

## Micro architecture of the Testes following administration of Coconut Oil (*Cocos nucifera L.*) on Adult

Albino Wistar Rats

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**Abstract:** This study was aimed at determining the effect of different dosages of coconut oil on the histology of the testis. The rats in the control group were administered with distilled water while those in the low, moderate and high dose groups were administered with coconut oil extract of 0.5, 1.1 and 2.2ml/kg body weight respectively once daily for fourteen days. On day 14 of the experiment, the rats were sacrificed. The testes of rats from various groups were carefully dissected out and weighed. There was no significant decrease ( $p < 0.05$ ) in mean testicular weight of the treated rats; low dose ( $1.14 \pm 0.05$ ), moderate dose ( $1.16 \pm 0.05$ ) and high dose ( $1.12 \pm 0.06$ ) groups compared to the control rats ( $1.20 \pm 0.04$ ). The histological sections revealed proliferation of seminiferous epithelium especially that of the high dose group. In conclusion, coconut oil has no deleterious effect on the histology of the testes and could lead to increased spermatogenesis.

**Keywords:** *Cocos nucifera*, testis, histology, spermatogenesis

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### I. Introduction

Coconut (*Cocos nucifera*) is consumed locally as food, while its oil (coconut oil) is used for cooking, soap making and production of margarine (Ishaq & Odeyemi, 2012). Coconut oil is extracted using heat, pressure and/or chemical solvents (Ishaq & Odeyemi, 2012). In recent years, this oil has attained superstardom in the healthy foods' world. Many people are adopting its use, nutritionists advocating it and patients acclaiming its many virtues and as such, a number of health benefits have been attributed to this oil. Coconut oil consists of a mixture of triglycerides containing only short and medium chain saturated fatty acids (92%) i.e. healthy fats and unsaturated fatty acids (8%) (Dayrit, 2003). One tablespoon of coconut oil contains 14 grams of total fat, 12 grams of which are saturated fat. This means that about 86% of the total fat content of coconut oil comes from saturated fat (USDA, 2017). Medium chain triglycerides taken at fairly high doses reduce blood cholesterol levels and coconut oil is nature's richest source of medium chain triglycerides (Calbom and Calbom, 2005). When it comes to cooking, refined coconut oil has an advantage over most other oils because it can be heated to higher temperatures (up to 450°F) without becoming damaged (Srivastava *et al.*, 2010; Fullana, Carbonell-Barrachina and Sidhu, 2004). Coconut oil is beneficial in different ways to man. The oil plays a great role in skin care, hair care, stress relief, weight loss and cholesterol level maintenance, immunomodulatory effects, cardiovascular uses and more recently in Alzheimer's disease. Other potential benefits include prevention of different biological conditions due to its active polyphenol components (Nevin & Rajamohan, 2004). Coconut oil is a good source of iron, sodium, potassium and calcium because the concentrations of these elements in the oil meet up with the adequate quantity needed by the body daily. Thus, coconut oil has both nutritional and pharmacological benefits and being free from lead, it is safe for human consumption (Sani *et al.*, 2014). Research has shown that coconut oil raises the level of total and Low Density Lipoprotein (LDL) cholesterol in the blood more than a diet with unsaturated plant oils like safflower oil (Cox *et al.*, 1994). However, some studies have shown that consuming coconut oil is associated with an increase in High Density Lipoprotein (HDL) - good cholesterol (Feranil *et al.*, 2011; Voon *et al.*, 2011).

Over the years, infertility has been on the increase in both males and females. The increase in male infertility however has become a source of global concern. This has been attributed to factors such as smoking, drinking of alcoholic beverages and use of restricted drugs, stress, poor nutrition and lack of exercise (Berkley, 2004). Also, injury to the testes, blockage in the vas deferens, excessive heat to the testes, vitamin deficiencies and varicocele have also been associated with infertility in men (Swierzewski, 2009). With the upsurge of the use of coconut oil as a pro-fertility agent to alleviate the problem of infertility, there is need to know the side effects excessive intake could play out. It is known that the fatty acid composition of sperm membranes, especially their unsaturated components, determine their biophysical characteristics such as fluidity and

