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Original Research Article

Carbaryl developed histological alterations in the liver of albino rats

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ABSTRACT
One of the most worrisome hazardous compounds that are purposefully added to our environment is pesticides. Because of its broad-spectrum activity in commercial agricultural, poultry, cattle, home and garden pest control, and other areas, carbaryl, a synthetic 1-naphthyl-N-methylcarbamate insecticide, is widely employed. There is, however, a dearth of knowledge on Carbaryl's impact on the liver. Carbaryl was given to albino rats, and the results on their liver histology were examined. There was noticeable severe liver damage in patients who received Carbaryl. Further research would be necessary to determine the true effects of this herbicide on the liver.

1. Introduction

The remarkable increase in agricultural output over past two decades has largely been made possible by extraordinary advancement in agricultural techniques, adoption of superior and high yield varieties, and the reduction of losses through the use of pesticides, fungicides, and herbicides. The majority of chemicals used today are synthetic and unnatural for living systems. It is just unacceptable that pesticides are being used widely and sometimes excessively to the point that their metabolites are found in human and rat tissues like milk, meat, poultry, etc. The pesticides or their residues may enter the rat and human systems via number of different channels. A primary goal of research is to examine effects of Carbaryl, an insecticidal carbamate. Organophosphates, carbamates, and synthetic pyrethoid pesticides persist and have a propensity to concentrate in non-target organisms as they ascend up the food chain, which makes them more harmful to fish, birds, other wildlife, and ultimately to humans. The total ecological effects are therefore severe and considerable [1].

Because of their potent anti-cholinesterase effect, substances that fall under general category of substituted carbanic acid Easters have found extensive use in agriculture as insect oxidants. The organic carbamate inhibitor is thought to cabamylate the cholinesterase enzyme during the inhibitory process [2], and it is asserted that N-Methyl Carbamates are broken down by human liver. For its broad-spectrum activity in commercial agricultural, poultry, rat, home and garden pest management, carbaryl, a synthetic 1-naphthyl-N-methyl-carbamate insecticide, is widely employed. The most often found carbamate in juice samples was examined [3]. Human toxicity exists due to reversible cholinesterase inhibitor carbaryl. The Environmental Protection Agency (EPA) of United States has classified it as a probable human carcinogen [4]. An investigation on the kidney effects of carbaryl on rats in proximal convoluted tubule [5].

Congenital deformity in chicken and duck embryos treated with -carbaryl was documented in a number of experimental experiments [6–8]. On cutaneous exposure to carbaryl for 4 weeks, the male albino rats' heart, liver, kidney, lung, and brain had histological alterations [9]. Hepatocellular injury was also indicated by a rise in the activity of acid phosphatase and transaminase [10]. Furthermore, carbaryl has been reported to inhibit liver enzymes [11]. This investigation was done to learn more about how carbaryl affects the histology of the liver.

2 Materials and methods

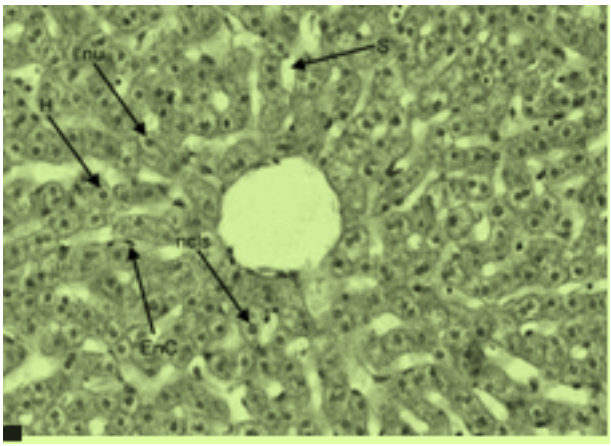
In the current study, young mice weighing 30 Gm were chosen as the study's model. They were divided into three groups, with group 1 (N = 10) getting distilled water intraperitoneally injected. Rats in group II received 200 mg/kg of coron oil five days per week for a period of five weeks. Carbaryl in corn oil was intraperitoneally injected into group III rats five days per week for 4 weeks, at a dose of 200 mg/kg.

