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Bisphenol A (BPA) confers direct genotoxicity to sperm with increased sperm DNA fragmentation

• D.H. Wu, Y.-K. Leung, M.A. Thomas, R. Maxwell, S.-M. Ho
Obstetrics and Gynecology, University of Cincinnati, Cincinnati, OH; Environmental Health, University of Cincinnati, Cincinnati, OH

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Objective

BPA is associated with disruptive effects on spermatogenesis and reproductive organ development in animals, but studies on direct effects of BPA on human sperm function are lacking. We previously reported increased sperm total motility and rapid progression in response to estrogenic compounds and aimed to investigate the in vitro effects on sperm DNA fragmentation induced by BPA and 17β-estradiol (E).

Design

Prospective Controlled Study.

Materials and Methods

Samples were collected from men undergoing semen evaluation (n = 27; 21 with normal and 6 with abnormal semen parameters). Washed sperm resuspended in HTF+10% SSS was incubated with either 1nM E, 1μM or 10 nM of BPA (B1, B2). Sperm DNA fragmentation was measured by the alkaline comet assay. Comet length (CL), %DNA in tail (T%), and tail moment (TM) were assessed using CometScore software and compared to vehicle control (C) at 24 hours after initial exposure. Data was analyzed using repeated measures ANOVA and paired t-tests.

Results

The samples had a mean ± SD sperm count of 73.0 ± 44.3 mil/mL, motility of

64.5 ± 9.0%, and normal morphology of 8.6 ± 5.5%. E and both concentrations of BPA significantly increased sperm DNA fragmentation with increased CL (P<0.001) and increased TM (P=0.001). Mean CL was 95.1 ± 12.2px for C, compared with 106.6 ± 12.5px for E (P<0.001), 105.9 ± 15.0px for B1 (P<0.001), and 105.4 ± 14.1px for B2 (P=0.001). Mean TM was 64.7 ± 19.5px for C, compared with 74.8 ± 20.8px for E (P<0.001), 74.3 ± 19.5px for B1 (P<0.001), and 75.5 ± 17.0px for B2 (P=0.003). T% did not differ between the 4 arms (P=0.88). The effects on CL and TM persisted after differentiating samples by normal vs abnormal semen parameters (P=0.6; P=0.8).

Conclusion

This is the first study to show evidence of direct genotoxity to sperm from BPA exposure in vitro. The significant increase in sperm DNA fragmentation favoring double-strand breaks caused by BPA mimic effects of E, and may allow for future studies examining the direct mechanisms of these toxic effects.