flexibility as appropriate for their specific functions including sperm motility and fertilizing capacity (Khatibjoo *et al.*, 2011). Adding some quantity of corn oil, coconut oil, groundnut oil and palm oil etc. to your meal may improve semen quality of any animal, mammal as well as human (Naji, 2013) but consuming high rates of these fatty diets induce systematic metabolic changes in blood plasma, liver and urine samples involving several metabolic pathways (An *et al.*, 2013). Diets combining high fat and cholesterol can also produce changes in lipid composition and enzyme metabolism (Quiles & Ramirez-Tortosa, 2008). Due to the abundance of coconuts in the Southern part of Nigeria (Odenigbo & Otisi, 2011) and considering its high demand especially for the extraction of coconut oil, the present study was carried out to examine the effect of coconut oil on the histology of the testes using Albino Wistar rats.

## **II. Materials and method**

Twenty adult male albino Wistar rats were used for the study. These rats were acquired from University of Calabar Animal Farm. The rats were put into four well ventilated cages which were obtained from the Faculty of Basic Medical Sciences' Animal House, University of Calabar. The rats were fed using Grower's Feed (obtained from Vital Feeds) and distilled water. They were maintained under standard conditions with a temperature range of 25-27°C under day/night 12-12hr photoperiodicity. The control group was treated orally with diet and distilled water while the experimental groups were treated orally with diet, water and coconut oil.

### **Coconut oil extraction**

Sixteen mature coconuts were obtained from the market. The coconut oil was extracted using a modified wet extraction method described by nevin and Rajamohan (2004, 2006). The solid endosperm of mature coconuts were crushed and made into viscous slurry. About 500ml of warm water was added to the slurry obtained and squeezed through a fine sieve to obtain coconut milk. The resultant coconut milk was left for 24hrs to facilitate the gravitational separation of the emulsion as described by Onsaard *et al.* (2005) and Nour *et al.* (2009). Demulsification produced layers of an aqueous phase (water) on the bottom, an emulsion phase (cream) in the middle layer and an oil phase on top as described by Nour *et al.* (2009). The oil on top was scooped and heated for about 4hrs to remove moisture. The obtained virgin coconut oil (VCO) was then filtered through a fine sieve, stored at room temperature and used for the experiment.

### **Experimental design**

The twenty adult male albino Wistar rats were divided into control and experimental groups. The experimental group was further divided into three groups; low, moderate and high dose with five animals in each. The control group received normal saline and experimental groups; Low Dose (LD), Moderate Dose (MD) and High Dose (HD) received coconut oil extract of 0.5ml/kg b.w, 1.1ml/kg b.w and 2.2ml/kg b.w respectively for fourteen days via orogastric tube. After 14 days, the animals were sacrificed and testes collected for histopathological studies

### **Statistical analysis**

All data obtained from the analysis were normally distributed. The differences between the treated and control groups were statistically evaluated using a ANOVA and SPSS Computer Package since the data were parametric. All data will be expressed as mean values  $\pm$ SEM with significant values at  $p < 0.05$ .

## **III. Results**

This analysis was carried out using the testes of 20 adult male albino Wistar rats that were divided into four groups (n=5/group) thus: Control rats which received distilled water, Low dose (LD) rats which received 0.5ml/kg body weight, Moderate dose (MD) rats which received 1.1ml/kg body weight and High dose (HD) rats which received 2.2ml/kg body weight of oil.

### **Testicular Weights**

In this present study, coconut oil caused significant decrease ( $p < 0.05$ ) in the testicular weight of treated rats; low dose ( $1.14 \pm 0.05$ ), moderate dose ( $1.16 \pm 0.05$ ) and high dose ( $1.12 \pm 0.06$ ) groups compared to the control rats ( $1.20 \pm 0.04$ ). The moderate dose group presented a significantly higher ( $p < 0.05$ ) value compared to the low dose and the high dose groups. (Table 1)

