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Does preconception paternal exposure to a physiologically relevant level of bisphenol A alter spatial memory in an adult rat?

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ABSTRACT

Bisphenol A (BPA) is a ubiquitous environmental endocrine disrupting compound (EDC); public health concerns have been fueled by findings that maternal BPA exposure can change sex differences in the brain and in some behaviors. We investigated whether a physiologically relevant dose of BPA ingested by male rats before conception would affect spatial memory and hippocampal acetylcholinesterase (AchE) in their adult offspring. Twenty-two 60-day-old male rats (F0) received either a BPA diet (50 μg/kg/day) or vehicle alone for 10 weeks before being mated with non-exposed females. The paternal rats and their forty adult offspring's (F1) behaviors were then examined in the Morris Water Maze (MWM) and their AchE activities in the hippocampus were evaluated. BPA exposure led to spatial memory deficits along with decreased AchE activities in the hippocampus ($p = 0.01$) in adult F0 rats. This paternal exposure also induced impairment in spatial memory acquisition in both sexes while retention only in females in F1 rats, as well as abolished sex differences in the hippocampus AchE. Overall, these data provide new evidence that paternal BPA exposure, at a "safe" dose, may induce transgenerational alterations in spatial memory in a sex-specific manner.

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Introduction

Among endocrine disruptors (EDCs), the xenoestrogen bisphenol A (BPA) deserves particular attention due to its widespread exposure to humans. Indeed, detectable levels of BPA have been found in samples of urine, blood, breast milk and saliva of both humans and nonhuman subjects (Patisaul and Bateman, 2008). Recent findings suggest that chronic exposure to BPA in rodents during child development may result in extensive physical and mental alterations (Hajszan and Leranth, 2010; Wolstenholme et al., 2011). It has also been reported that BPA can interfere with the physiology and morphology of an adult brain. For example, acute or chronic BPA administration can impair both visual and spatial memories and synaptic plasticity in gonadal intact male rats (Eilam-Stock et al., 2012; Leranth et al., 2008) and block estrogen-induced memory enhancement in adult ovariectomized rats (Inagaki et al., 2012). However, in most of these behavioral studies, BPA was given during development and few have investigated the effects of adult BPA exposure in male subjects. In addition, most of the male subjects were exposed to BPA at high concentrations (Leranth et al., 2008) or administration was by subcutaneous injection (Eilam-Stock et al., 2012) which may not be relevant to the human condition as current human BPA exposure occurs with low dosage and mainly by oral ingestion. For these reasons, we chose to investigate the effects of chronic dietary exposure to BPA at a physiologically relevant level on spatial memory using an adult male rat model.

Another avenue for BPA effects on child development is through an action on infant–maternal interactions which may have an epigenetic impact on the behavior of the offspring (Wolstenholme et al., 2011). It has been observed that in cynomolgus monkeys, dams that ingested BPA during the gestation period cared less for their pups than did control females which resulted in increased resistance and exploration in their male offspring (Nakagami et al., 2009). The effects of this chemical on juvenile mice were more dramatic when the pups were reared by foster dams, as both diet and dam affected their social behavior and anxiety (Cox et al., 2010). Another cross-fostering study also revealed that the increase in anxiety-like behaviors and corticosterone response in F2 offspring of lipopolysaccharide-exposed mothers were reversed by the cross-fostering treatment and this phenotype was due to the reduction of maternal care experienced by the F2 animals (Walker

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et al., 2012). This evidence therefore, confirms that maternal-mediated transmission may be dependent on maternal care rather than epigenetic mechanisms and that exposure to a gestational female does not necessarily constitute a transgenerational phenotype but rather multigenerational exposure. However, unless the germ cell is directly affected, then a transgenerational phenomenon is possible (Manikkam et al., 2012; Skinner et al., 2011).

Given the above-mentioned factors, a model based on paternal exposure seems plausible, because males do not have direct physiological or behavioral interaction with their offspring, since they only contribute sperm for the next generation. This, therefore, avoids confounding effects such as maternal behavior. This overall evidence suggests that there is a need for additional research to examine paternally mediated effects of BPA particularly, as BPA is a ubiquitous pollutant.

