



Brain Serotonin 5-HT₂ α Receptor Density in Male Rats Can Be Affected by Bisphenol A and Passive Avoidance Learning

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Abstract

Background: Bisphenol A (BPA) may adversely affect the nervous system.

Objectives: This study aimed to assess the effects of passive avoidance learning on BPA-induced toxicity, as indicated by serotonin 5-HT₂ α receptor density in the cerebellum, basolateral amygdala, and hippocampus of male rats.

Methods: Thirty male rats (200 - 220 g) were allocated to six experimental groups. BPA was administered orally for 15 consecutive days. Learning and memory performance were assessed using a passive avoidance shuttle-box paradigm. Serotonin 5-HT₂ α receptor density was evaluated by immunohistochemistry in the selected brain regions. Immunohistochemical labeling was quantified by measuring color parameters using image-analysis software.

Results: BPA at both doses reduced 5-HT₂ α serotonin receptor density in the examined brain structures. Passive avoidance learning mitigated the toxic effects of BPA on 5-HT₂ α receptor density in these structures.

Conclusions: Overall, BPA exposure downregulated 5-HT₂ α receptors in the hippocampus, amygdala, and cerebellum of male rats, whereas passive avoidance learning and memory were associated with an increased receptor density.

Keywords: Bisphenol A, Passive Avoidance Learning, Immunohistochemistry, 5-HT₂ α Subunit of the Serotonin Receptor, Male Rats

1. Background

Humans and other living organisms are substantially exposed to xenoestrogens (1). Xenoestrogens can act as exogenous agents and cause changes or disturbances in the synthesis, secretion, transport, binding, action, and elimination of steroid hormones (2). Bisphenol A (BPA) is a xenoestrogen that exhibits estrogenic activity and acts as a weak partial agonist at estrogen receptors α and β (3, 4).

Sex steroid hormones are essential for the central nervous system (5, 6). Steroid hormones can influence neurogenesis and synapse formation in the hippocampus; thus, they can directly increase hippocampal plasticity, resulting in enhanced hippocampus-dependent learning and memory (7).

Exposure to BPA has been associated with decreased dendritic arborization in the dentate gyrus (8, 9). BPA may compete with endogenous steroid hormones for receptor binding, thereby disrupting synaptogenesis and inducing long-lasting alterations in central nervous system organization. These effects include changes in key

neurotransmitter systems, particularly serotonin, which may ultimately impair learning and memory processes (10). Experimental evidence suggests that BPA interferes with synapse development, leading to reduced dendritic branching and spine density in the hippocampus. Such structural changes are accompanied by alterations in neurotransmitter regulation, including serotonergic signaling, and are associated with cognitive deficits (11, 12).

Serotonin is a neurotransmitter present in serotonergic neurons in the central nervous system and in other tissues and is involved in various functions, including learning and memory (13, 14).

2. Objectives

Learning and memory disorders may be associated with changes in serotonin receptor density. Most previous research has examined the role of serotonin in learning and memory, whereas few studies have investigated how passive avoidance tasks influence serotonin receptor density in the brain. This study aimed to determine how passive avoidance learning affects the toxicity of BPA on 5-

HT2 α receptor density in the CA1 region, cerebellar Purkinje cells, and the basolateral amygdala (BLA).

3. Methods

3.1. Animals and Experimental Design

Thirty adult male Sprague-Dawley rats weighing 200 - 220 g were used. Food and water were provided ad libitum. The rats were housed under a 12-hour light/dark cycle (lights on at 6 AM and off at 6 PM) at a controlled temperature of $20 \pm 4^\circ\text{C}$. After a 1-week adaptation period, the animals were randomly divided into six groups ($n = 5$ each): 1) NL-sham, which received BPA vehicle without learning; 2) L-sham, which received BPA vehicle plus learning; 3) and 4) NL-BPA groups, which received BPA at 2 doses without learning; and 5) and 6) L-BPA groups, which received BPA at 2 doses plus learning. The animals received BPA at 5 or 50 mg/kg/day by oral gavage for 15 days, after which the passive avoidance test was performed. BPA was purchased from Sigma-Aldrich Co. and dissolved in normal saline as the vehicle.

