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双酚 A 对小鼠肝脏和肾脏细胞的氧化损伤

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摘要: 为探究双酚 A(BPA)的氧化毒性,分别以剂量为 20、40 和 80 mg·kg^{-1}·d^{-1}的 BPA 对雄性昆明小鼠灌胃处理 1 周,并测定了小鼠体内活性氧自由基(ROS)水平、还原型谷胱甘肽(GSH)含量、丙二醛(MDA)含量和 DNA-蛋白质交联系数(DPC)。与对照组相比,各 BPA 暴露组小鼠肝脏和肾脏细胞中的 ROS 生成量、MDA 含量和 DPC 系数均升高,而 GSH 含量下降($P < 0.05$或 $P < 0.01$)。ROS 生成量、GSH 含量和 DPC 系数均显示出剂量-效应关系。研究表明,BPA 可扰乱小鼠肝脏和肾脏细胞的氧化应激平衡,诱导细胞氧化损伤。

关键词: 双酚 A; 昆明小鼠; 活性氧; 还原型谷胱甘肽; 丙二醛; DNA 蛋白质交联系数; 氧化损伤
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Oxidative Damage of Bisphenol A to Mouse Hepatic and Nephric Cells *in Vivo*

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Abstract: To investigate the oxidative toxicity of bisphenol A (BPA), Kunming male mice were fed daily with BPA at the doses of 20, 40 and 80 mg·kg^{-1}·d^{-1} for one week. The reactive oxygen species (ROS) level, glutathione (GSH) content, malondialdehyde (MDA) content and DNA-protein cross-linking (DPC) coefficient of hepatocytes and nephrocytes were detected to estimate the oxidative stress in the liver and kidney of male mice. Compared with the control, ROS generation, MDA content and DPC coefficient increased while GSH content decreased in BPA exposure groups ($P < 0.05$ or $P < 0.01$). ROS level, GSH content and DPC coefficient all showed dose-effect relationships. It is demonstrated that BPA could induce oxidative damage by disturbing the balance between ROS and antioxidant defense system in liver and kidney of mice.

Keywords: bisphenol A; Kuming mice; reactive oxygen species (ROS); glutathione (GSH); malondialdehyde (MDA); DNA-protein cross-linking coefficient (DPC); oxidative damage

Bisphenol A (Fig. 1) is a xenoestrogen that was first synthesized in 1891 [1-2]. Since the 1950s, BPA has been widely used as a synthetic monomer in the manufacturing of polycarbonate plastic, polystyrene resins and dental sealants. Then polycarbonates are further extensively used in food containers such as wa-

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ter bottles, plates and mugs [3]. According to the reports [4-5], the maximum intake of BPA into organism is up to 50 $\mu g \cdot kg^{-1} \cdot d^{-1}$ through the skin, respiratory and digestive tract. Roughly 6.5 billion pounds of BPA are produced each year. BPA is reported to be the major estrogenic compound that leaches into nearby water and food supplies [6]. Now BPA has been the subject of numerous risk assessment reviews, with increased attention over the last decade related to evaluating its potential for producing adverse health effects through an endocrine disruption mechanism [7].

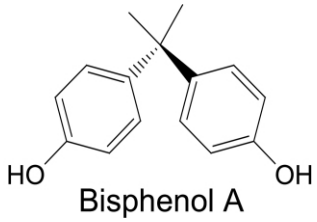

Fig. 1　Chemical structure of BPA

Normal metabolism of the body can produce reactive oxygen species (ROS) which are the chief culprit of human disease and aging due to their high oxidation potential [8-9]. Many studies have shown that the endocrine disrupting chemical BPA can lead to the elevation of transcription activity of ROS genes, and thus the levels of ROS will sharply increase [10]. The ROS produced by mitochondria in the organisms can be removed by some enzymes, such as glutathione (GSH) [11-12]. When the body's own antioxidant capacity reaches its limit, the excess of oxidizing substances will oxidize the cell macromolecules and the end product of lipid oxidation is malondialdehyde (MDA) which can cause polymerization and cross-linking of proteins, nucleic acids and other biological macromolecules [13]. The cellular toxicity of MDA can be monitored by measuring DNA-protein cross-linking coefficient (DPC). Therefore, intracellular ROS, GSH, MDA contents and DPC can be used as biomarkers of oxidative damage to detect the toxicity of BPA. At present, both in vivo and in vitro experiments have demonstrated that BPA can act as an endocrine disrupting chemical. However, the current studies mainly focused on reproductive effects of BPA. Besides their inherent effects on endocrine system, BPA is also known to inflict oxidative stress by affecting the redox status in the exposed organs [14]. The present research primarily aimed to estimate the oxidative toxicity of BPA. In this study, SPF class male Kunming mice which have many similarities to human beings in gene code were used to assess the toxicity of BPA.