The rats were kept in a 12-hour light-to-dark cycle with unlimited access to food and drink. Prior to the start of the experiment and before the rats were sacrificed, the body weights were taken. Within 24 hours of the final injection, the rats in all groups were slaughtered. The liver was taken out of the rats after a thorough anaesthesia. The liver was promptly fixed in 10% formalin after being sliced into smaller (5 mm) pieces. By using the paraffin wax embedding procedure, the blocks were made ready for section cutting with a microtone. Hematoxylin and eosin (H&E) stain was used to colour sections that ranged in thickness from 5 to 7 micro-meter [12].

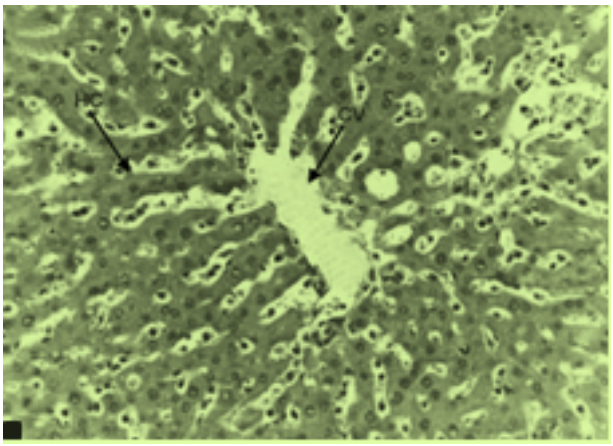
3 Histological changes

Whereas the liver in experimental group III was reddish brown in colour with some pinpoint sub capsular haemorrhages throughout the surface, the liver in groups I and II was grossly

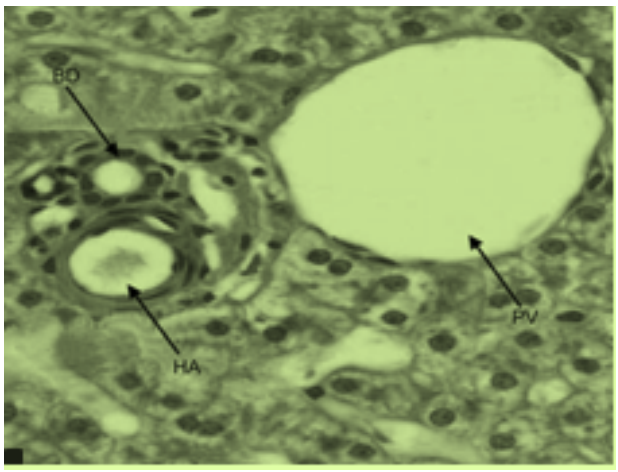
a massive, dark, reddish maroon-colored organ hanging under the diaphragm by peritoneal ligaments (Figs. 1A to F).



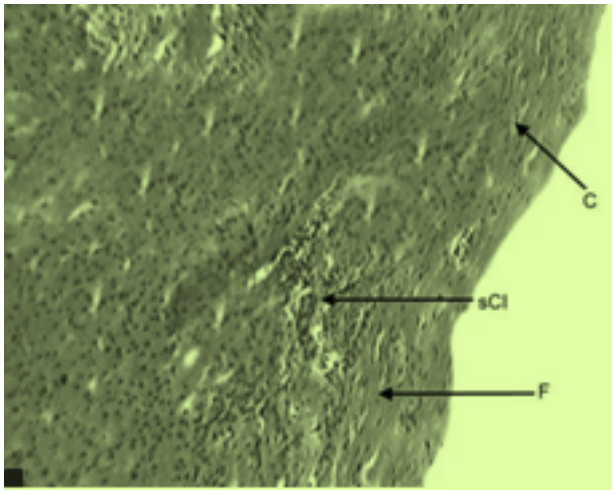
Figures 1A: Photomicrograph of transverse sections of the livers of group I rats reveal that the sinusoids are lined by endothelial cells (EnC), which are polyhedral in form and have spherical euchromatic nuclei and conspicuous nucleoli (S). H&E stain (400×).