3.2. Learning Procedure

Learning and memory were assessed using a passive avoidance learning and memory procedure. All animal procedures were reviewed and approved by the Institutional Research Ethics Committee of the School of Veterinary Medicine of Shiraz University (95INT1M1755).

3.3. Tissue Preparation

In the NL groups, 24 hours after the last BPA exposure, and in the L groups, 24 hours after completion of the passive avoidance learning sessions, the rats were anesthetized with sodium thiopental, and the hearts were perfused with 10% formaldehyde. The whole brain was then removed and washed with normal saline. The brains were fixed for 24 hours in 10% formaldehyde in 0.1 M phosphate buffer (pH 7.4), followed by postfixation in 4% formaldehyde in 0.1 M phosphate buffer (pH 7.4). Tissue processing was performed using an automated tissue processor, and paraffin blocks were prepared. Serial 5 μm sections of the amygdala, hippocampal CA1 region, and cerebellar Purkinje cell layer were prepared and mounted on 25% L-lysine-coated glass slides.

3.4. Immunohistochemistry

The slide-mounted sections were dried and then stored at -20°C until antibody labeling. After rewarming to room temperature, the sections were washed, delimited with a

hydrophobic marker, and incubated overnight with a primary antibody against serotonin 5-HT2 α receptors (dilution 1×10^{-3} ; Abcam, UK). The next day, the sections were rinsed with phosphate-buffered saline (PBS), incubated with an EnVision secondary antibody (Dako Co., Denmark), and washed again with PBS. Finally, the sections were rinsed in PBS 3 times for 10 minutes and reacted with 3,3'-diaminobenzidine (DAB) chromogen (Dako Co., Denmark). The sections were then counterstained with hematoxylin for nuclear staining. Negative-control slides were incubated with PBS without the primary antibody. [Figure 1A-C](#) shows representative negative-control images from the cerebellar Purkinje cell layer, BLA, and CA1 region. Receptor density was quantified from digital images using Image Analyzer (version 1.33), which calculates a value based on hue, saturation, and intensity. In this system, lower numerical values correspond to higher receptor density, whereas higher values indicate lower receptor density.

3.5. Statistical Analysis

Statistical analyses were performed using SPSS version 21. One-way analysis of variance (ANOVA) and Tukey post hoc tests were used to determine differences among groups. $P < 0.05$ was considered statistically significant, and data are presented as mean $\pm$ SEM.

4. Results

In cerebellar Purkinje cells, BPA exposure at 50 mg/kg/day significantly decreased serotonin 5-HT2 α receptor density compared with both the NL-sham group ($P < 0.05$) and the NL-BPA 5 mg/kg/day group ($P < 0.01$) ([Figure 2A](#)). In animals pre-exposed to BPA at 50 mg/kg/day, passive avoidance training was associated with a marked decrease in 5-HT2 α receptor density compared with both the L-sham and L-BPA 5 mg/kg/day groups ($P < 0.001$) ([Figure 2A](#)). Overall, passive avoidance learning increased 5-HT2 α receptor density in Purkinje cells. [Figure 2B](#) shows a photomicrograph depicting the density of 5-HT2 α serotonin receptors in cerebellar Purkinje cells across all groups. Exposure to BPA at 50 mg/kg/day resulted in a significant ($P < 0.05$) decrease in serotonin 5-HT2 α receptor density in the BLA nucleus compared with the NL-sham and NL-BPA 5 mg/kg/day groups ([Figure 3A](#)). In rats pre-exposed to 2 different doses of BPA, the passive avoidance task significantly reduced serotonin 5-HT2 α receptor density in the BLA nucleus compared with the L-sham group ($P < 0.01$). According to [Figure 3A](#), the passive avoidance task significantly ($P < 0.001$) increased the density of 5-HT2 α serotonin receptors in cerebellar Purkinje cells. [Figure 3B](#) illustrates a photomicrograph of

