Egypt. J. Vet. Sci. **Vol. 56,** No. 3, pp. 469-481 (2025)

Egyptian Journal of Veterinary Sciences
https://ejvs.journals.ekb.eg/

Histological Effect of Diphenol-A in Eyes of Chicken Embryos

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Abstract

DIPHENOL-A (bisphenole-A) can leach into meals and beverages from containers containing Diphenol A. Diphenol A exposure is concerning due to the potential consequences on the brain and organs of fetuses, babies, and children. To be assess the teratological and histopathological changes in the eyes of chick embryos that exposure to Diphenol we used number of 150 fertilized chicken eggs divided into 6 groups (Diphenol given in the second day of incubation, injected in to the air sac). G1, Negative control group chicks embryos without any exposure. G2, Positive control treated with corn oil. G3, treated with 0.0125 µL /egg Diphenol. G4, 0.025 µL /egg Diphenol. G5, 0.05 µL /egg Diphenol. G6, 0.1 µL /egg Diphenol. The chick embryos was euthanized at a developmental stage of (21) day. The study examined eye tissues for histopathological changes, revealing a range of abnormalities in different groups in subsequent treatment groups (G3 to G6), pathological alterations were identified. Groups G3 to G6 showed blood vessels, hemorrhagic foci, moderate degeneration in nerve fiber layers, edema in the nuclear fiber layer, and severe degenerative changes in the lamina pectoris, fibrous layer cells, and rods and cones layer of the retina. The study concluded that Diphenol may have teratological and histological abnormalities in the eyes of chicken embryos and that warrant further investigation.

Keywords: Diphenol-A, Teratological, histological changes, Chicken embryos.

Introduction

Diphenol A compounds (BPA), an endocrine-disrupting chemical commonly produced and utilized in the plastics industry, pollutes the environment and is absorbed into the body via food and beverages in polycarbonate containers. According to Joao *et al*. (2020) [1], 359 million tons of plastics were produced worldwide, with 40% allocated to packaging, and 100 tons of BPA per year can end up in the environment due to plastic decomposition, resulting in concentrations ranging from 5 to 21 g/L in environmental waters . Higher amounts of BPA have been found in studies near treated wastewater plants or landfills [2; 3]. BPA pollution gets into rivers and seas, presumably as a result of... plastic container migration from industrial waste dumps [4]. The effect of Diphenol A (BPA) on the eyes of developing chicken embryos has garnered significant attention in recent years due to concerns about the potential adverse impacts of this widely used industrial chemical on embryonic development and overall health [5].

BPA is a synthetic compound found in various consumer products, including plastics, food containers, and thermal paper receipts. Its ability to mimic estrogen in the body has raised questions about its safety, particularly during critical stages of embryogenesis [6; 7]. To evaluate the developmental toxicity of BPA, model Xenopus laevis embryos were treated with varying concentrations (0.1, 1, 10, and 20 M) of BPA at the two-cell stage, according to one study.

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(Received 29/02/2024, accepted 24/04/2024)
DOI: 10.21608/EJVS.2024.273731.1884

Embryonic growth and behavior were tracked from 24 to 96 hours following BPA exposure. BPA doses above 1 µL were found to be teratogenic in Xenopus embryos, resulting in a short tail axis, a defective gut, and a bent notochord as the principal abnormalities. Embryos exposed to 20 M BPA were substantially injured on both sides, exhibiting deformity, decreased behavioral ability, and tissue damage. Xenopus embryo DNA integrity and apoptosis were also studied [8].

Understanding the impact of BPA exposure on the eyes of chicken embryos serves as a valuable model for assessing its potential effects on ocular development in both avian and mammalian species, including humans [9]. The embryonic chicken eye shares structural and developmental similarities with the human eye, making it a relevant and informative subject for such investigations.

Effects of prenatal and lactation exposure to low doses of BPA on brain development in rats. This study demonstrated that prenatal exposure to BPA affected neocortical development in mice by accelerating neuronal differentiation/migration during the early embryonic stage, which was associated with up- and down-regulation of genes important for brain development [0]. It underscores the importance of research in this area, given the potential consequences for eye health and development, and the potential relevance of these findings to human health and environmental concerns [11]. To assess the histopathological changes in the eyes of chick embryos resulting from exposure to Diphenol this study is designed.

Material and Methods

Experimental Design

Diphenol

Diphenol powder was purchased from Japan's Solarbio Beijin a company. Diphenol dissolved in corn oil and then four concentrations from it was used (0.0125, 0.025, 0.05 and 0.1) µL [12].

Ethical Considerations

The experiment adhere to ethical guidelines for animal research, ensuring the welfare of the chick embryos and compliance with ethical standards at No. UM.MED.048.

Experimental Groups

Number of 150 fertilized chicken eggs divided into 6 groups, placed in an incubator under controlled conditions of temperature and humidity. Eggs rotated regularly to prevent adhesion of the embryo to the shell membrane according to the following groups: 1.G1, Negative Control Group: Chicks embryos without any exposure; 2. G2, Positive Control Group: Chicks embryos with corn oil; 3. G3, 0.0125 µL /egg Diphenol injected in to the air sac; 4. G4, 0.025 µL /egg Diphenol injected in to the air sac; 5. G5, 0.05 µL /egg Diphenol injected in to the air sac and 6. G6, 0.1 µL /egg Diphenol injected in to the air sac.