### **Histological Sections**

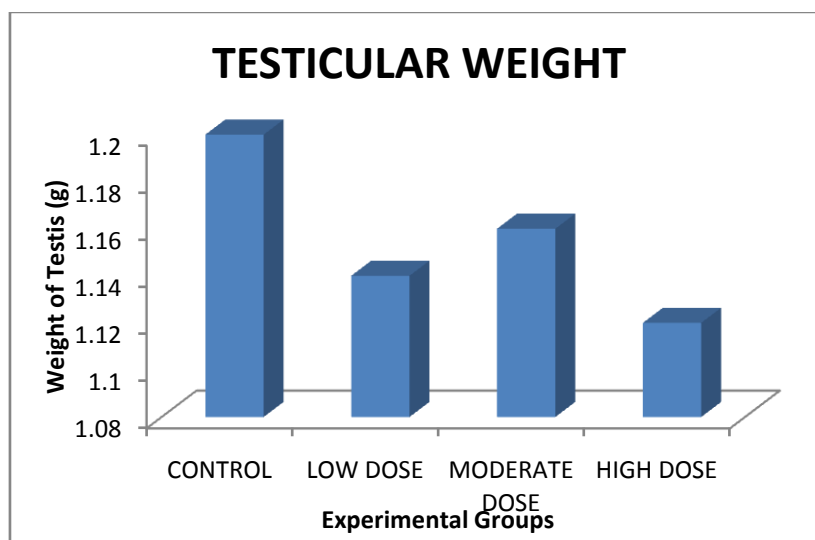
Histological sections of control rats showed normal histo-architecture of seminiferous tubule, spermatocyte, lumen, basement membrane, spermatogonium, Leydig cell, Sertoli cell, spermatids (Plate 1). Sections of testes of Low dose rats treated with coconut oil at dose level of 0.5ml/kg body weight and Moderate dose rats treated with coconut oil at dose level of 1.1ml/kg body weight showed normal histo-architecture as

seen in the control with proliferating spermatogonia at different stages of maturation (Plates 2 & 3). While High Dose rats treated with coconut oil at dose level of 2.2ml/kg body weight revealed proliferation of spermatocytes and increased spermatogenesis (Plate 4).

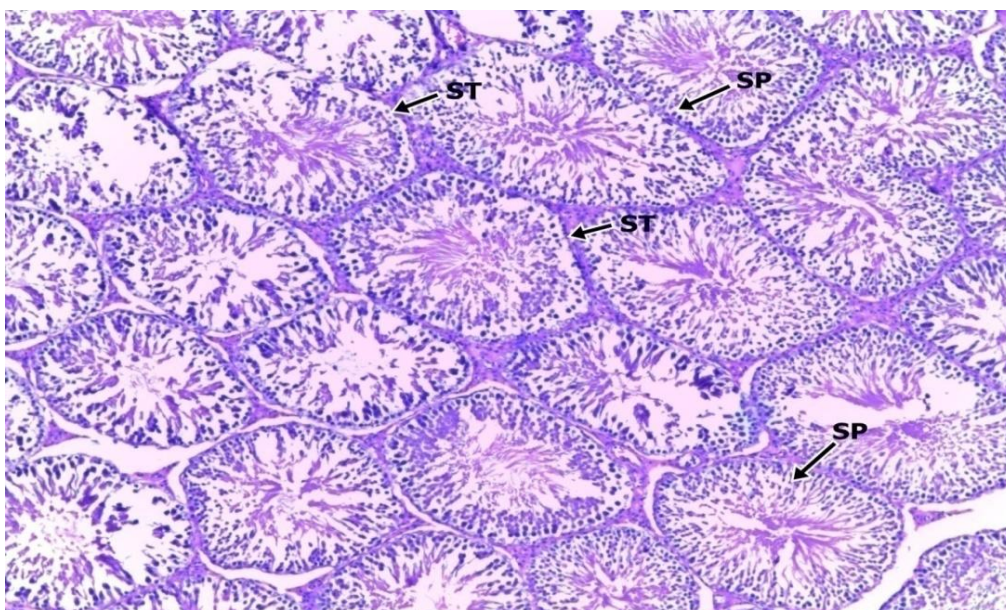
**Table 1:** Comparison of testicular weights between the control and experimental groups