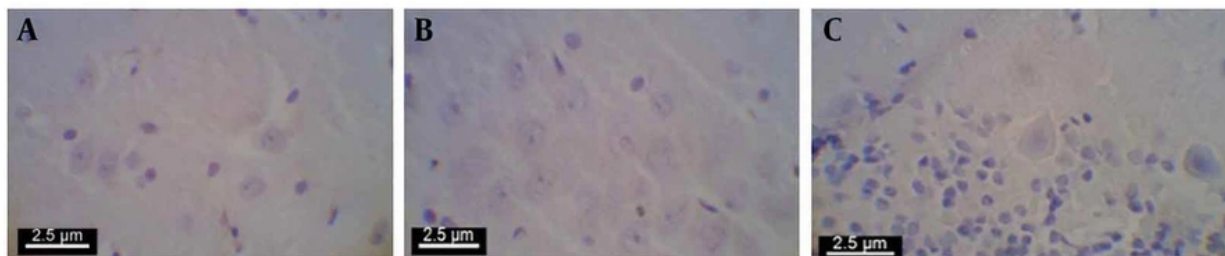


Figure 1. Negative-control photomicrographs for serotonin 5-HT α receptor density in brain regions: A, basolateral amygdala (BLA); B, hippocampal CA1 region; C, cerebellar Purkinje cell layer.

the density of 5-HT α serotonin receptors in the BLA nucleus across all groups.

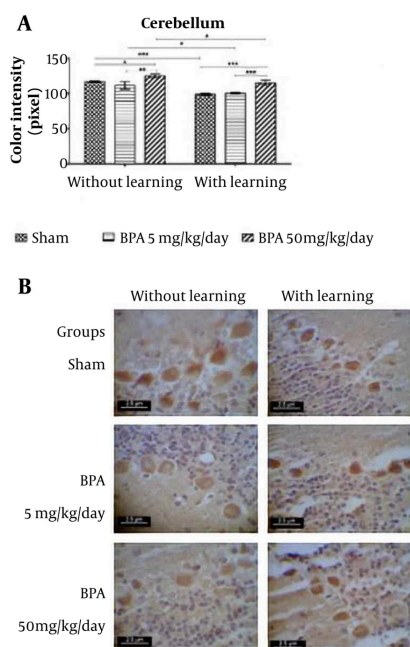


Figure 2. Serotonin 5-HT α receptor density in cerebellar Purkinje cells of male rats following bisphenol A (BPA) exposure and passive avoidance training. A, Color intensity of immunohistochemical staining in cerebellar Purkinje cells across groups. Numerical values are inversely related to serotonin 5-HT α receptor density: lower values indicate higher density and vice versa. B, Representative photomicrographs showing the effects of passive avoidance training on serotonin 5-HT α receptor density in cerebellar Purkinje cells of BPA-exposed male rats across groups. ** $P < 0.01$; *** $P < 0.001$. Data are presented as mean $\pm$ standard error of the mean (SEM).