Diphenol Exposure

The solution of Diphenol prepared at the of concentration (0.0125, 0.025, 0.05, and 0.1) µL/Egg, a deferent concentration of Diphenol solution prepared and injected into the air sac of each egg using a sterile syringe and needle (On the second-day of egg incubation). The positive control group eggs will undergo the same procedures as the experimental group, except they will be exposed to a control solution without Diphenol (corn oil) and negative control handled with the same procedure without any exposure.

Incubation Continuation

-Eggs carefully sealed after treatment and returned to the incubator. Incubation continue until the desired developmental stage is reached for the evaluation of histopathological changes. Chick embryos euthanized at a specific developmental stage (21) day. The embryos will be sacrificed, and the eyes carefully dissected.

-The dissected eye tissues will be fixed in appropriate histological fixatives for a specified period.

Histopathological Analysis

Eye tissues fixed in formalin for histopathological processing. Tissue samples embedded in paraffin blocks, sectioned, and stained with hematoxylin and eosin (H&E) stain. Histopathological slides examined under a light microscope by a qualified pathologist. Histopathological changes such as cellular morphology, tissue architecture, inflammation, and any abnormalities recorded and analyzed.

Data Analysis: The histopathological findings quantified and compared between the control and experimental groups.

Results

The histopathological examination of histological slides revealed a range of pathological changes in different groups. In the control group (G1 and G2), (Fig. 1,2 and 3) normal histological structures of chicken embryo eyes were observed, including a healthy conjunctival membrane, normal cornea, iris, ciliary body, sclera, well-preserved retinal layers, a normal vascular pectinic plate, and normal-sized aqueous and vitreous spaces. However, in subsequent treatment groups (G3 to G6), various pathological alterations were identified. Group (G3) (Figs 4,5, 6,7,8 and 9) exhibited blood vessel under the epithelial pigment cell layer and the ciliary body. Olso showing petechial hemorrhagic foci were observed in the ocular choroid layer, along with slight vacuolization in the ganglion cell layer of the retina and subretinal edema. Group (G4) (Figs.11,15,16), displayed moderate degeneration in the nerve fiber layer in addition to the G3 findings. Further progression of pathological changes was noted in-group (G5) (Figs. 12, 13, 14, 16, 17, 18), including edema in the nuclear fiber layer and slight edema between photoreceptor neurons. The most severe changes were observed in group (G5), characterized by severe degenerative changes and vascular congestion in the lamina pectoris, vacuolization in fibrous layer cells covering the lamina pectoris, and vacuolization and focal degeneration in the rods and cones layer of the retina. In the sixth treatment group (G6) (Figs. 19, 20 and 21), widespread vacuolar degenerative changes were observed across all retinal layers, along with focal edema in the rods and cones layer and the presence of hemorrhagic foci under the fibrous layer covering the lamina pectoris, accompanied by vacuolar degeneration in these cells.

Discussion

The histopathological findings from this study provide valuable insights into the impact of Diphenol exposure on the developing eyes of chicken embryos across different treatment groups (G3 to G6) compared to the negative and postive control group (G1, G2). In the negative and positive control group, the normal histological structures of chicken embryo eyes were well-preserved, serving as a baseline for comparison.

When it comes to groups that have been exposed to Diphenol, it appears that the severity of pathological changes is related to the dose. As our results showed that the severity of histopathological changes is related to the value of the dose given, as the two highest doses of 0.1, 0.05 M/Egg showed the most severe histopathological effects when compared to the lower doses. These findings are congruent with those of another study. This outcome has been established [13. The findings suggest a dose-dependent relationship between Diphenol exposure and the extent of histopathological changes in the developing embryo eyes.

This result is similar with prior research on zebra fish embryos, it was observed that the use of a low dose of BPA impacted the heart rate and caused the body and head of the larvae to lengthen. Quantitative morphometric and histological research demonstrated that BPA exposure affected the angle and length of craniofacial cartilage elements and disturbed chondrocytes. BPA damages pharyngeal cartilage through numerous cellular processes. These findings show that BPA interferes with the normal development of cartilage and cranial structures in zebrafish embryos [12].

The observed circulatory abnormalities, hemorrhagic foci, edema, and degenerative modifications in several retinal layers suggest that Diphenol exposure may have a negative impact on ocular development. More research is needed to understand the underlying mechanisms and the long-term effects of such exposure, which may have implications for understanding the influence of Diphenol on eye development in both avian and mammalian species, including humans [14].

The effects of Diphenol exposure on embryonic eye tissue, as seen in this study's histopathological findings, suggest that it may have a negative impact on eye development. It has been claimed that early xenoestrogen exposure, such as BPA, may be the underlying cause of the increased frequency of infertility, genital tract abnormalities, and breast cancer observed in European and US human populations over the last 50 years [15; 16].