Experimental Groups	Testicular weight (g)
Control	1.20±0.04
Low dose	1.14±0.05
Moderate dose	1.16±0.05
High dose	1.12±0.06

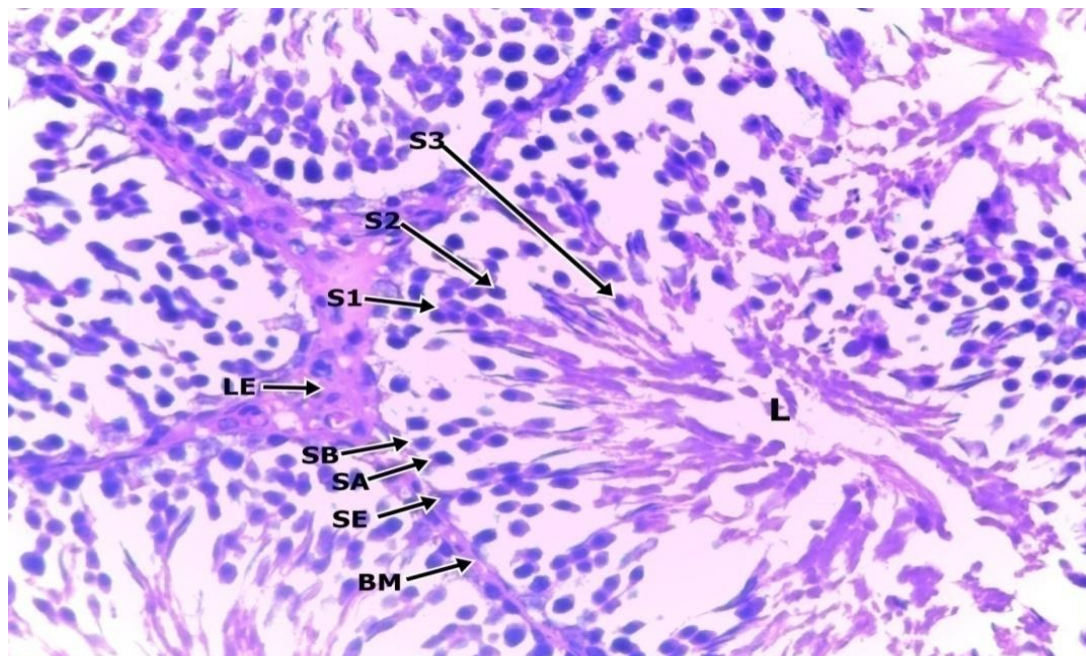
Values are expressed as mean±SEM, n=5



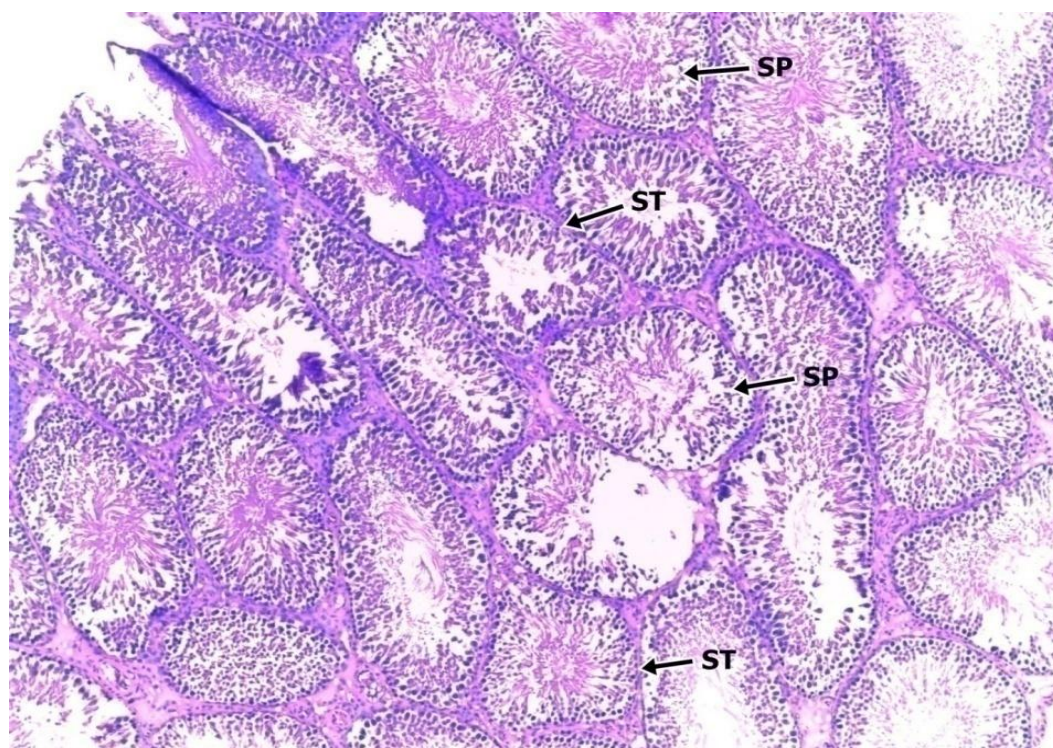
**FIG 1:** Chart showing testicular weights in the different experimental groups