It is also important to note that a plethora of studies have examined developmental BPA exposure in laboratory animals but they have often generated inconsistent results. As a result, explicit guidelines for BPA research have been described. The suggested recommendations include the control of exogenous estrogens (Birnbaum et al., 2012), statistical control for litter effects, internal measures of BPA (Patisaul and Bateman, 2008) and the use of oral administration for the most relevant extrapolation to humans (Betancourt et al., 2012). However, the last recommendation could be problematic for laboratory animal research because the oral administration (typical method) of orogastric gavage can be stressful for rodents. Gestational stress can in addition, result in later alterations of offspring behavior; therefore, controlling or evaluating this potential confound would be beneficial. The proposed guidelines mentioned above were incorporated into the current study

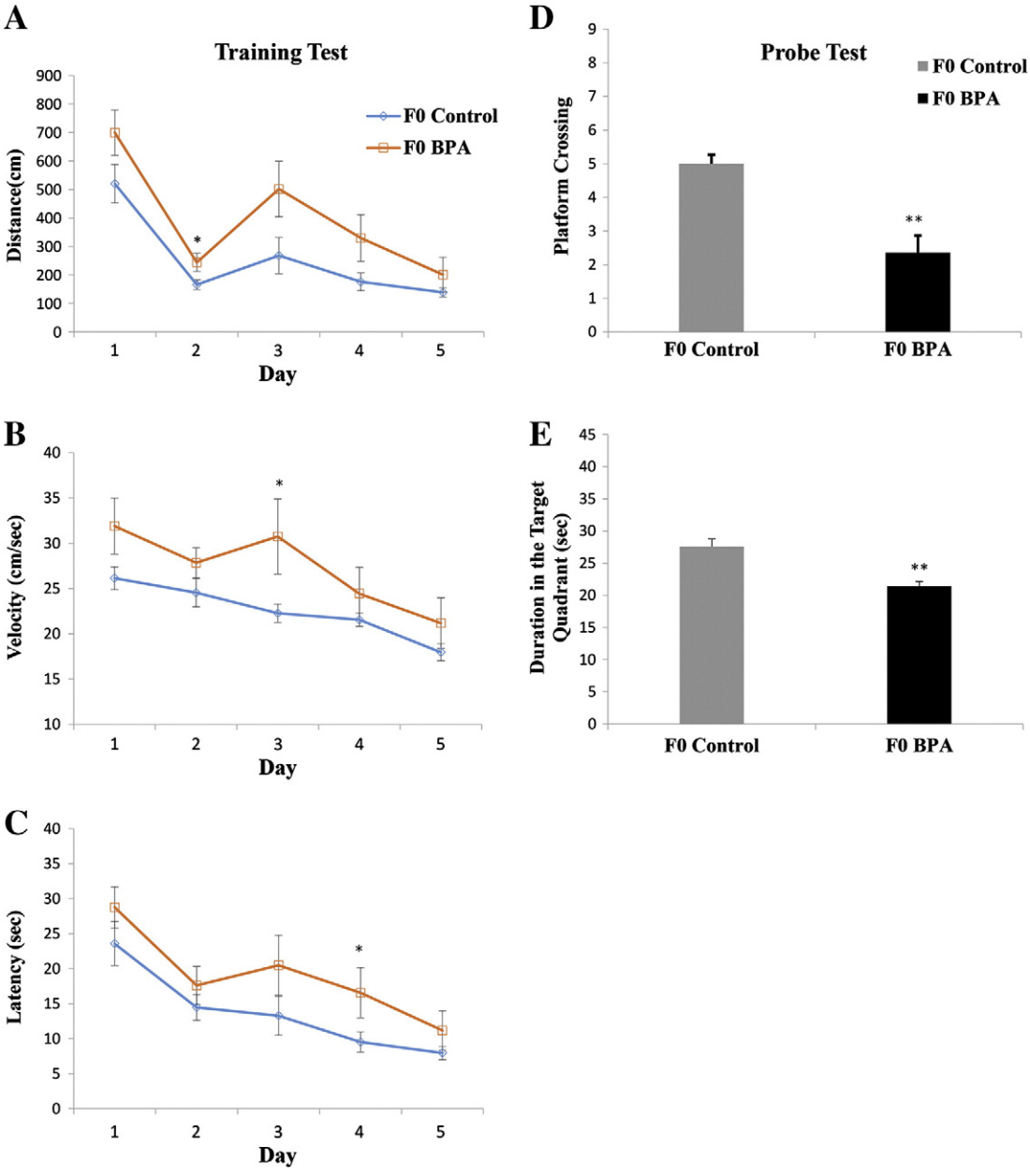

Fig. 1. Effects of adult BPA exposure on spatial memory in male F0 rats. The MWM tests were performed in 144-day-old male rats after 10 week treatment with BPA (50 μg/kg/day). Data from the training test are averages of 4 trials per day for 5 consecutive days. (A) Distance moved: F0 BPA rats swam longer distance before reaching the platform than F0 controls. (B) Mean velocity: F0 BPA rats swam faster than F0 controls. (C) Escape latency: F0 BPA rats needed more time to find the platform than F0 controls. On the sixth day, the escape platform was removed and animals were reintroduced into the pool to perform the probe test. F0 BPA rats made fewer platform contact accuracies and spent less time in the platform quadrant than F0 controls during the entire 60 s trail (D and E, respectively). Mean ± S.E.M., $n = 11$, $^{*}p < 0.05$ and $^{**}p < 0.01$ vs. control.

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