Diphenol exposure, particularly at higher dosages (G3 to G6), caused circulation

abnormalities in the ocular tissue. This comprised blood vessel congestion, as demonstrated by ciliary body and pectin plate congestion in G3 and petechial hemorrhagic foci in the ocular choroid layer. BPA also inactivates hypoxia-inducible factor-1 (HIF-1), a master transcription factor that regulates erythropoiesis, angiogenesis, and glycolytic pathways during hypoxia, by binding to the heat shock protein, HSP90, and destabilizing HIF-1, promoting its proteasomal degradation [17- 20].

Hemorrhagic events within the eye tissue were observed in some therapy groups, including G3, and G6. This is problematic since hemorrhages can impair normal ocular function and structure. It could be explained by increased intracranial and venous pressure [21].

Edema, or fluid collection in tissues, was seen in numerous therapy groups, including G3, G5, and G6. Edema under the retina, subretinal edema, macular edema, and edema in the nuclear fiber layer all indicate an abnormal accumulation of fluid, which can impair eye function. Interstitial fluid may be caused by Müller cells in the retina, and may also be caused in some ways by bulk flow in the parenchyma, which is controlled by astrocytes. The absence of blood flow in the plexus may lead to blood flow directly into the capillaries, including blood flow through Müller cells [22]. Degenerative changes were observed in the nerve fiber layer (G4 and G5), pigmented cell layer (G4), and photoreceptor layer (G4).

These degenerative changes may indicate cellular damage and dysfunction within the eye tissue. Inflammation is a biological response to events that disrupt cell and tissue homeostasis. TLRs, inflammatory receptors, and complement components, for example, trigger complex cellular cascades by identifying or sensing disease-causing and damage-associated chemical patterns, respectively.

Vacuolization, or the creation of empty spaces among cells or tissues, was found in G3, G5, and G6 layers of the retina. This suggests that there has been cellular damage and structural alterations within the eye.

One study on the inflammatory and degenerative effects of the eyes discovered that bacteria and debris derived from degenerating cells become sequestered between the retinal pigment epithelium basal lamina and Bruch›s membrane, that this cellular debris decorates a chronic inflammatory stimulus, and that this cellular debris is an interesting site for the «nucleation» of speckle formation. Cell debris that has become trapped becomes a target for encapsulation by a number of mediators [23,24].

There appears to be a link between Diphenol dose and the severity of pathological alterations. Higher doses (G3 to G6) had more apparent and severe effects on eye tissue than lower ones (G3).

Effects of prenatal and lactation BPA exposure on brain and eye development in rats that prenatal BPA exposure affected neocortical development in mice by accelerating neuronal differentiation/migration during the early embryonic stage, which was associated with up- and down-regulation of brain development genes [25, 26]. These findings imply that Diphenol exposure during embryonic development can be harmful to ocular tissue, potentially altering normal development and function. It emphasizes the importance of knowing the possible dangers of Diphenol exposure, particularly in the setting of eye development, and may have implications for future study on the influence of Diphenol exposure on eye health in both animal models and people.

Conclusion

The histopathological analysis underscores the progressive nature of the pathological changes induced by Diphenol. From initial vascular alterations to severe degenerative changes affecting multiple retinal layers, the findings highlight the deleterious impact of Diphenol on ocular morphology and function. These observations suggest a need for further investigation into the underlying mechanisms and potential therapeutic interventions to mitigate such pathological effects on ocular tissues.

Conflicted interest

None

Acknowledgment

The researchers express their gratitude and appreciation to everyone who participated to the completion of this research, as well as the support provided by the University of Mosul.

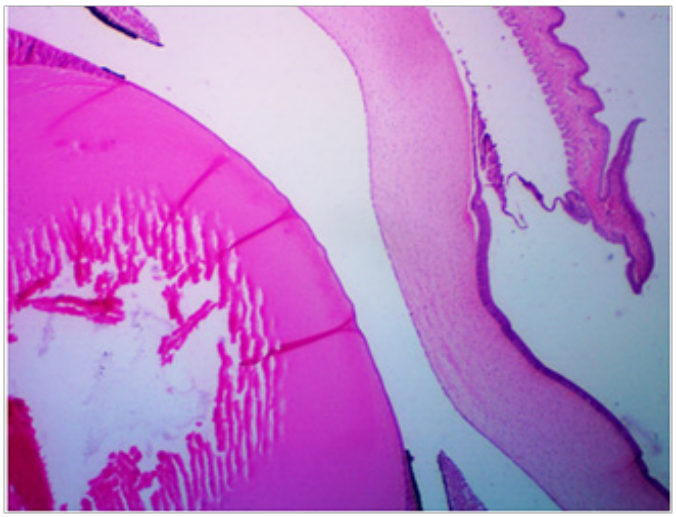

Fig.1. of the cornea with the H&E stained intraocular lens. Standard ruler 100 micrometers. Magnification power 40X. .X

