Journal of Science and Practice of Pharmacy
December 2021; 8 (1): 417-424
Available at http://www.jsppharm.org; https://doi.org/10.47227/jsppharm.v8i1.2
ISSN: 2449-0458 (print); 2449-0466 (electronic)

Original Research Article

Toxicological evaluation of virgin coconut oil extracted from *Cocos nucifera* L. (Arecaceae)

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Abstract

Purpose: Virgin coconut oil is claimed to have a lot of health benefits, especially in lowering lipid levels in serum and tissues, and the current consumption rate is very high, especially in developing countries. Determination of the toxicological effects on possible organs that are likely to be susceptible to toxicity by virgin coconut oil will provide supportive scientific evidence to consumers, practitioners, and providers.
Methods: Virgin coconut oil was obtained from fresh coconut using wet milling method. LD 50 was determined using mice. Sub-acute toxicity was determined by administering 0.25 ml and 0.5ml of virgin coconut oil to female and male rats respectively for 30 days with normal saline as control. The tissues of the lung, heart, spleen, liver, and kidney (female and male rats); fallopian tube, ovary (female rats); prostate, bladder, testis, and epididymis (male rats) were examined.
Results: Most of the organs in both male and female rats retained normal tissue architecture, however, there was mild Kupffer cell activation in the liver and mild sinus histiocytosis with an otherwise normal follicular architecture in the spleen of both male and female rats.
Conclusion: Virgin coconut oil could be termed to be relatively safe, but its overall safety profile needs to be further evaluated.

Keywords: Virgin coconut oil, toxicological profile, wet-milling, ethnomedicine

Introduction

Coconut (*Cocos nucifera* Linn), of the family Arecaceae is called *Ekuro* (Yoruba), *Aku* (Ibo), and *Kwakwa* (Hausa) [1]. It is a unique source of various natural products (vitamins, minerals, amino acids, and phytohormones) useful for the development of medicines against various diseases [2]. It is an important fruit tree that contains two very important components i.e. coconut water and coconut milk, which is the source of coconut oil. The fruit can be made into a variety of foods and beverages [3]. Virgin coconut oil (VCO) extracted by wet process directly from coconut milk under a controlled temperature has been claimed to have more beneficial effects than copra oil (CO) since it retains most of the unsaponifiable components [4]. This signifies a good potential in ameliorating human disease conditions. Virgin coconut oil obtained by wet process has a beneficial effect in lowering lipid components compared to copra oil. It reduces total cholesterol, triglycerides, phospholipids, LDL, and VLDL cholesterol levels and increases HDL cholesterol in serum and tissues [4].

The parts of its fruit like Coconut kernel and Coconut water are of great medicinal value because of their antimicrobial and antioxidant properties [2]. Coconut oil is known to exhibit antimicrobial activity against *Streptococcus mutans* and *Candida albicans* [5,6]. *Cocos nucifera* has significant inhibitory action against common oral pathogens, indicating the presence

of highly effective antimicrobial compounds [7]. Koschek *et al* in their studies indicated that catechin, one of the compounds present in the extract of *C. nucifera* plant is capable of inhibiting tumor cell lines [8].

Toxicological studies, including the determination of median lethal dose (LD 50) and other such parameters essential for the proper dosage of herbal medicines are desirable and necessary. If there is a need for more detailed studies, such herbal medicines may be subjected to sub-acute and chronic toxicity tests. It has been stated by the WHO that the most critical assessment of herbal medicine is safety evaluation [9]. Although it has been indicated that phytotoxicity is very low, nonetheless, from scientific, professional, and moral viewpoints, toxicological assessment should be conducted on all herbal medicines intended for either human or veterinary use [9].

It has been stated that all substances have potential toxicity and that all medicinal herbs can, therefore be harmful, but most would have to be ingested in impossible amounts to cause harm [9]. Promoters of virgin coconut oil claim that it is high in healthy saturated fats that have different effects than most other fats present in diets. These fats are claimed to boost fat burning and provide the body and brain with quick energy. They are also claimed to raise the HLD cholesterol in the blood, which is linked to reduced heart disease risk [4]. Toxicity evaluation of virgin coconut oil is therefore desirable in view of its claimed numerous benefits and wide usage. The present study was aimed at evaluating the acute and sub-acute toxicity of virgin coconut oil as a prelude to ascertaining its safety profile.