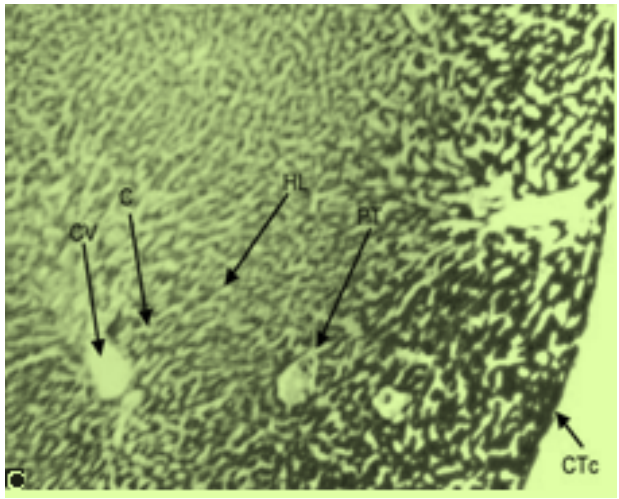
Figures 1B: Photomicrograph of transverse section of the liver of group I rat demonstrating the portal triad of the hepatic artery (HA) portal vein (PV), and bile duct (BD) in addition to the liver parenchyma H&E stain (800×).



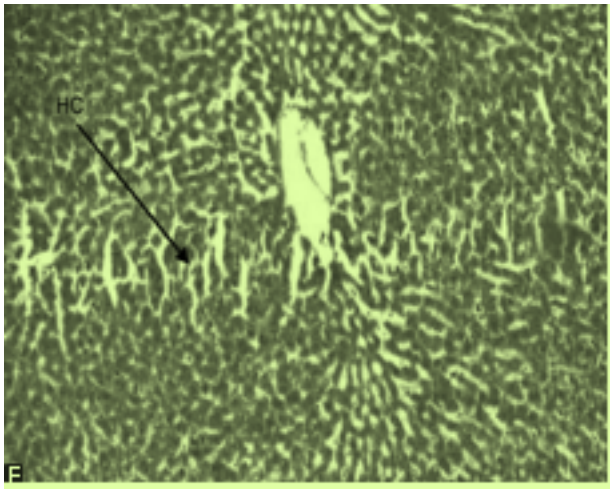
Figures 1C: Photomicrograph of the connective tissue capsule (CTc) and the radial arrangement of cords (C) around the central vein (CV) with the portal triad (PT) at the periphery of the hepatic lobule are visible in a photomicrograph of a transverse section of the liver of a group II rat (HL), the H&E stain (100×).



Figures 1D: Photomicrograph of transverse section of the liver of group II rat showing the hepatocyte cods arranged radially around the major vein, H&E stain (800×).



Figures 1E: Photomicrograph of transverse section of group III rat liver demonstrating enlarged capsule (C), fibrosis (F), and subcapsular inflammatory cells (sCI), H&E stain (100×).



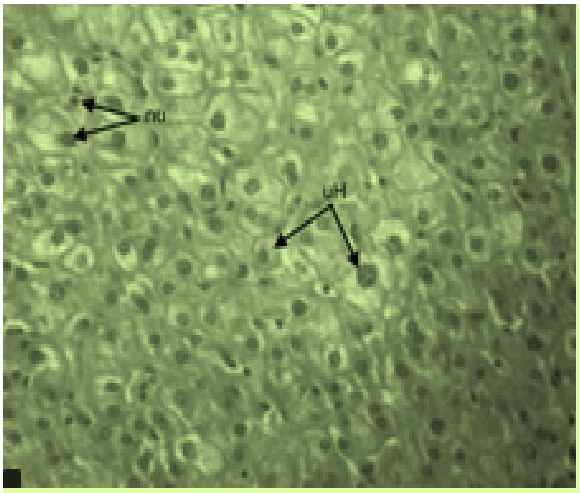
Figures 1F: Photomicrograph of hepatocytic cord disruption is visible in the group III liver's transverse section, H&E stain (100×).

The histomorphological analysis of groups I and II showed the liver to be identical. The connective tissue capsule in the liver of experimental rats (group III) exhibited fibrotic alterations, inflammatory cells, and spots of thickening. The hepatocyte cords' one cell thick, neatly organised layout was messed up in numerous places. In comparison to groups I and II, the majority of the hepatocytes in group III were larger. Hepatocytes with thick and pyknotic nuclei may be seen in many places. Several of the hepatocytes at the sites were binucleated. There were spots where there had been macro- and microvesicular fatty alterations.