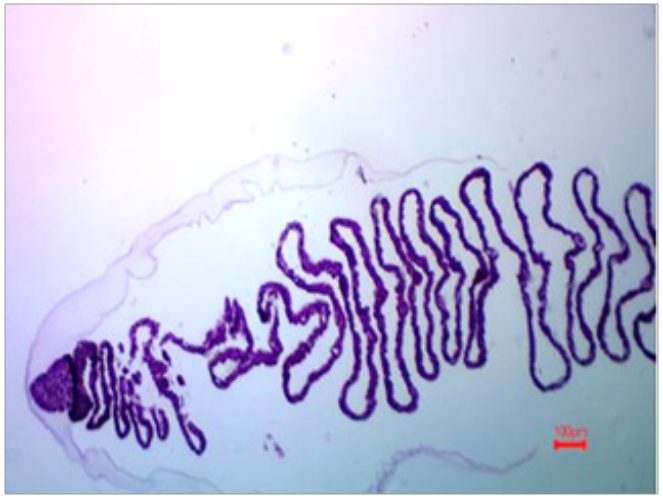

Fig.2. Showing the pectin body. H&E dye. Standard ruler 100 µm. Magnification power 40X.

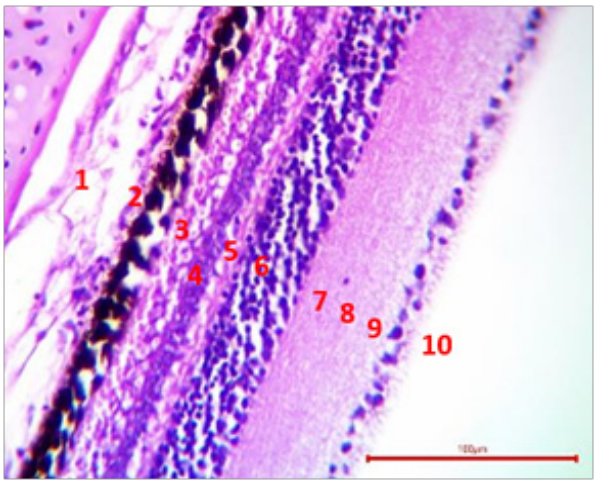

Fig.3. Showing the retina with all its layers with the conjunctiva stained H&E. Standard ruler 100 µm. Magnification power 400 X.

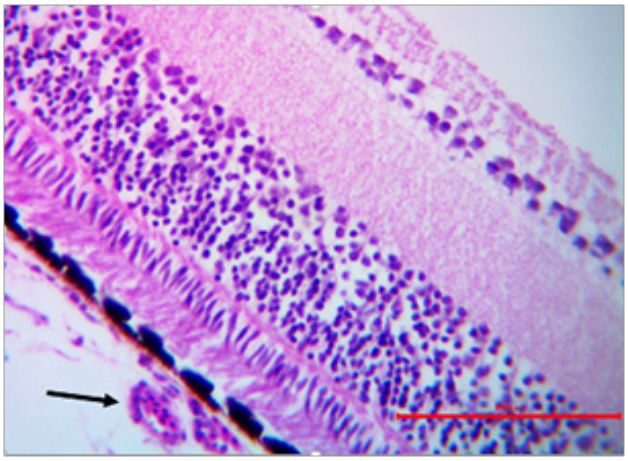

Fig. 4. Shows congestion of blood vessels under the layer of pigment epithelial cells (arrow). H&E tint. Standard ruler. 100 µm. Magnification power. 400X.

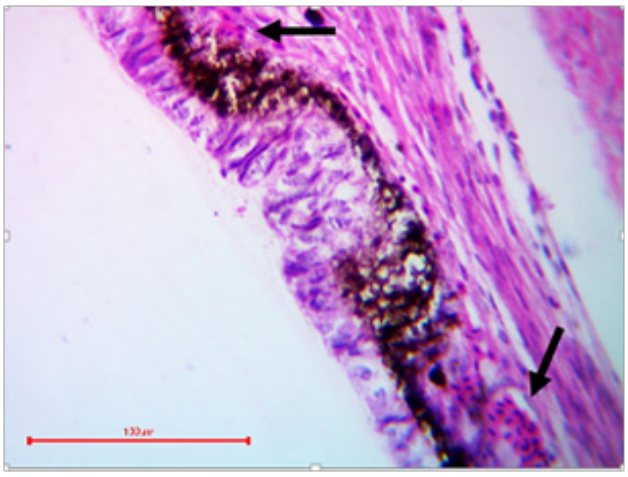

Fig. 5.Shows minor bleeding in the ciliary body (arrows). H&E stain. Standard ruler 100 µm. Magnification power 400X.

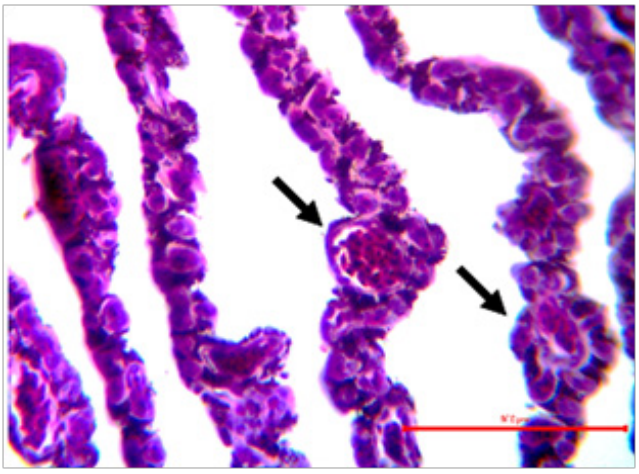

Fig.6. shows congestion of blood vessels in the pectin plate (arrows). H&E tint. Standard ruler. 100 µm. Magnification power. 400X.