1　Materials and methods

1.1　Reagents and apparatus

BPA (purity ≥99%), 5,5′-dithiobis(2-nitrobenzoic acid) (DTNB, purity ≥98%) and 2,7-dichlorohydrofluoroscein diacetate (DCFH-DA) were purchased from Sigma-Aldrich. Thiobarbituric acid (TBA) and Hoechst 33258 were purchased from Sinopharm Chemical Reagent Co. Ltd. of China. All the other chemicals were of analytical grade and purchased from Sinopharm Chemical Reagent Co. Ltd. of China.

Low-temperature refrigerated centrifuge (Eppendorf-5417R, Germany), continuous-wave and fluorescent microplate spectrophotometer (Bio-teck FLX800, America), and fluorescence spectrophotometer (Hitachi F-4500, Japan) were used in this experiment.

1.2　Experimental animals

SPF class male Kunming mice (weighing about 20-24 g) were purchased from the Center for Laboratory Animal Administration, Center for Disease Control and Prevention of Hubei Province (Wuhan, China). The 36 male mice were randomly divided into 4 treatment groups (n = 9 each, one control group and three BPA exposure groups). The mice of each group were isolated in separate cages at 27℃ with 45% – 49% relative humidity, and fed with normal diet and water.

1.3　Methods

1.3.1　Exposure design

BPA was dissolved in plant oil at the concentration of 2, 4 and 8 $mg \cdot mL^{-1}$. The mice in BPA exposure groups were fed intragastrically with 20, 40 and 80 $mg \cdot kg^{-1} \cdot d^{-1}$ BPA via dull needles for 7 days. Control group was fed with plant oil merely. The

time of gastrogavage was limited from 11:30 to 12:30 everyday.

1.3.2 Sample preparation

Kunming mice were sacrificed on the 8th day. Their livers and kidneys were separated quickly and washed with 0.01 mol·L^{-1} ice-cold phosphate buffer solution (PBS, pH 7.5). The homogenate was made by bag blender (using about 0.3 g tissue in 3 mL PBS). Centrifugate the homogenate at 10 000 r·min^{-1} at 4℃ for 10 min to obtain the supernatant. The rest samples were minced with ophthalmic scissors (1 mm^3 tissue) and the cell suspension was obtained after filteration through four layers of lens cleaning paper. The cell density was regulated to 10^7 cells·mL^{-1}.

1.3.3 ROS assay

The supernatant of homogenate was diluted 40-fold with PBS (pH 7.5), 100 μL of which was distributed into the 96-well microplate. 100 μL of 5 μmol·L^{-1} of DCFH-DA was added and mixed with the above supernatant. The above 96-well microplate was then kept in the dark for 10 min. Dichlorofluoroscein (DCF) is the oxidized product of DCFH-DA. The fluorescence of dichlorofluoroscein, which is directly proportional to the level of ROS in cells, was measured by the microplate spectrofluorometer at the excitation wave of 485 nm and emission wave of 525 nm [15].

1.3.4 GSH assay

The sulfhydryl group of GSH can react with DTNB and produce a yellow-colored 5-thio-2-nitrobenzoic acid (TNB) [16]. Measurement of the absorbance of TNB between 405 nm and 414 nm provides an accurate estimation of GSH content in the sample. The supernatant of homogenate was mixed with the organic solvent (V (trichloromethane) : V (butyl alcohol) =3:1), and then centrifuged at 10 000 r·min^{-1} for 5 min. 100 μL of the obtained supernatant and 100 μL of 60 μg·mL^{-1} DTNB solution were added to the 96-well microplate. After being kept in the dark for 5 min, the absorbance value of TNB was measured at 412 nm. The concentration of GSH was calculated according to the standard curve.