Methods

Preparation of virgin coconut oil

Virgin coconut oil was prepared by removing the husk and shell from freshly harvested matured coconuts. The meat was shredded and cold-pressed to extract the coconut milk without any addition of chemicals [4]. The milk was covered with cheesecloth and set aside at room temperature for 20 hours to naturally ferment. The oil separated through gravity with the water at the bottom while oil and protein curds remained on top. The oil was carefully filtered and separated from the curd. The oil was kept in a tight container at room temperature.

Animals

Male and female Swiss albino mice (22. 93 ± 1. 44g) and Wistar rats (231. 00 ± 13. 83g) were obtained from the Animal House, at the Department of Pharmacology and Toxicology, Faculty of Pharmacy, University of Benin, Benin City. All animals were kept under standard environmental conditions and were handled according to the international protocol for use of animals in experiments [10]. They were fed with standard pellets and tap water ad libitum. Ethical approval for the study was obtained from the College of Medicine, University of Benin Animal Ethics Committee (ADM/F. 22A/Vol. viii/349).

Toxicological evaluation

Acute toxicity test

The Swiss albino mice were divided into 6 groups of 6 animals each. They were orally administered the virgin coconut oil extracted from *Cocos nucifera* at doses of 01, 0.2, 0.3, 0.4, and 0.5 ml. Mice administered 5.0 ml/kg of normal saline served as the control group. On day one, the animals were observed for 20 mins for every 6h and for at least 20 mins for the next two days. Lethality as well as gross toxicological features were recorded daily for each group. These features included the state of hyperactivity, diarrhea, convulsion, and pile erection [11]. Observation of the animals was continued for the next twelve days.

Sub-acute toxicity test

In the test to determine the effects of administering the virgin coconut oil on key organs in Wistar rats, standard procedures [12, 13, 14, and 15] were adopted. Wistar rats (15) were randomly distributed into 3 groups of 5 rats each. The first (A) group served as control and received 5.0 ml/kg of normal saline while the second (B) and the third (C) groups received oral doses of 0.25 ml (female rats) and 0.50 ml (male rats) of the oil per day for 30 consecutive days. On day 30, the rats were sacrificed under

chloroform anesthesia with the hearts, lungs, liver, spleen, kidney, fallopian tube, ovary, bladder, testis, and epididymis removed and preserved in 10% formaldehyde solution. The organs were sectioned (6.0 µm thick) and embedded in paraffin wax and stained with H & E [16].

Results

In the acute toxicity study, the virgin coconut oil from Cocos nucifera caused no deaths up to the oral dose level of 0.5 ml in mice, thus the LD 50 was greater than 0.5 ml (LD 50 > 0.5 ml). The mice did not show any significant gross toxicological features. Behavioural tendencies, mode, and movement of mice as well as agility and posture were at best normal. There were no wet stools, rather stools were dry and rounded. The animals did not show signs of convulsion or exhibit writhing.

Daily administration of the oil for 30 days in rats did not produce any major toxicological symptoms nor were deaths recorded before they were sacrificed. Histopathological analysis of the lungs (Fig. 1) of the 0.25 ml virgin coconut oil treated group showed that the lungs retained their normal architecture. However, at 0.5 ml (Fig. 2), there was mild activation of the bronchioloalveolar lymphoid aggregates. In the heart (Figs. 3 & 4), there was treatment-induced focal coronary vascular intimal erosion with an otherwise normal myocardial architecture. The oil, induced mild coronary vascular erosion. In the spleen of both male (Fig. 5) and female rats (Fig. 6), the oil, induced mild sinus histiocytosis with an otherwise normal follicular architecture. There was also mild Kupffer cell activation in the liver (Figs. 7 & 8) while the nephrons (Figs. 9 & 10) retained their normal architecture. The architecture of the tissues of the fallopian tubes (Fig. 11), ovaries (Fig. 12) as well as the prostate (Fig. 13), bladder (Fig. 14), testis (Fig. 15), and epididymis (Fig. 16) was normal.