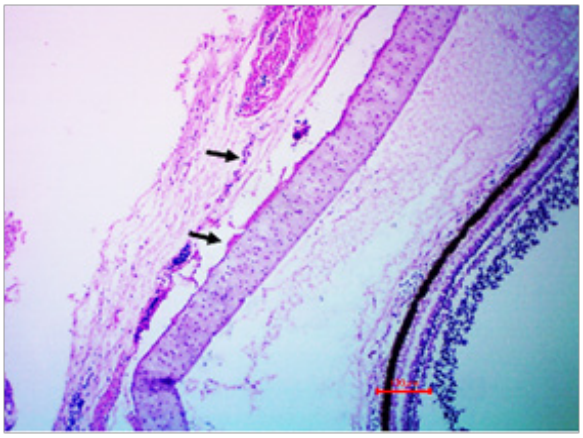

Fig.7. Shows the presence of petechial hemorrhagic foci observed in the choroid layer of the eye. H&E stain. Standard ruler 100 µm. Magnification power 40.

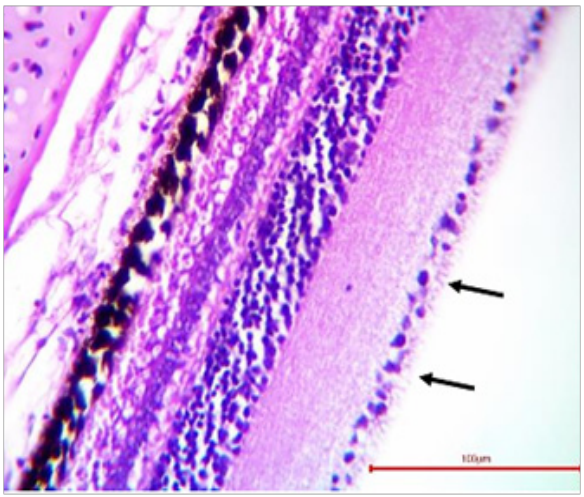

Fig.8. A slight vacuole in the ganglion cells layer in some areas of the retina. H&E stain. Standard ruler 100 µm. Magnification power 400 X.

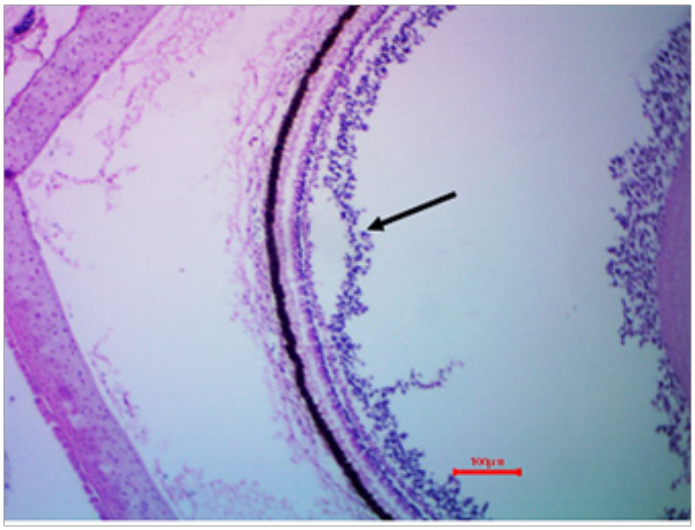

Fig. 9. The presence of edema under the retina and in the ophthalmic nerve disc. H&E stain. Standard ruler 100µm. 40x magnification

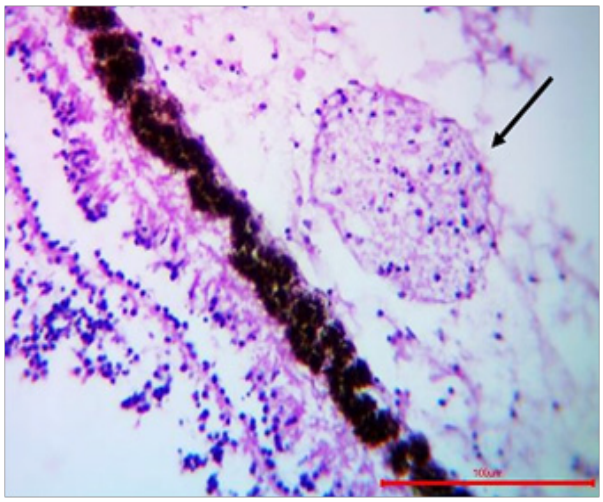

Fig.10. Nodules were observed in the axonal bundles of the ophthalmic nerve. Tint H&E Standard ruler 100 µm Magnification power 40.