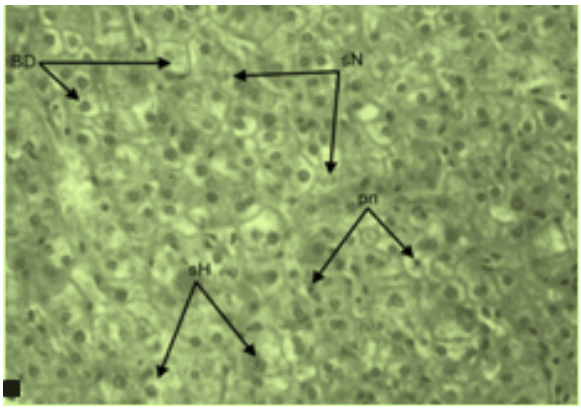
Due to hepatic deterioration, the region surrounding the major vein displayed hepatocytes with strongly eosinophilic cytoplasm and inflammatory infiltration around the portal triads. Due to increased metabolic activity, many liver cells in regions far from the major vein exhibited enhanced cytoplasmic basophilia.

Furthermore, bile ductule growth and fibrosis were observed in and around several portal triads. Congestion was observed in the central vein and branches of the hepatic artery, and the sinusoids, central veins, and portal vein branches all appeared dilated. There were certain haemorrhage sites where the normal parenchyma was occasionally replaced by sizable blood-filled voids.

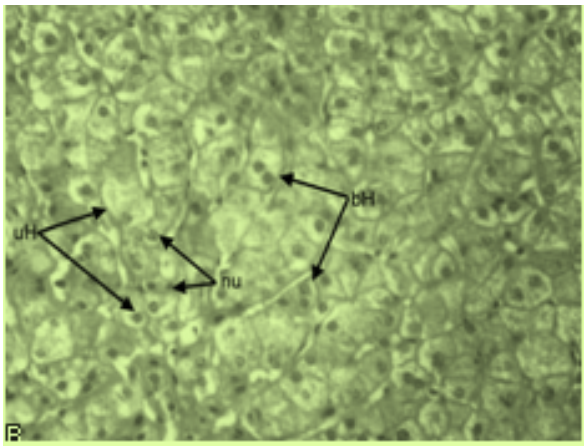
When compared to the normal control and vehicle control rats in the current investigation, the histomorphological changes in the liver of the rats treated with carbaryl were noticeably different. Disrupted hepatocytic cord pattern, capsular fibrosis, subcapsular inflammatory cells, enlarged hepatocytes, signs of increased cellular metabolism concurrent with ballooning degeneration, micro- and macrovesicular fatty change, cytoplasmic basophilia, fibrosis, and inflammatory infiltrate around the portal triads, along with dilated and congested blood vessels, proliferation of bile ductules (Figs 2A to J).



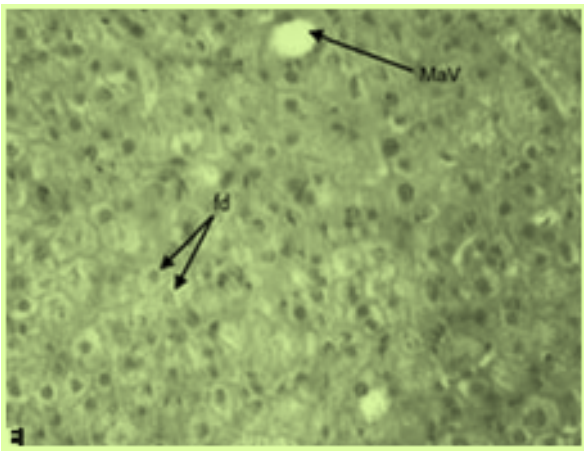
Figures 2A: A transverse segment of a group III rat's liver demonstrating the nuclei and hepatocytes' varying sizes (pleomorphism), H&E stain (400×).



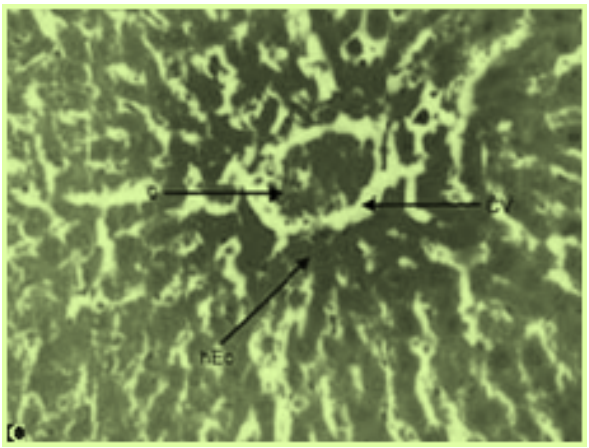
Figures 2B: Transverse section of group III rat liver demonstrating pleomorphism in the size of the hepatocytes (uH) and their nuclei (nu), as well as the proportion of binucleate hepatocytes (bH), H&E stain (400×).