1.3.5 MDA assay

The MDA-TBA adduct that is formed by the reaction between MDA and TBA under high temperature (90 – 100℃) and acidic conditions can be measured colorimetrically at 530 – 540 nm [17]. 0.4 mL of supernatant of homogenate was mixed with 2 mL of 0.6% (V/V) TBA solution and incubated at 100℃ for 15 min. The treated samples were dissolved by 10% (V/V) trichloroacetic acid (TCA), then washed by tap water until cooling to room temperature, and centrifuged at 10 000 × g for 10 min. The absorbance value of the sample was measured at 450, 532 and 600 nm. The following equation [18] was used to calculate the concentration of MDA.

$$C = 6.45 \times (A_{532} - A_{600}) - 0.56 \times A_{450}$$

Where C (μmol·L^{-1}) stands for the concentration of MDA; A_{450}, A_{532} and A_{600} stand for the absorbance at 450, 532 and 600 nm, respectively.

1.3.6 DPC assay

After centrifugation at 1 000 r·min^{-1} for 5 min, the supernatant of the cell suspension was mixed with 0.5 mL of ice-cold PBS and 0.5 mL of 2% (W/V) sodium dodecyl sulfonate. 100 μL of 10 mmol·L^{-1} Tris-HCl-KCl (pH 7.4) was then added into the above solution, and the resultant mixture passed through a polypropylene pipette tip six times to favor shearing of DNA for a uniform length after being lyzed in the water bath at 65℃ for 10 min. After being cooled in the ice water for 5 min, the obtained sample was centrifuged at 10 000 r·min^{-1} for 5 min. The supernatant was moved into 5 mL centrifuge tube, while 1 mL of cleaning buffer (0.1 mol·L^{-1} KCl, 0.1 mmol·L^{-1} EDTA, 20 mmol·L^{-1} Tris-HCl, pH 7.4) was added into the remaining sediment to resuspend the cells. The above process was repeated for three times to collect 3 mL of the supernatant into 5 mL centrifuge tube. The supernatant was the unbound fraction of DNA.

The remaining sediment was then added with 0.5 mL of cleaning buffer and 0.5 mL of proteinase K. The resulting solution was diluted 250-fold with deionized water and digested in the water bath at 50℃ for 3 h. After being chilled in the ice water for 5 min, the

obtained sample was centrifuged at 1 200 r·min^{-1} for 5 min to collect supernatant. 1 mL of either the supernatant containing the unbound fraction of DNA or the supernatant containing the DNA previously involved in DNA-protein cross-linking was then mixed with 1 mL of fleshly prepared fluorescent dye Hoechst 33258. After being kept in the dark for 30 min, the absorbance value of the supernatant was measured by fluorescence spectrophotometer with excitation wave of 350 nm and emission wave of 450 nm.

DNA contents of the samples were determined quantitatively through a corresponding DNA standard curve generated from a set of calf thymus DNA. The DPC coefficient was measured as a ratio of the percentage of the DNA involved in DPC over the percentage of the DNA involved in DPC plus unbound fraction of DNA [19-20].

1.4 Statistical analysis

Data were analyzed using Origin 6.0. Student's t-test was conducted to analyze statistically significant differences between exposure groups and control group. $P < 0.05$ and $P < 0.01$ were considered as significant difference and extremely significant difference.

2 Results

After 7 d exposure of different doses of BPA in vivo, the ROS level, GSH content, MDA content and DNA-protein cross-linking coefficient of mouse hepatic and nephric cells were shown in Fig. 2 – Fig. 5.

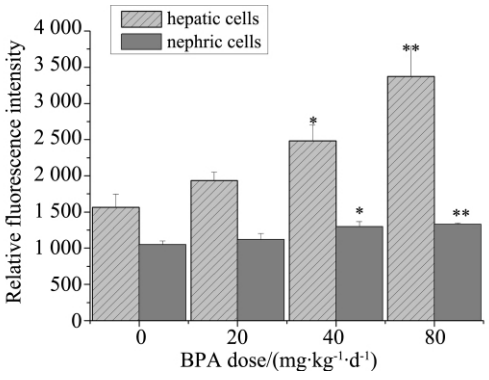

Fig. 2　Effects of BPA exposure on ROS levels in mouse hepatic and nephric cells

Note: *, ** denotes $P < 0.05$, $P < 0.01$, compared with control group.