Discussion

The wet milling method of extraction was used because it retains the unique flavor, smell, and taste of fresh coconut in the final product. Also, the natural fermentation/wet-milled process yields the least amount of virgin coconut oil but the purest in form and highest in quality. A more modern way of separating the oil from the curd is through centrifugal force, but such a process introduces heat due to friction and might cause some deterioration in the natural nutritive content of the oil.

It has been realized that a high degree of precision may not be necessary to compare toxicity [17]. Therefore, the LD 50 is now an approximate value estimated from the smallest number of animals possible. The dose is used to calculate the initial dose to be tried in man during the clinical trials. It has been suggested that a simple limit test, aimed at determining the effect on animals of the largest dose likely to be administered to a human being is often enough [18]. The primary purpose of an acute toxicity test is to determine the nature and extent of the adverse reactions to a single dose or an overdose of the drug [8]. Increasing doses of virgin coconut oil up to 0.5 ml administered to mice per os were not lethal so the LD 50 could not be determined. It is an indication of the low toxicity of virgin coconut oil, therefore it can be said to be relatively safe and may be used for the treatment and management of the varied ailments it is currently advertised for.

The general purpose of the sub-acute toxicity tests is to determine the organs that are likely to be susceptible to toxicity by the herbal medicines. Histological features of adult female and male Wistar rats administered 0.25 ml and 0.5 ml respectively of virgin coconut oil did not induce any toxic changes in the selected visceral organs examined. However, in the lungs and spleen of the male rats and the liver of both sexes, it mildly activated the local immune system. This was evidenced by the activation of the bronchioloalveolar lymphoid aggregates (lungs); the sinus histiocytes (spleen) and the perisinusoidal Kupffer cells (liver). This indicates that the oil was not toxic to the organs at the administered doses, though the immune system was activated to a mild degree.

Conclusion

These results obtained from the toxicological evaluation should be useful to both manufacturers and patients, and serve as a guide on the judicious use of virgin coconut oil as its safety can to an extent be guaranteed, having shown no remarkable toxicological effects at the administered doses. It is however advised that

herbal practitioners should be aware of the likely effects that may occur, especially on prolonged usage.

Acknowledgements

The author wishes to express his appreciation to Prof. Freddy Agoreyo of the Department of Physiology, School of Basic Medical Sciences, University of Benin, Benin City for his assistance.

Conflict of Interest

No conflict of interest is associated with this work.

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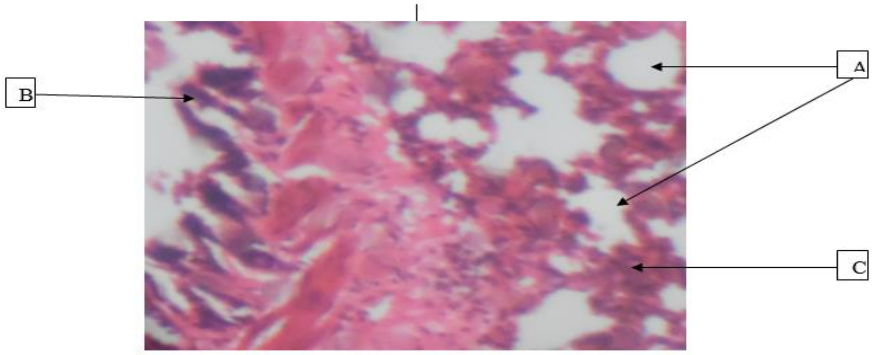

Fig. 1: Photomicrograph of the lung of a female rat administered 0.25ml of virgin coconut oil showing A, normal alveolar sacs, B, terminal bronchiole, and C, interstitial space (H&E x 100)

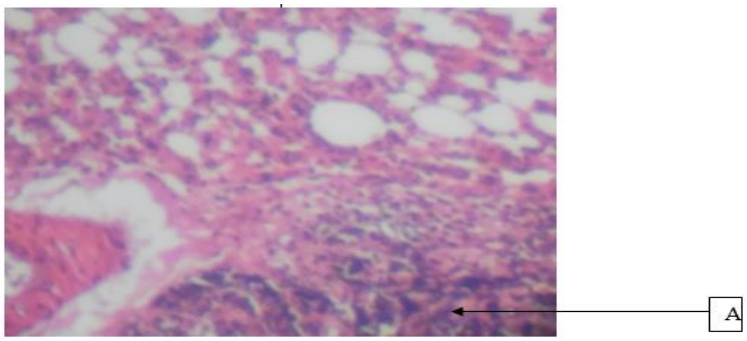

Fig. 2: Photomicrograph of the lung of a male rat administered with 0.5ml of virgin coconut oil showing A, mild activation of the bronchioloalveolar lymphoid aggregates (H&E x 100)