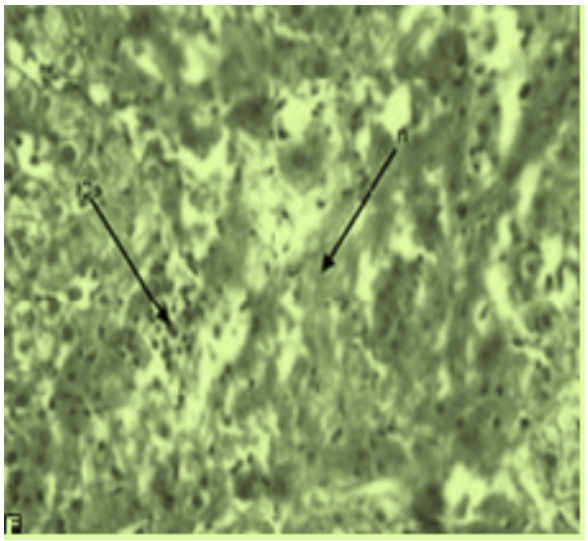
Figures 2C: Transverse section of degenerating swelling and empty hepatocytes (sH) with a central vein are visible in a transverse section of the liver of a group III rat in H&E stain, H&E stain (400×).



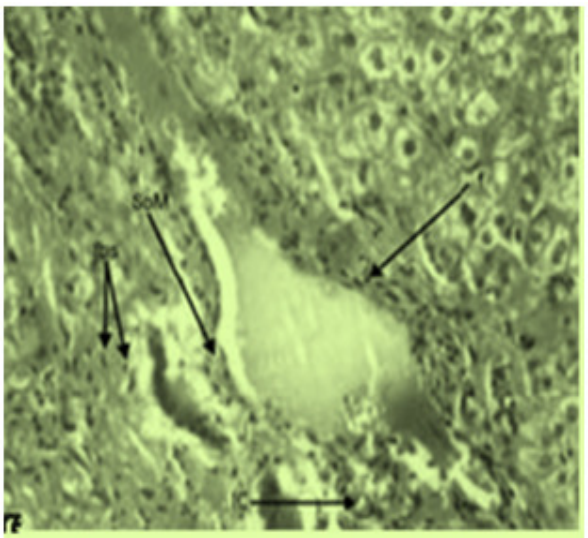
Figures 2D: Transverse section of liver of group III rat showing few hypereosinophilic cells (hEc) and congestion (C) in hazy cell membrane, few hepatocytes with swollen and partially lysed nuclei (sN), i.e. ballooning degeneration (BD) of hepatocytes, and some with dense and pyknotic nuclei (pn), H&E stain (400×).



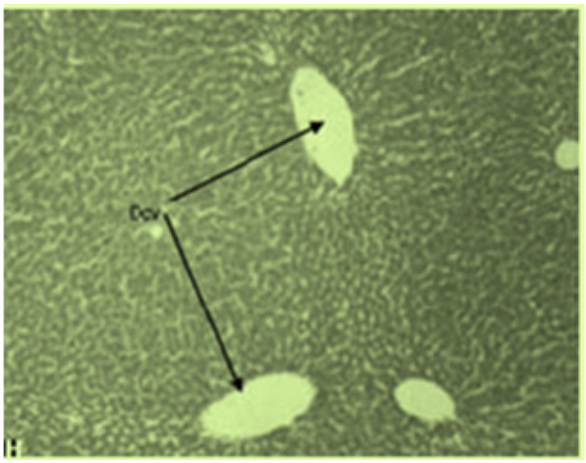
Figures 2E: Transverse section of the group III rat's liver demonstrating areas of macro- and microvesicular fatty alteration, H&E stain (400×).