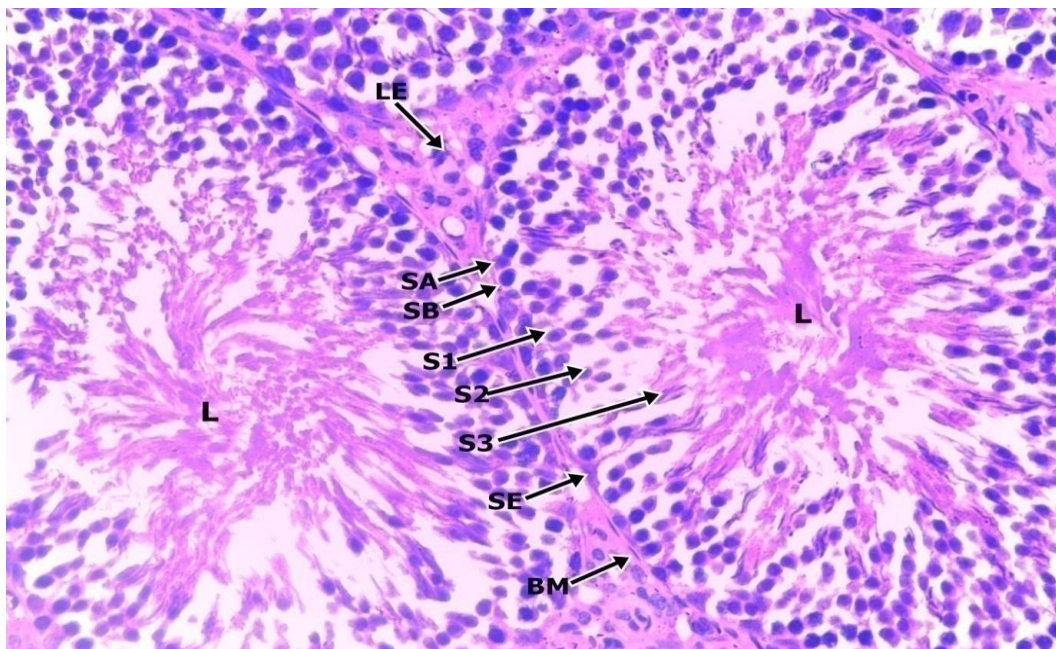
**PLATE 1A:** Testis of control rat showing normal histo-architecture. ST- Seminiferous tubule, SP- Spermatocyte H&E x100