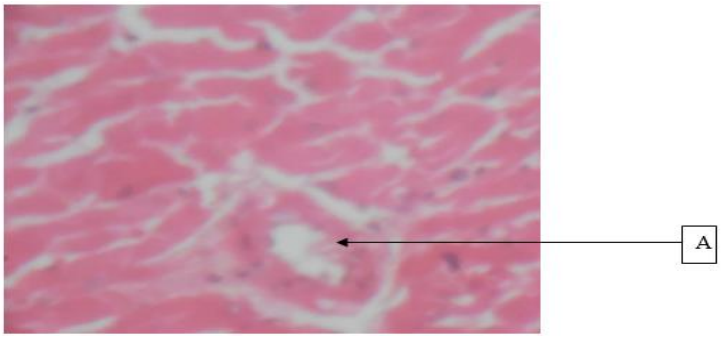

Fig. 3: Photomicrograph of the heart of a female rat administered with 0.25ml of virgin coconut oil showing A, focal intimal erosion (H&E x 100)

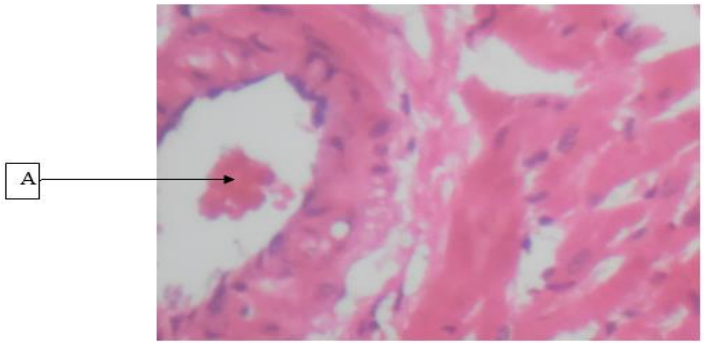

Fig. 4: Photomicrograph of the heart of a male rat administered with 0.5ml of virgin coconut oil showing A, mild vascular congestion (H&E x 100)

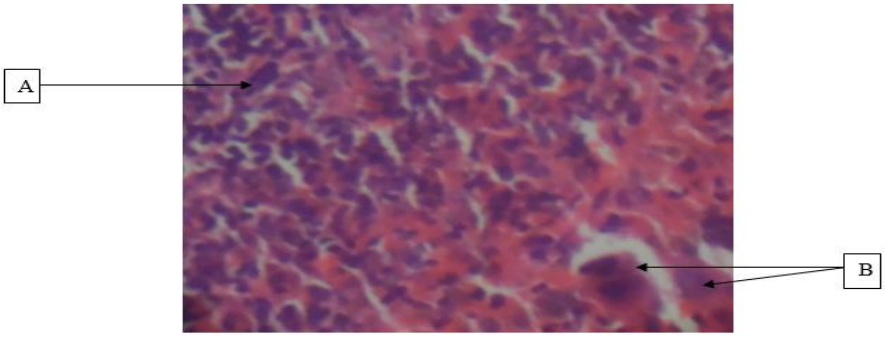

Fig. 5: Photomicrograph of the spleen of a female rat administered with 0.25ml of virgin coconut oil showing A, normal follicular architecture, and B, mild sinus histiocytosis (H&E x 100)