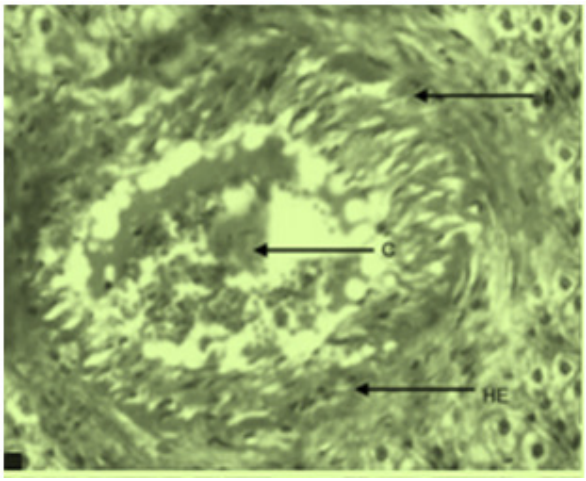
Figures 2F: Transverse section of liver of group III rat demonstrating necrosis (n) and inflammatory cells (ICs), primarily lymphocytes in the liver parenchyma, H&E stain (400×).



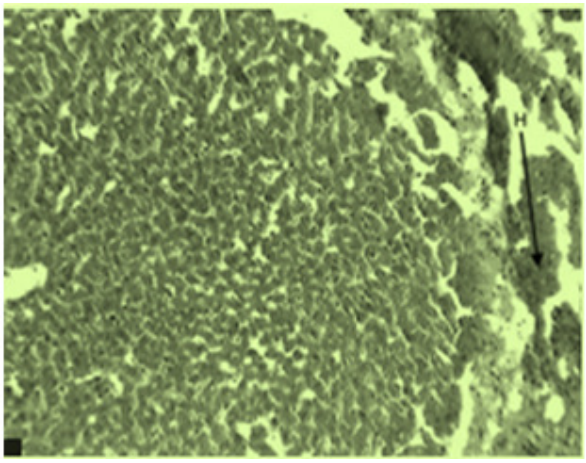
Figures 2G: Transverse section of liver of group III rat demonstrating inflammatory cells and fibroblasts in the space of Mall (SoM), H&E (400×).



Figures 2H: Transverse section of group III rat liver demonstrating fibrosis (f) in the number of principal veins (Dcv), H&E stain (100×).



Figures 2I: Transverse liver slice of a group III rat demonstrating congestion and fibrosis (f) surrounding the hepatic artery (HE), H&E stain (400×).



Figures 2 J: Transverse liver slice of a group III rat demonstrating hemorrhage (H) in the liver parenchyma , H&E stain (100×).

4 Results and discussion

Following the administration of the first dose of carbaryl in the current investigation, the rats became instantly hyperactive and agitated. For 30 minutes, this was accompanied by sneezing, shivering, and trembling. These

results are consistent with Gaines' observations [13], where carbaryl poisoning manifested as muscle fasciculations, tremors, excessive salivation and lacrimation, diarrhoea, and involuntary urination following a single oral or cutaneous dose. Furthermore reported [14–17] were similar cholinergic effects. In response to carbaryl treatment, several hepatocytes grew in size. Several metabolically active organs exhibit hyperactivity in addition to the liver.

Few hepatocytes in the current investigation seemed enlarged and empty at some places, with diffuse cell membranes. They also have larger nuclei. Several of these cells had their nuclear membranes gone. The nucleus' size is a sign of how actively the cell is functioning. This shows that these cells are overactively involved in the metabolism of carbaryl based on the observed increase in nucleus size. These results point to a continuing, escalating hepatocyte deterioration.

Several hepatocytes had cytoplasm that seemed to be frothy and to contain numerous small vacuoles. This suggests that the liver cells have undergone a fatty transformation. These observed ultrastructural alterations imply that carbaryl metabolism may be taking place in the cytoplasm, and that this overactivity gradually depleted the cell and caused degeneration. Additionally, it implies that carbaryl may have an impact on a variety of bodily systems through a transdermal route in addition to the intraperitoneal route used in the current investigation.

Hepatocytes that had shrunk and had a strongly eosinophilic cytoplasm were also visible in the vicinity of the portal triads and central vein. Their nucleus was pyknotic and dense. Khera [18] observed hepatic degenerative alterations in the duck and chick embryos after carbaryl injection.

Wills et al. [19] showed that when carbaryl was given orally to men for 6 weeks at doses of 0, 0.06, and 0.12 mg/kg daily, there were no appreciable histological or biochemical alterations in normal body processes. The different (lesser) dose and different medication administration strategy may be the cause of the inconsistent observations.

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