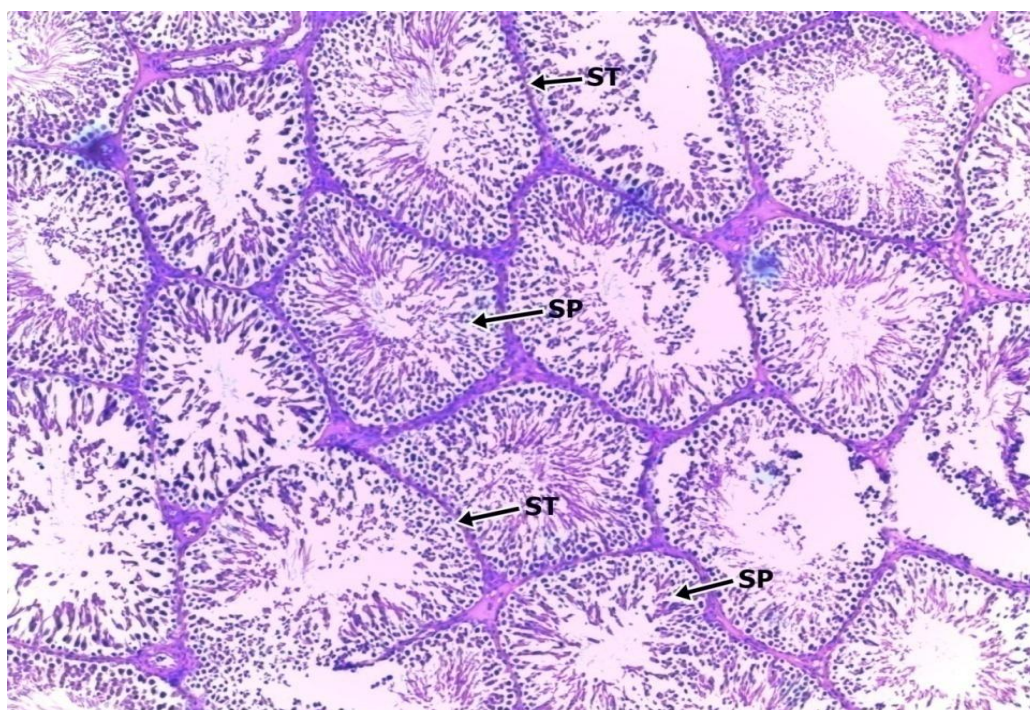
**PLATE 1B:** Control rat showing normal histo-architecture. L- Lumen, BM- Basement Membrane, SA-Spermatogonium A, SB- Spermatogonium B, LE- Leydig cell, SE- Sertoli cell, S- Spermatid H&E x400.



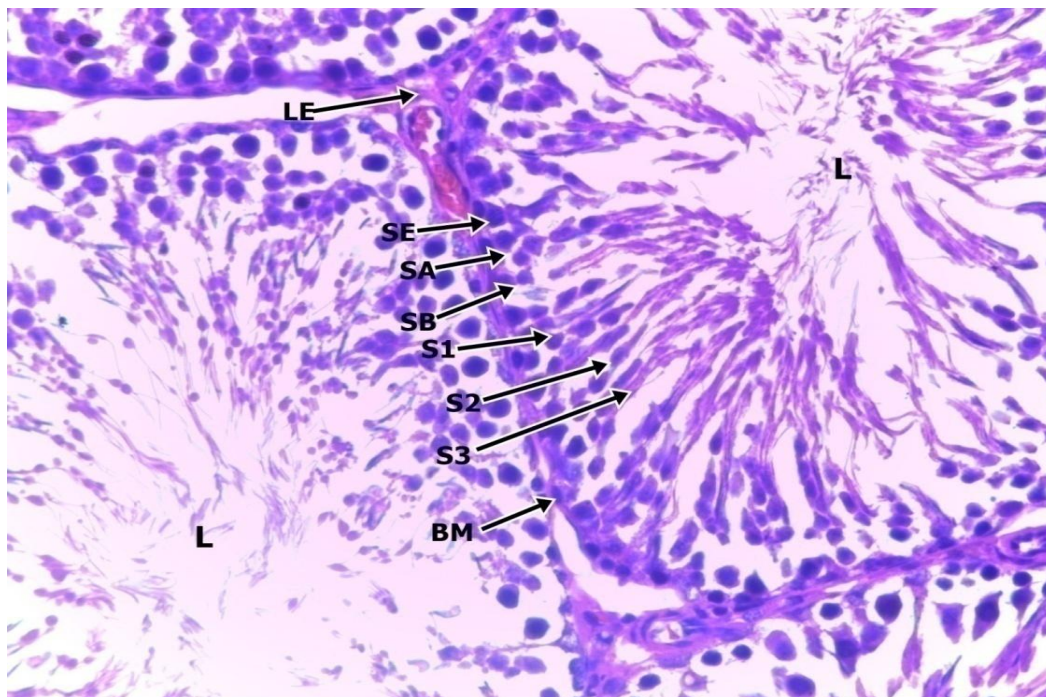
**PLATE 2A:** Testis of Low dose rat treated with coconut oil at dose level of 0.5ml/kg body weight showing normal histo-architecture. SP- Spermatocyte, ST- Seminiferous Tubule H&E x100



