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Octylphenol and Bisphenol-A reduce sperm production, and differentially affect plasma reproductive steroid hormone profiles and accessory reproductive organs in peripubertal male Wistar rats

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ABSTRACT

It has been proposed that a global decline in sperm counts, semen quality and several male reproductive disorders are associated with exposure to environmental chemicals, including those possessing estrogenic activities. In the present study we examined the effects of two estrogenic chemicals, octylphenol (OP) and bisphenol-A (BPA), on plasma and testicular levels luteinizing of steroid hormones, plasma levels of luteinizing hormone (LH) and insulin-like growth factor I (IGF-I), luteinizing hormone releasing hormone (LHRH)-stimulated plasma LH and steroid hormones, and the effects on testis, accessory reproductive organs, epididymal sperm motility and epididymal sperm counts in peripubertal male Wistar rats. Fifty-day-old rats in the OP group (n=11) and BPA group (n=11) received daily s.c. injections of the respective chemical at a dose of 3mg/kg BW dissolved in 0.2ml DMSO. Rats in the control group (DMSO group; n=10) received 0.2 ml DMSO alone. The treatment period was five weeks. At two weeks, a jugular blood sample was taken, and on the next day, second blood sample was taken 1 h after an s.c. injection of LHRH (250ng). Body weights were recorded at regular intervals. At 5 weeks, rats were deeply anesthetized and heart blood was collected. Left epididymis was used to determine sperm motility and sperm head counts. The weight of testis and accessory reproductive organs were recorded. In the OP and BPA groups, sperm motility parameters such as rapid linear movement, linearity index and straightline velocity were reduced but did not reach significant levels. Conversely, the motility parameters such as slow nonlinear and rapid to slow ratio in the same two groups were increased though not significantly different from those of controls. Epididymal sperm counts were significantly ($P < 0.05$) decreased in both the OP and BPA groups compared to that in the DMSO group. Body weight was not different between the three groups at 5 weeks of treatment. However, relative weight of the right epididymis was significantly ($P < 0.05$) reduced in the OP group compared to that in the DMSO group. On the other hand, relative weight of the ventral prostate gland was significantly ($P < 0.05$) increased in both the OP and BPA groups compared to that in the DMSO group. LHRH treatment increased plasma LH to higher levels in the groups, but the increases were more apparent in the OP and BPA groups. The increased LH levels were associated with elevated plasma testosterone (T) and progesterone (P) levels in all groups except the OP group in which P level was reduced. However, despite higher LH levels in the OP and BPA groups after LHRH treatment, the incremental response of T and P was not as high as that observed in the DMSO group. At 5 weeks, plasma T levels were significantly ($P < 0.05$) reduced in the OP and BPA groups but these two groups had high P levels compared with those in the DMSO group. T and P levels in testicular homogenates were not different between the three groups. Plasma IGF-I levels were significantly ($P < 0.05$) increased in the OP and BPA groups. The present data agree with previous reports that OP and BPA can reduce sperm counts resulting from lowered plasma T. The data also indicate that the chemicals may have an adverse effect on sperm motility. Though the pituitary gland appears to respond normally to LHRH, the increased LH in the OP and BPA groups was unable to produce higher plasma T and P, suggesting that OP and BPA had possibly interfered with the mechanism responsible for secretion of the hormones. The increased growth of the ventral prostate gland may possibly be associated with increased plasma IGF-I, the release of which is stimulated by exogenous estrogens.