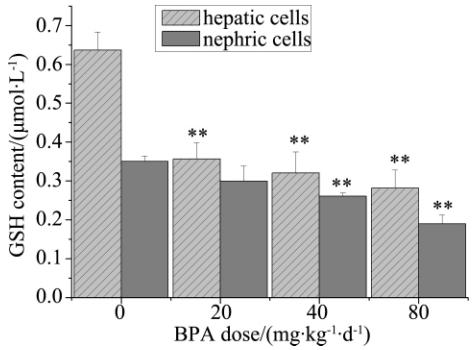

Fig. 3　Effects of BPA exposure on GSH contents in mouse hepatic and nephric cells

Note: *, ** denotes $P < 0.05$, $P < 0.01$, compared with control group.

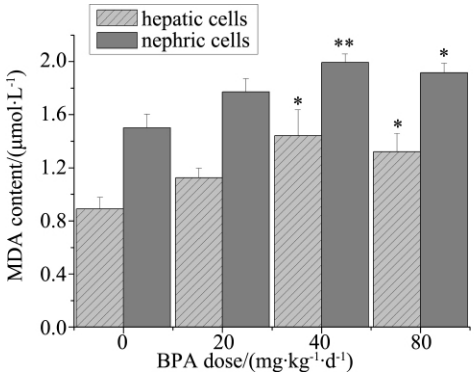

Fig. 4　Effects of BPA exposure on MDA contents in mouse hepatic and nephric cells

Note: *, ** denotes $P < 0.05$, $P < 0.01$, compared with control group.

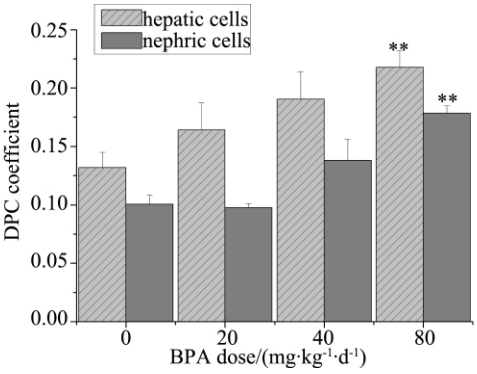

Fig. 5　Effects of BPA exposure on DPC coefficients in mouse hepatic and nephric cells

Note: *, ** denotes $P < 0.05$, $P < 0.01$, compared with control group.

2.1 Generation of ROS

ROS generation increased with increasing BPA dose. Compared with the control group, ROS levels sig-

nificantly increased in 40 mg·kg^{-1}·d^{-1} and 80 mg·kg^{-1}·d^{-1} dose groups ($P < 0.05$ or $P < 0.01$) (Fig. 2).

2.2 Content of GSH

As shown in Fig. 3, compared with the control group, the GSH contents of hepatic cells in all BPA exposure groups significantly decreased ($P < 0.01$), while the GSH contents of nephric cells in 40 mg·kg^{-1}·d^{-1} and 80 mg·kg^{-1}·d^{-1} exposure groups significantly decreased ($P < 0.01$).

2.3 Content of MDA

Fig. 4 demonstrates that the MDA contents in both hepatic and nephric cells in exposure groups roughly increased with increasing BPA dose. Though the MDA content of 80 mg·kg^{-1}·d^{-1} exposure group was slightly lower than 40 mg·kg^{-1}·d^{-1} group, the MDA contents of the two groups were both significantly higher than that of the control group ($P < 0.05$ or $P < 0.01$).

2.4 DNA-protein cross-linking coefficient

Both in hepatic and nephric cells, the DPC coefficients roughly enhanced with increasing BPA dose. There were significant differences between 80 mg·kg^{-1}·d^{-1} exposure group and control group ($P < 0.01$) (Fig. 5).

3 Discussion

Mammalian cells are equipped with both enzymatic and nonenzymatic antioxidant defense mechanism to reduce the cellular damage resulting from interaction between lipid, protein and DNA molecules and ROS [21]. Despite the presence of this antioxidant system, the overproduction of ROS in both intra- and extracellular space often occurs upon exposure of cells or individuals to certain chemicals, radiation and hyperoxia [22]. In other words, an unbalanced production of ROS in cells has been postulated to play a role in the pathogenesis of a number of clinical disorders [23].