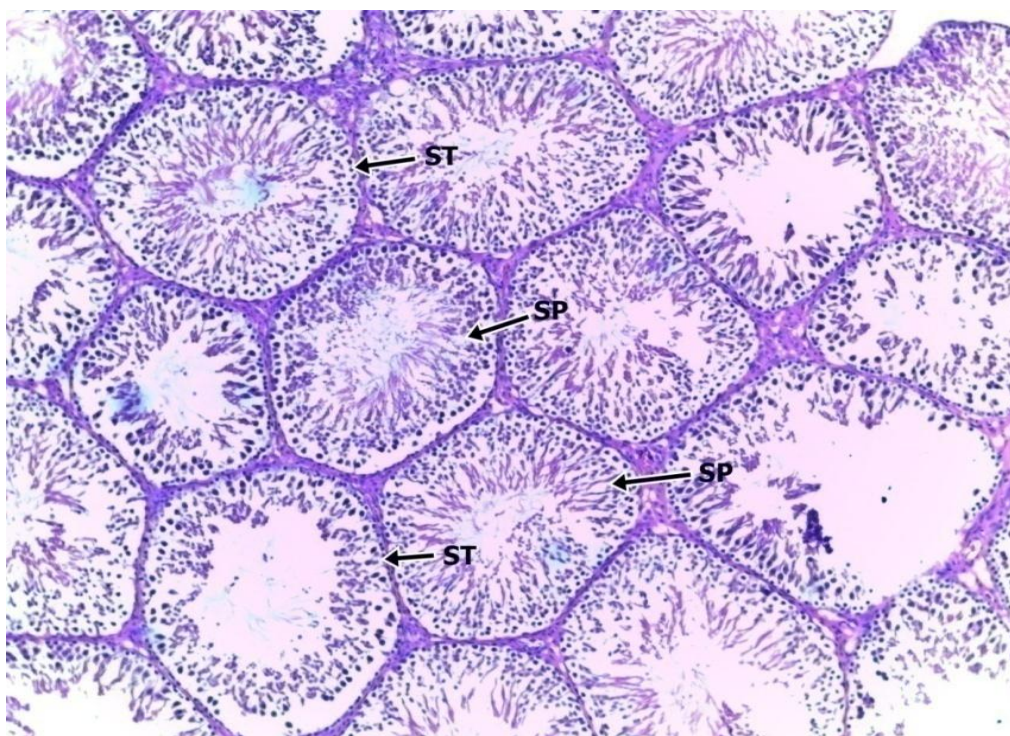
**PLATE 2B:** Testis of Low dose rat at dose level of 0.5ml/kg body weight showing proliferating spermatogonia at different stages of maturation. H&E x400