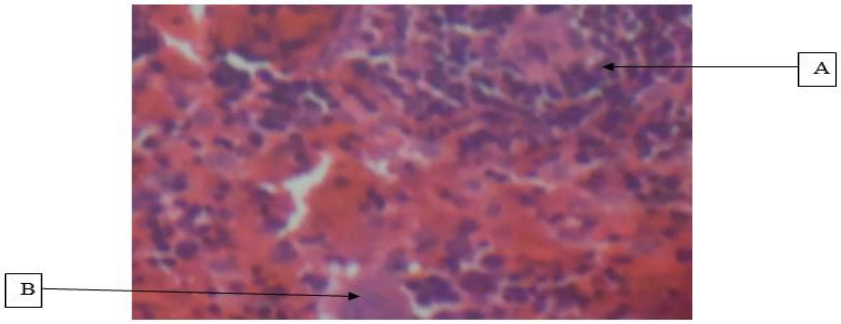

Fig. 6: Photomicrograph of the spleen of a male rat administered with 0.5ml of virgin coconut oil showing A, normal follicular architecture, and B, mild sinus histiocytosis (H&E x 100)

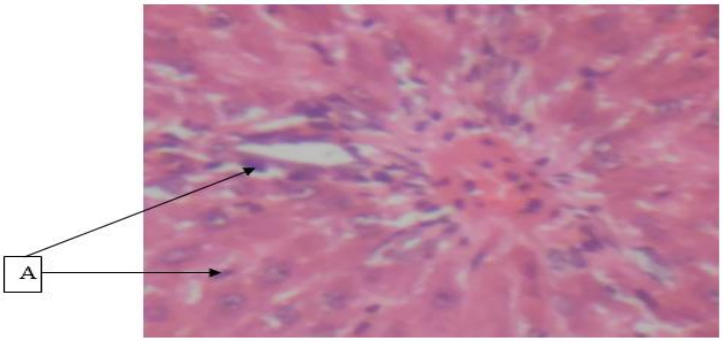

Fig. 7: Photomicrograph of the liver of a female rat administered with 0.25ml of virgin coconut showing A, mild Kupffer cell activation (H&E x 100)

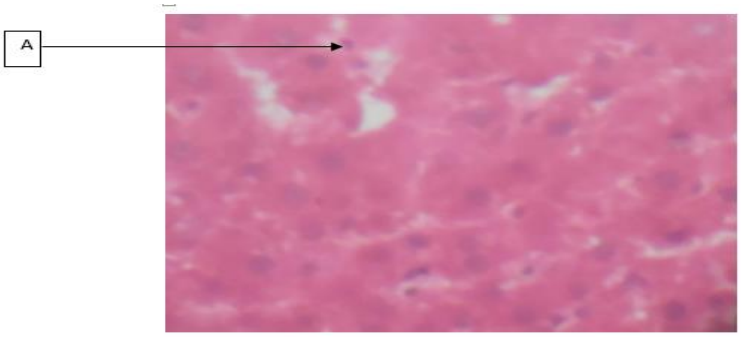

Fig. 8: Photomicrograph of the liver of a male rat administered with 0.5ml of virgin coconut oil showing A, mild Kupffer cell activation (H&E x 100)

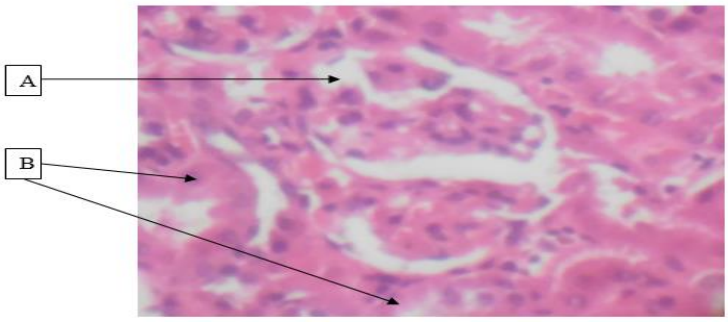

Fig. 9: Photomicrograph of the kidney of a female rat administered with 0.25ml of virgin coconut oil showing A, normal glomerulus and B, tubules (H&Ex 100)