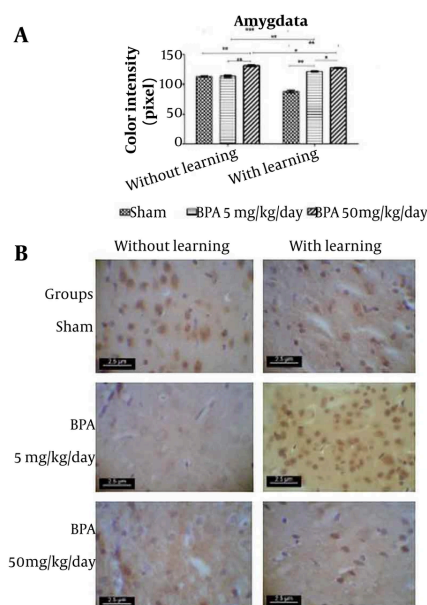


Figure 3. Serotonin 5-HT α receptor density in the basolateral amygdala (BLA) of male rats following bisphenol A (BPA) exposure and passive avoidance learning. A, Color intensity of immunohistochemical staining in the BLA across groups. Numerical values are inversely related to serotonin 5-HT α receptor density: lower values indicate higher density and vice versa. B, Representative photomicrographs showing the effects of passive avoidance learning on serotonin 5-HT α receptor density in the BLA of BPA-exposed male rats across groups. * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$. Data are presented as mean $\pm$ standard error of the mean (SEM).

Figure 4A shows that exposure to low ($P < 0.01$) and high ($P < 0.001$) doses of BPA significantly decreased serotonin 5-HT α receptor density in CA1 compared with the NL-sham group. However, comparison of L versus NL conditions indicated that passive avoidance training significantly increased overall 5-HT α receptor density in CA1 ($P < 0.001$).

Representative images of 5-HT₂α immunoreactivity in CA1 across all groups are presented in Figure 4B.

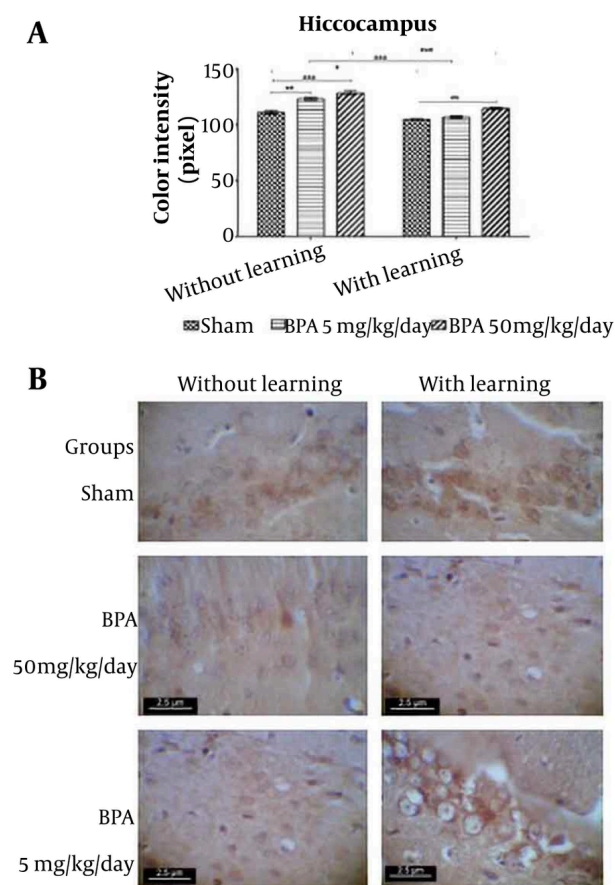


Figure 4. Serotonin 5-HT₂α receptor density in the hippocampal CA1 region of male rats following bisphenol A (BPA) exposure and passive avoidance training. A, Color intensity of immunohistochemical staining in the hippocampal CA1 region across groups. Numerical values are inversely related to serotonin 5-HT₂α receptor density: lower values indicate higher density and vice versa. B, Representative photomicrographs showing the effects of passive avoidance training on serotonin 5-HT₂α receptor density in the hippocampal CA1 region of BPA-exposed male rats across groups. *P < 0.05; **P < 0.01; ***P < 0.001. Data are presented as mean ± standard error of the mean (SEM).

5. Discussion

In our previous study, BPA at the same doses used in the present study impaired learning acquisition, memory consolidation, and memory retention (15). Studies of exogenous estrogen administration suggest that estradiol can facilitate the acquisition of acute avoidance behavior, although these effects depend on dose and timing (16).

