 ±1.30a*	90.11 ±1.78a**
% of sperm abnormalities	29.78 ±6.81	60.24 ±9.47a**	61.83 ±7.60a**
Daily sperm production (DSP) 10 ⁶	25.19 ±2.66	17.47 ±2.53 a*	17.87 ±1.06 a
Efficiency of DSP	22.3 ±0.92	18.87 ±2.38a**b*	12.19 ±1.00a**

- BPS and AT 1-850 maternal exposure impaired key sperm parameters and daily sperm production.

RESULTS



- Maternal BPS and AT 1-850 exposure reduced testosterone levels in pubertal male offspring.

Figure 1. Testosterone levels in adult male offspring (PND90).

2- Testicular Histology

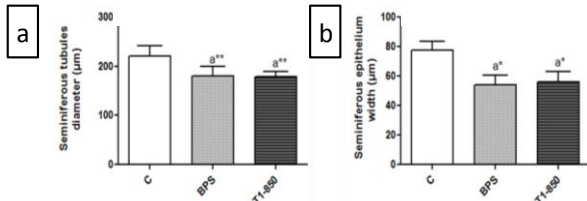
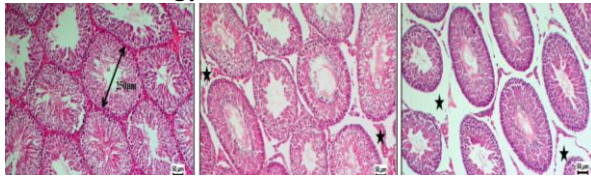


Figure 2. Histological structure of the testes in adult male offspring (PND90): (a) Effects of treatments on seminiferous tubules diameter; (b) Effects of treatments on seminiferous epithelium width.

- BPS and AT 1-850 exposure altered testicular morphology, reducing seminiferous tubule size and epithelial thickness.

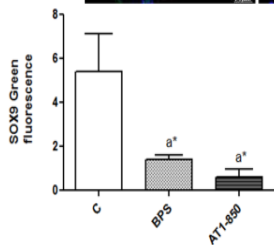
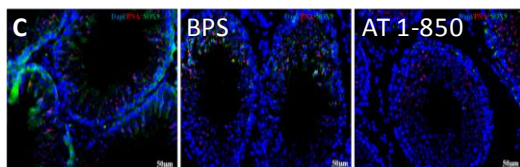


Figure 3. Expression of SOX9 protein in testes of adult male offspring (PND90). SOX9(green), DAPI (blue), PNA lectin (red) which marks the acrosome.

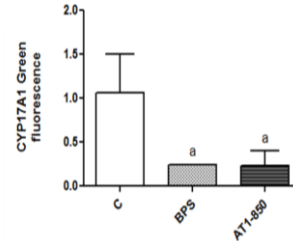
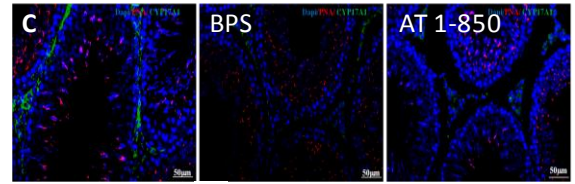


Figure 4. Expression of CYP17A1 protein in testes of adult male offspring (PND90). CYP17A1 (green), DAPI (blue), PNA lectin (red) which marks the acrosome.

- Altered SOX9 and CYP17A1 expression in exposed groups.

4- Gene Expression

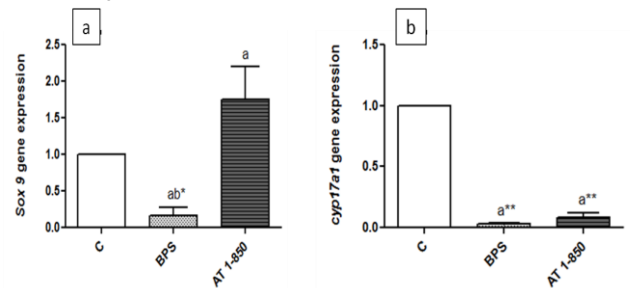


Figure 5. Expression of *Sox9* (a) and *Cyp17a1* (b) genes in testes of adult male offspring (PND90)

- Altered SOX9 (↓ in BPS, ↑ in AT) and CYP17A1 (↓ in both exposed groups).

CONCLUSIONS

- BPS impairs steroidogenesis and spermatogenesis.
- BPS alters testicular morphology
- Similar effects produced by the THR antagonist AT 1-850 support the involvement of thyroid hormone receptor signaling.
- Developmental exposure to BPS may compromise long-term male fertility through thyroid-testis axis disruption.