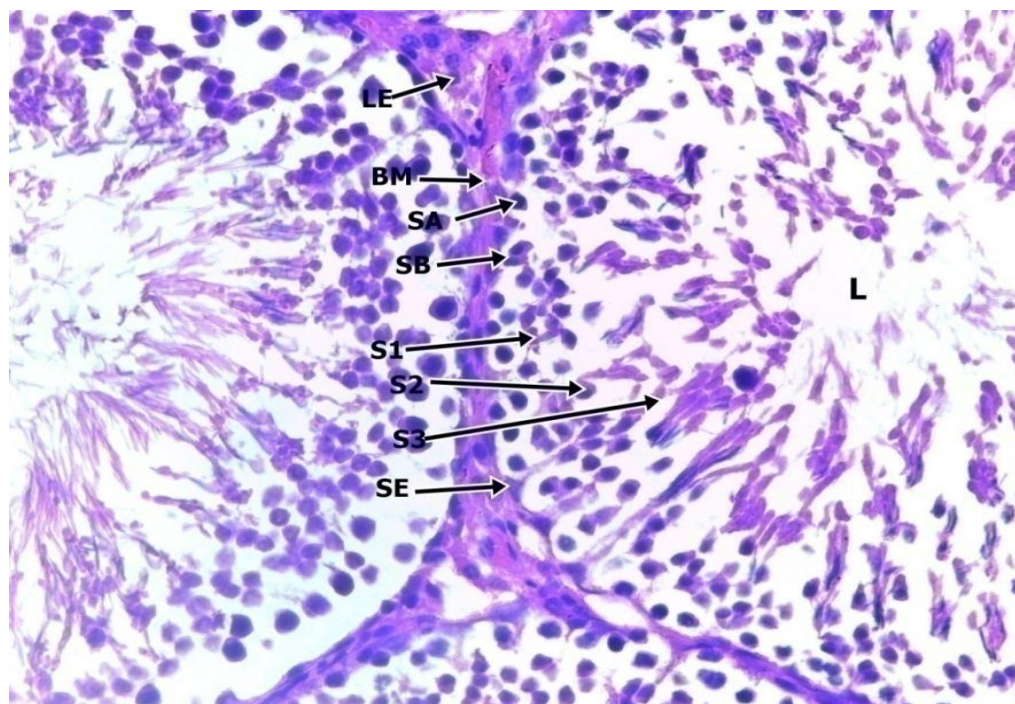
**PLATE 3A:** Testis of Moderate dose rat treated with coconut oil at dose level of 1.1ml/kg body weight showing normal histo-architecture. H&E x100



**PLATE 3B:** Testis of Moderate dose rat treated with coconut oil at dose level of 1.1ml/kg body weight showing spermatogenesis. H&E x400



**PLATE 4A:** Testis of High Dose rat treated with coconut oil at dose level of 2.2ml/kg body weight showing enormous proliferation of spermatocytes. H&E x100



**PLATE 4B:** Testis of High Dose rat treated with coconut oil at dose level of 2.2ml/kg body weight showing increased spermatogenesis. H&E x400

#### IV. Discussion

Results from this study revealed no significant difference in mean testicular weight between the control and coconut oil-treated rats, a decrease in the mean testicular weight of the treated rats was however observed. Maina *et al.* (2008) reported that an increase or decrease in relative or absolute weight of an organ after administering a chemical or drug is an indication of the toxic effect of that chemical. Research has shown that blockage of the efferent ducts by cells sloughed from the germinal epithelium or the efferent ducts themselves can lead to an increase in testis weight due to fluid accumulation (Hess *et al.*, 1991; Nakai *et al.*, 1993), an effect that could offset the effect of depletion of the germinal epithelium on testicular weight. Also, the weight of male reproductive organs usually provides a useful reproductive risk assessment in experimental studies (Raji *et al.*, 2005) and testicular size is the best primary assessment for spermatogenesis, since the tubules and germinal elements account for approximately 98% of the testicular mass (Sherines and Howards, 1978). The histological analysis revealed increased spermatogenesis in treated group that received oral administration of coconut oil compared to the control. There was no adverse histological change; rather there was marked proliferation of spermatogenic cell lines, transformation from primordial cells to spermatids and further to spermatozoa. The seminiferous tubules were moderately enlarged as well as the central lumen. Brucefife (2000) reported that coconut oil has an antioxidant effect. Testicular membranes are rich in fatty acids (lipid) which are prone to oxidative injury, it is reasonable to consider that lipid peroxidation (damage to cell membrane) may contribute to gonadal dysfunction (Emanuele and Emanuele, 2001). Testicular cell membrane is stabilized by vitamin (A) which is responsible for reducing lipid peroxidation (Emanuele and Emanuele, 2001).

#### V. Conclusion

Coconut oil has no deleterious effect on the histology of the testes and could lead to increased spermatogenesis

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