In our study, BPA entered the blood circulation of mice through digestive system and then reached the liver and kidney. A rapid increase of ROS levels in hepatic and nephric cells was observed in BPA exposure groups compared with control group. A series of cleaning mechanism such as GSH was activated in these organs for detoxification and expulsion of toxins [24]. As a nonenzymatic antioxidant against ROS, GSH contents of mice in BPA exposure groups then correspondingly decreased. MDA that is the end product of lipid oxidation was detected to increase with exposure doses of BPA. MDA can result in polymerization and cross-linking between DNA and protein [25-26]. Higher DPC coefficients were found in exposure groups with higher BPA doses. The variation of data of the four biomarkers well described the mechanism of oxidative damage induced by BPA.

BPA (at low dose of 20 mg·kg^{-1}·d^{-1}) was expected to merely induce limited ROS. GSH activated in cytoplasm could then clear the excessive ROS. Therefore, oxidative damage was avoided or inhibited in mice of exposure group with low BPA dose. However, in exposure groups with higher BPA doses (40 and 80 mg·kg^{-1}·d^{-1}), such a large amount of ROS was induced that it could not be cleared by GSH completely. The excessive ROS could oxidize the lipid of cell membrane, which directly led to the increase of MDA levels. The excessive MDA would further cause polymerization and cross-linking between DNA and protein. Eventually, cell oxidative stress and apoptosis was induced.

Moreover, it is a little beyond our expectation that MDA content of mice in 80 mg·kg^{-1}·d^{-1} group was slightly lower than that of 40 mg·kg^{-1}·d^{-1} group. This might be due to the following reasons: MDA is a sensitive biomarker of oxidative damage and can be influenced by a series of complex factors. At high exposure doses, the body physical function declined and the lipid content was remarkably different from that of the normal organisms. The dose-effect rule could then hardly be followed strictly.

In summary, our study demonstrated that BPA could induce cell oxidative stress and apoptosis especially at dose higher than 40 mg·kg^{-1}·d^{-1}. ROS level, GSH content and DPC coefficient all showed dose-effect relationships between measured values and BPA

exposure doses.

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新书介绍

1.《二氧化硫生物学: 毒理学、生理学、病理生理学》

该书是孟紫强教授及其学生历经3年完成的一本专著,已于2012年5月由科学出版社出版,定价98元。该书是作者20余年来对SO_2及其衍生物——亚硫酸盐、亚硫酸氢盐和焦亚硫酸盐在毒理学、病理生理学及生理学方面的重要研究成果的系统性总结和升华,同时也对国内外其他学者在本领域的重要研究成果进行了介绍。全书共计19章,内容包括: 硫的生命必需性与硫循环、SO_2生物学研究历程与概况、SO_2的吸收与代谢、SO_2及其衍生物的物理和化学性质、一般毒性与流行病学、细胞生物学与病理形态学、细胞遗传毒理学与三致作用(突变、畸变与癌变)、生物化学与分子毒理学、病理生理学与细胞因子、生理学与信号分子作用、以及其对植物的生物学作用等方面的最新理论和研究成果。该书力求对SO_2及其衍生物的理化性质和生物学作用进行全面论述,希望读者能够应用这些研究成果去解决有关生物学、医学和及环境保护问题,并对他们的相关科学研究有所裨益。该书适于从事生物学、医学、环境科学和环境管理等方面的专业人员以及相关专业的大学生、研究生、教师和科研人员等阅读参考。

2.《沙尘暴医学与毒理学》

该书是孟紫强教授及其学生张全喜、杨振华共同完成的一本专著,已于2012年5月由中国环境科学出版社出版,定价35元。该书对沙尘暴的发生、传输及理化特性作了简要介绍,对浮尘天气、扬沙天气,特别是沙尘暴天气对健康影响的流行病学调查及其致病原因进行了重点论述,对沙尘细颗粒物毒理学作用及其与疾病的剂量-效应关系的研究作了详尽论述。因此,该书对于理解和解决沙尘暴及其他沙尘天气引发的健康问题,以及对于防止沙尘天气危害的科学对策的制定等均有参考价值。