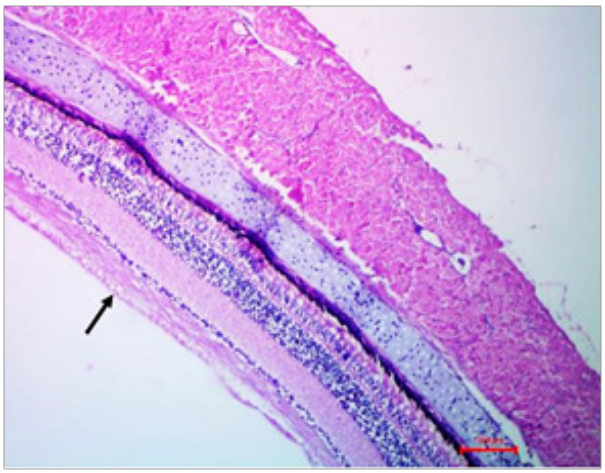

Fig.11. Represented by the presence of moderate degeneration in the layer of neurons. H&E tint. Standard ruler. 100 µm. Magnification power. 40X

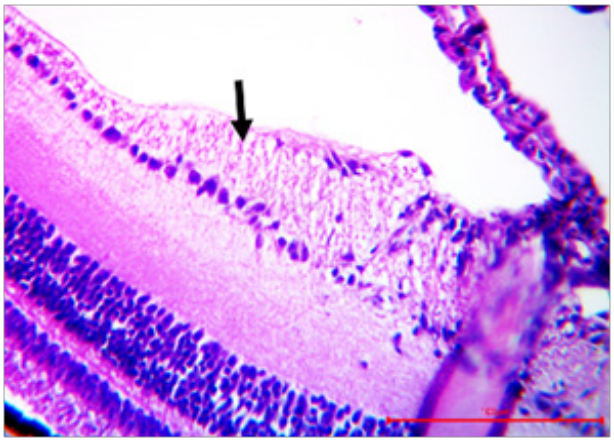

Fig.12. Shows the presence of edema in the layer of nuclear nerve fibers. H&E stain. Standard ruler 100 µm. Magnification power 400.X

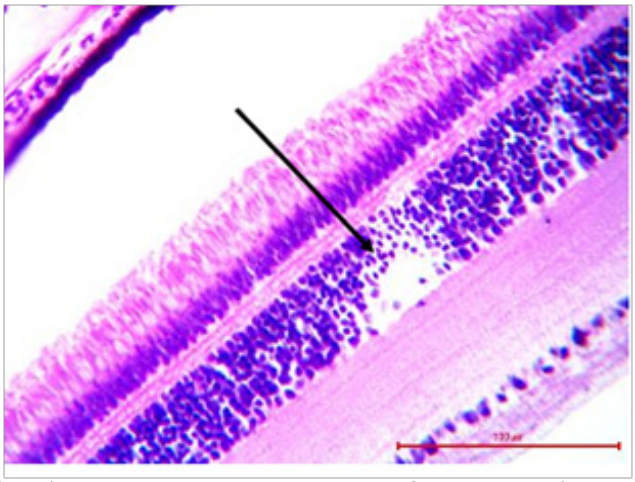

Fig.13. The presence of a slight edema between the layer of photoreceptive nerve fibers in the retina. H&E stain. Standard ruler 100 µm. Magnification power 400.X

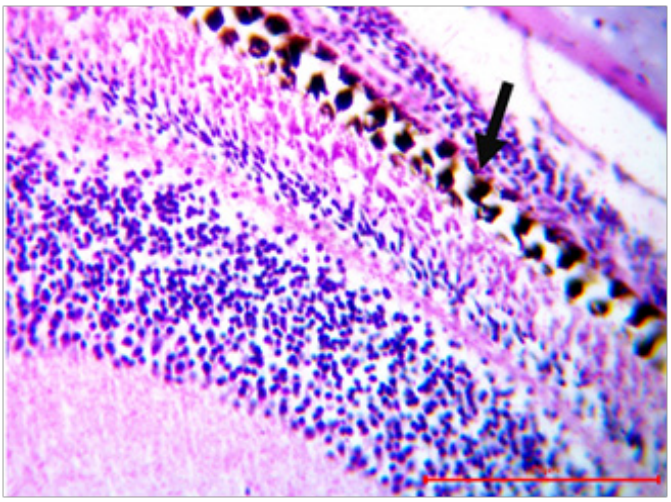

Fig.14. Shows moderately severe local degenerative changes in the pigmented cell layer (arrow) of H&E. Standard ruler. 100 µm magnification power X 400.

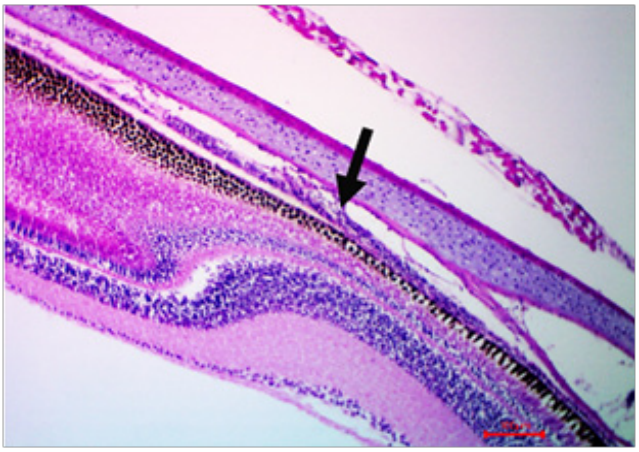

Fig. 15. The layer of photoreceptors known as the rods and cones layer. (arrow) H&E stain. Standard ruler 100. µm. Magnification power 40.