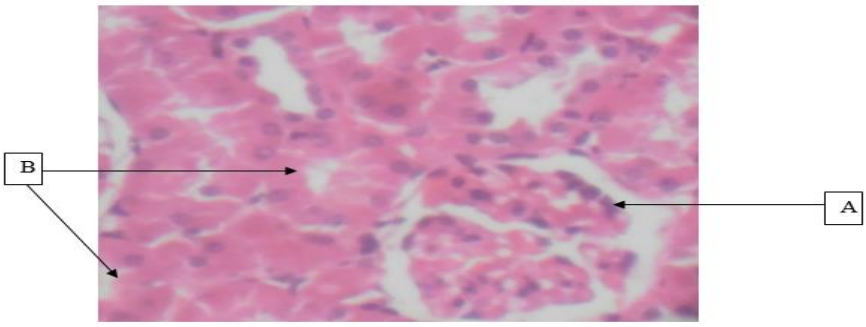

Fig. 10: Photomicrograph of the kidney of a male rat administered with 0.5ml of virgin coconut oil showing A, normal glomerulus and B, tubules (H&E x 100)

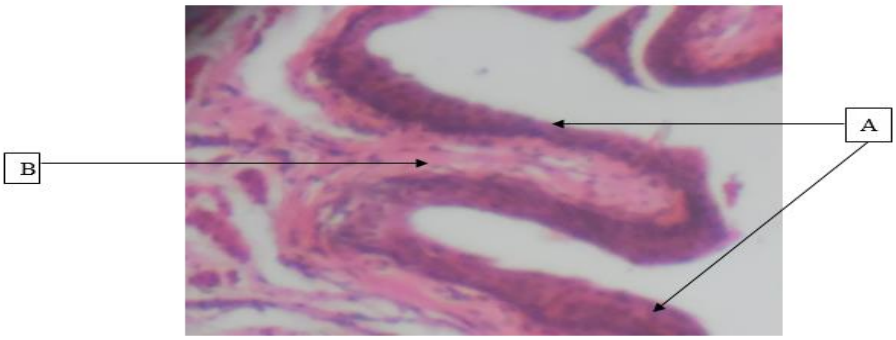

Fig.11: Photomicrograph of the fallopian tube of a female rat administered with 0.25ml of virgin coconut oil showing A, normal mucosal fold, and B, connective tissue core (H&E x 100)

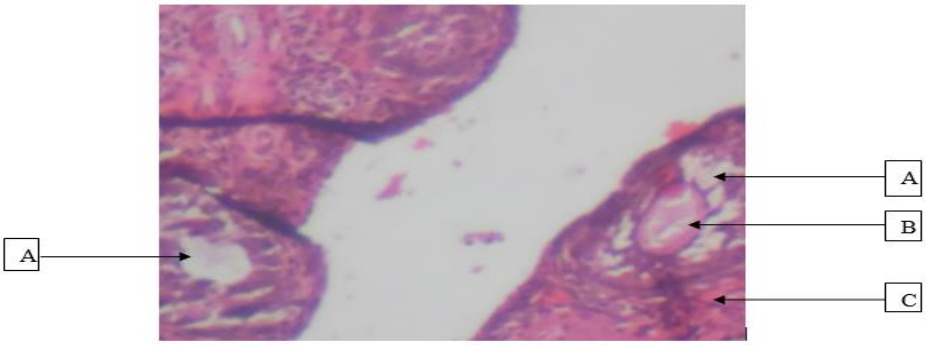

Fig. 12: Photomicrograph of the ovary of a female rat administered with 0.25ml of virgin coconut oil showing A, normal follicles, B, ovum and C, stroma (H&E x 100)

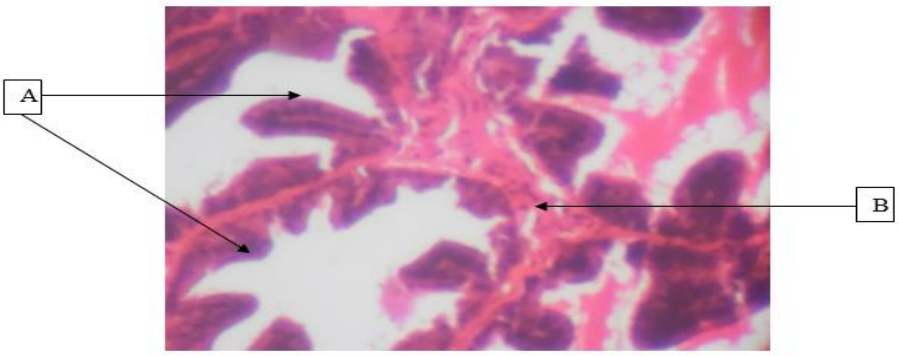

Fig. 13: Photomicrograph of the prostate of a male rat administered with 0.5ml of virgin coconut oil showing A, normal acini and B, fibromuscular stroma (H&E x 100)