In the present study, BPA at 50 mg/kg/day decreased 5-HT₂α receptor density in cerebellar Purkinje cells. Both BPA doses produced greater reductions in the NL-BPA groups than in the L-BPA groups, suggesting that passive avoidance training may counteract BPA neurotoxicity. Passive avoidance learning was associated with increased 5-HT₂α receptor expression in Purkinje cells.

Research has shown that serotonin plays a critical role in learning, memory, and other cognitive processes (17). BPA negatively affects learning and memory functions in the cerebellum (11, 18). The cerebellum plays a crucial role in activities related to motor learning (19).

At the molecular level, BPA can interact with estrogen receptors α and β, functioning primarily as a partial agonist (20). Previous studies indicate that BPA exposure alters serotonergic neurons in the cerebellum and other brain regions (21, 22). BPA exposure has been reported to decrease serum estradiol levels in some rodent studies under certain experimental conditions (23).

In the present investigation, BPA at 50 mg/kg/day decreased serotonin 5-HT₂α receptor density in the BLA nucleus. Relative to the L-BPA group, the NL-sham and L-sham groups had higher receptor density in the BLA, whereas the low-dose L-BPA group showed lower receptor density in this nucleus.

Serotonin 5-HT₂α receptors are commonly postsynaptic in cortical and limbic structures, although presynaptic expression has been demonstrated in regions such as cerebellar granule cells (24). Some investigations have shown that the serotonin 5-HT₂α receptor is also presynaptic, modulating glutamatergic, GABAergic, and dopaminergic neurons in a region-dependent manner (25). The effects of BPA on estrogen and serotonin concentrations in the BLA depend on the BPA dose, the life stage at exposure, and the serotonin receptor type.

The inhibitory action of serotonin may occur either directly through 5-HT₂α receptor activation on BLA neurons or indirectly via GABAergic interneurons expressing 5-HT₃ receptors (26). Therefore, in the present investigation, L-BPA at 5 mg/kg/day appeared to reduce serotonin 5-HT₂α receptor density more than NL-BPA at 5 mg/kg/day.

Both NL-BPA doses reduced 5-HT₂α receptor density in CA1. Similarly, L-BPA at both doses decreased receptor density in CA1, but the reduction was smaller than in the NL-BPA groups, implying that passive avoidance learning mitigates BPA toxicity. Thus, passive avoidance training ultimately led to higher 5-HT₂α receptor density in CA1 than under NL conditions.

Synapse formation and remodeling in the hippocampus are essential for memory acquisition and consolidation (27). Estradiol enhances spine density and

hippocampal plasticity, thereby supporting memory, whereas BPA can diminish or block these estradiol-dependent synaptogenic effects (5).

In the rat hippocampus, 5-HT_{2A} receptors are expressed on both excitatory and inhibitory neurons, where they may contribute to signaling through effects on different neuronal populations. The high level of 5-HT_{2A} receptor expression in the hippocampal region may indicate that this receptor plays a key role in functions including learning and memory (24). Evidence suggests that the production of monoamines, such as serotonin, is regulated through mechanisms mediated by estrogen receptors.

5.1. Conclusions

Passive avoidance learning appears to increase serotonin 5-HT_{2A} receptor density in BPA-exposed rats, particularly in the hippocampal CA₁ region, basolateral amygdala, and cerebellar Purkinje cells, suggesting a protective effect against BPA-induced serotonergic alterations. Although the neurotoxicity of BPA is well documented, the novelty of this study lies in demonstrating that passive avoidance learning may serve as a neuroprotective intervention by mitigating BPA-induced downregulation of 5-HT_{2A} receptor density across hippocampal, amygdala, and cerebellar circuits.

Footnotes

AI Use Disclosure: The authors declare that no generative AI tools were used in the creation of this article.

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Conflict of Interests Statement: The authors declare that there is no conflict of interest.

Data Availability: The data presented in this study are uploaded during submission as a supplementary file and are openly available for readers upon request.

Ethical Approval: All experimental protocols were performed according to the Ethical Committee for Animal Experiments at Shiraz University (Animal welfare code: 95INT1M1755).

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