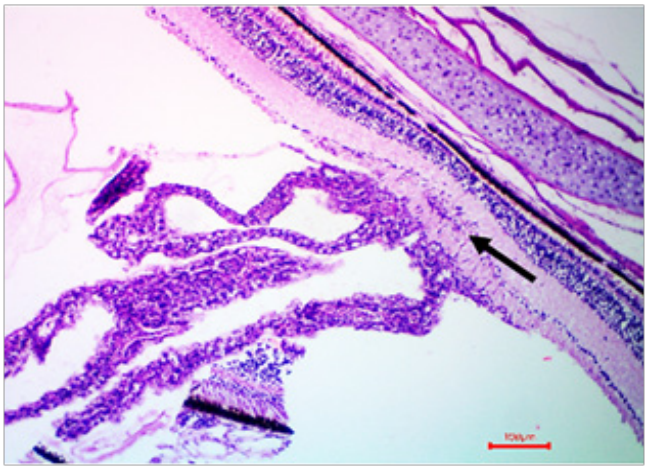

Fig.16. Shows severe degenerative changes and vascular congestion in the lamina pectoris (Pectin oculi) (arrow). H&E dye. Standard ruler 100. µm magnification power 40.X

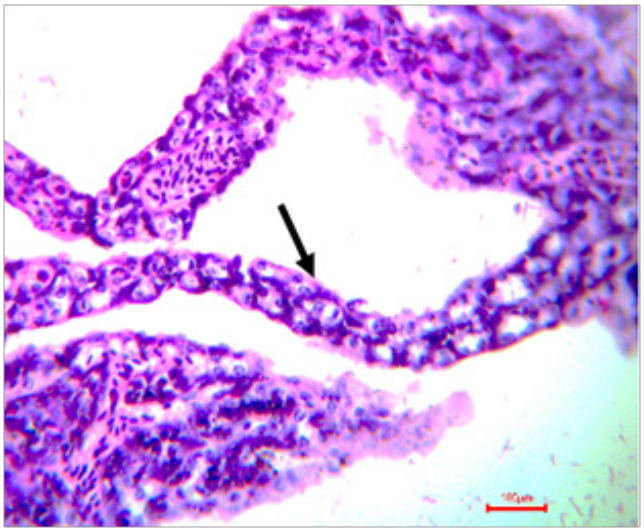

Fig.17. Vacuoles in the cells of the neural fibrous layer covering the pectin lamina. H&E stain. Standard ruler 100 µm. Magnification power 400.X

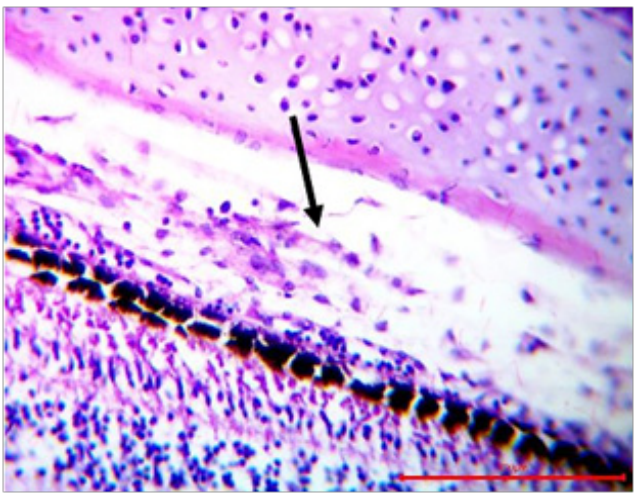

Fig. 18. Shows vacuolization and focal degeneration of the rods and cones layer in the retina. (arrow). H&E standard ruler. 100. µm tint. 400.X magnification power.

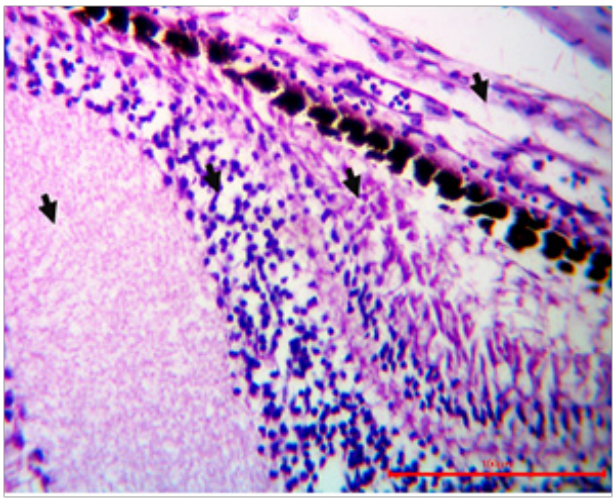

Fig. 19. The presence of vacuolar degenerative changes in all layers of the retina with edema. (arrows). H&E stain. Standard ruler. 100 µm. Magnification power 400.X.