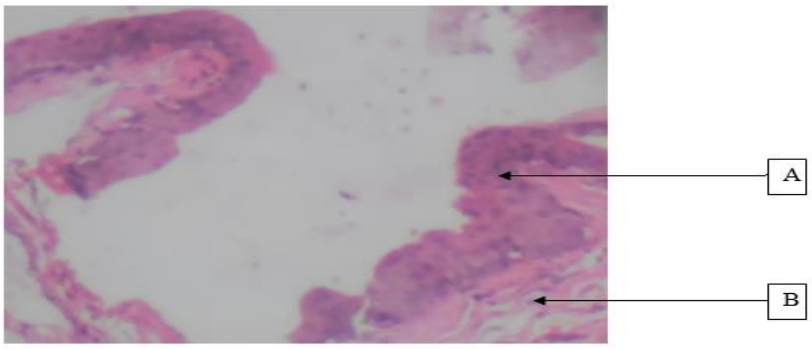

Fig. 14: Photomicrograph of the bladder of a male rat administered with 0.5ml of virgin coconut oil showing A, normal transitional epithelium, and B, connective tissue (H&E x 100)

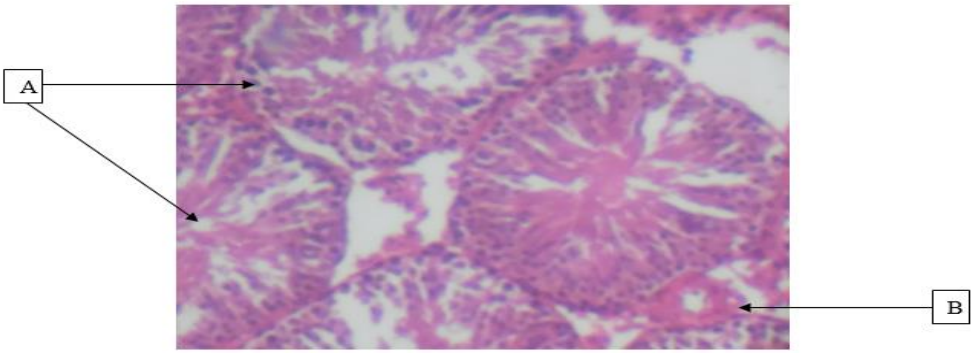

Fig. 15: Photomicrograph of the testis of a male rat administered with 0.5ml of virgin coconut oil showing A, seminiferous tubules in normal sequential maturation and B, vascularized interstitial space (H&E x 100)

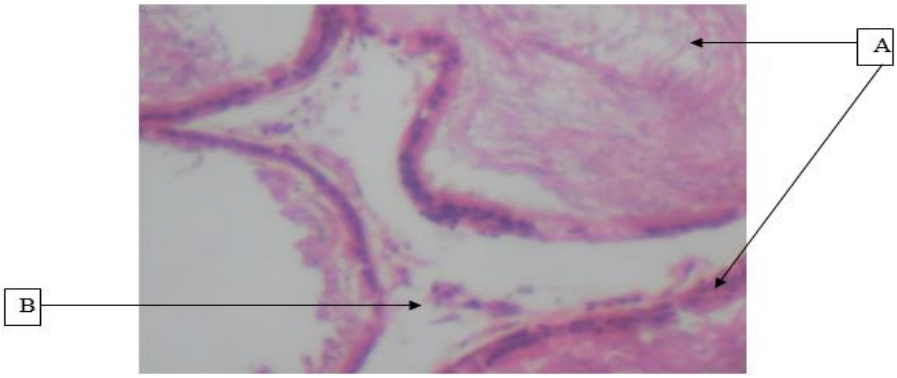

Fig. 16: Photomicrograph of the epididymis of a male rat administered with 0.5ml of virgin coconut oil showing A, normal tubules parked with mature spermatozoa and B, interstitial space (H&E x 100)