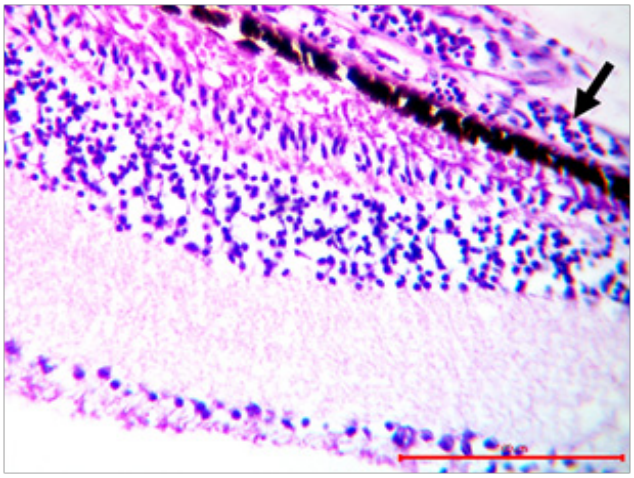

Fig.20. Focal edema in the layer of rods and cones. (arrow). H&E standard ruler. 100. µm tint. 400.X magnification power.

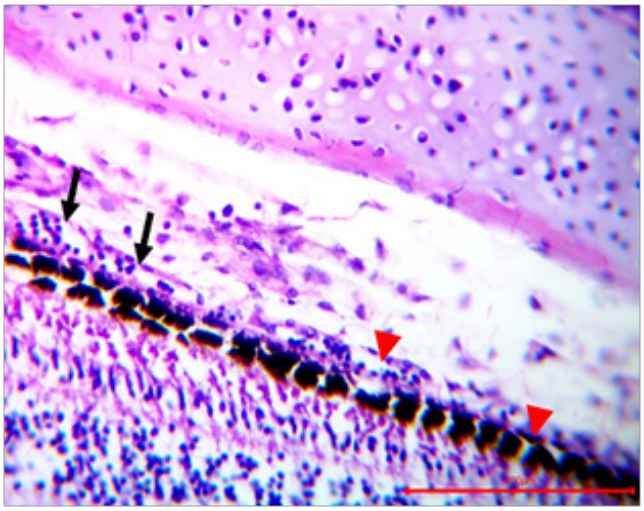

Fig. 21. Shows the presence of hemorrhagic foci under the neurofibrous layer covering the lamina pectoris (arrows) and the presence of vacuolar degeneration in the cells of this layer itself (arrowhead). H&E tint. Standard ruler. 100 µm. Magnification power 400.X

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التأثيرات النسيجية للدايفينول- أ في عيون أجنة الدجاج

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الخلاصة

يمكن أن يتسرب البيسفينول -أ إلى الوجبات والمشروبات من الحاويات التي تحتوي على البيسفينول. إن التعرض للبيسفينول-أ يثير القلق بسبب العواقب المحتملة على الدماغ وأعضاء الأجنة والرضع والأطفال. تقييم هذه الدراسة التغيرات المسخية والنسيجية المرضية في عيون أجنة الكتاكيت الناتجة عن التعرض لمادة البيسفينول. اخذت 150 بيضة دجاج مخصبة قسمت إلى 6 مجاميع (اعطي البيسفينول في اليوم الثاني من الحضانة، الحقن في كيس الهواء). G1، أجنة مجموعة السيطرة السلبية دون أي تعرض. G2، أجنة السيطرة الإيجابية المعاملة بزيت الذرة. G3، 0.0125 µL / بيضة بيسفينول. G4، 0.025 µL / بيضة بيسفينول. G5، 0.05 µL / بيضة بيسفينول. G6، 0.1 µL / بيضة. تم اجراء القتل الرحيم لأجنة الكتاكيت في مرحلة نمو (21) يومًا. فحصت الدراسة أنسجة العين بحثًا عن التغيرات النسيجية المرضية للعين، وكشفت عن مجموعة من التشوهات في مجموعات مختلفة في مجاميع العلاج اللاحقة (G3 إلى G6)، وتم تحديد التغيرات النسجية والمرضية. أظهرت المجموعات G3 إلى G6 وجود الأوعية الدموية، والبؤر النزفية، وانحطاطًا معتدلًا في طبقات الألياف العصبية، وذمة في طبقة الألياف النووية، وتغيرات تنكسية شديدة في الصفيحة الصدرية، وخلايا الطبقة الليفية، وطبقة العصي والمخاريط في شبكية العين. وخلصت الدراسة إلى أن البيسفينول قد يسبب تشوهات مسخية ونسيجية في عيون أجنة الدجاج وهذا يتطلب مزيدا من البحث والدراسة.

الكلمات المفتاحية: دايفينول-أ، التغيرات المسخية، النسيجية، العيون، أجنة الدجاج