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**Chronic LD50 of Coconut Oil**Ojianwuna, C.C.<sup>1</sup>, Mokogwu A.T.H.<sup>2</sup>, Avwioro O.G.\*<sup>2</sup>, Nwambuonwor C.C.<sup>2</sup>, Ojidere, C.H.<sup>1</sup>

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**Abstract**

*Coconut oil is edible and it is obtained from the kernel of mature coconuts of the coconut palm. It has been used in skin and hair care products as well as for the relief of stress, weight loss, cholesterol level maintenance, cardiovascular diseases and for maintenance of normal body system. Studied on the LD<sub>50</sub> of coconut oil was done using seven Wistar rats weighing 245±2g. The rats were grouped into 3, of 4 and 2 in each group. The 7<sup>th</sup> rat served as the control. All rats had normal feeds and water regularly throughout the experiment. They had oral ingestion of coconut oil twice daily with an increase of 0.5ml coconut oil daily until death of last rats on the 18<sup>th</sup> day (1.0ml/kg to 10.0ml/kg). In Group 1, two rats died at 9.5ml/kg, while the other 2 rats died at 10.0ml/kg on the 18<sup>th</sup> and 19<sup>th</sup> days respectively. This process was repeated with 2 rats to confirm the process, all two rats died at 9.5ml/kg. The weights of the rats and organs were not significantly different ( $p>0.05$ ) from the control rat throughout the experiment. Chronic LD<sub>50</sub> was found to be 9.2ml/kg b.w.*

**Keywords:** Coconut oil, LD50, Chronic

**Introduction**

*Coconut oil, also known as Copra oil is extracted from the kernel of mature coconuts of the coconut palm (Kumar, 1997). Coconut oil is colorless and the aroma varies from mild to intense depending on the method of its extraction. Some contents are high levels of lauric acid, myristic acid, oleic acid and palmitic acid and small levels of caproic acid palmitoleic acid, linoleic acid, stearic acid and linolenic acid.*

Coconut oil reduced lipid peroxidation and increased the activity of SOD in the serum of mice undergoing the forced swim test when their brains were subjected to chronic cold restraint (Yeap *et al.*, 2015). Coconut is rich in polyphenols and these contribute to the increased antioxidant enzyme levels, which in turn helps to reduce inflammation and lipid peroxidation in coconut oil- treated mice. The potential of coconut to prevent exercise- and chronic cold restraint stress-induced damage and to restore the antioxidant balance has been attributed to the presence of polyphenols and medium-chain fatty acids in it (Arunima, 2013). The protective activity of coconut oil on the liver has been established in an experiment with 2, 4-Dichlorophenoxyacetic acid (2, 4-D) induced liver damage in rats (Hanaa *et al.*, 2013). Intahphuak *et al.* (2010) studied the anti-inflammatory, analgesic, and antipyretic effects of coconut in rats using ethyl phenyl propiolate-induced ear oedema and carrageenan and arachidonic acid-induced paw oedema and found that coconut possesses moderate anti-inflammatory properties as well as moderate analgesic and antipyretic properties (Intahphuak *et al.*, 2010). *Coconut oil* has been found to be effective in the healing of wounds (Srivastava and Durgaprasad, 2008). Animals treated with coconut have been found to have faster wound healing activity and higher levels of various skin components than untreated animals occasioned by significant increase of pepsin-soluble collagen and glycohydrolase activities resulting in higher collagen cross- linking and turnover (Nevin and Rajamohan, 2010). Another study revealed that coconut can be used as an eye

rewetting agent in the management of dry eyes (Haliza *et al.*, 2015). Coconut oil has also been used in the soap industries, plastics, pharmaceuticals, cosmetics, rubber substitutes and synthetic resins (Gopala *et al.*, 2016). Coconut oil has been consumed extensively in some tropical countries for a long time, probably thousands of years ago. It has also been used extensively in processed foods, infant formulae, cosmetics and as hair oil. The oil has been shown to be antiviral, antifungal and antibacterial as it attacks viruses that have a lipid (fatty) coating, such as herpes, HIV, hepatitis C, the flu, and mononucleosis. It is therefore used in the treatment of pneumonia, sore throats, dental cavities, urinary tract infections, meningitis, gonorrhoea, and many other bacterial infections as well some as fungal infections such as candida, ringworm, athletes' foot, thrush, jock itch and diaper rash (Kabara, 2012). Many healthy individuals use coconut oil freely for the maintenance of body health. This research investigated the lethal dose of coconut oil so that people who drink it for pleasure can know the level at which it can cause death when taken orally.

## Materials and Methods

### Preparation of coconut oil

Mature coconut was obtained from the tree *Cocos nucifera* of the family Aracaceae at Lagos, Nigeria. The outer coat and the inner fibrous layer were removed with the aid of a short cutlass to expose the shell copra (dried kernel). This was crushed with a small hammer. The solid white flesh, the endosperm was removed with a small knife and put into a dry clean container. This was grinded into tiny bits of less than 0.1mm thick and allowed to stand at room temperature for 24 hours and centrifuged at 500 rpm to 30 minutes. The supernatant which was the coconut oil was collected into a clean bottle.

### Animals

Seven Wistar rats weighing  $240 \pm 2.0$  g were used for this experiment. They were kept in their cages for 5 days prior to commencement of the experiment to allow for acclimatization to the laboratory conditions. The rats were grouped into 3 of 4 and 2 in each group and one rat served as the control. All rats had normal feeds and

water regularly throughout the experiment.

Group 1: Each of the 4 rats in the group received from 1.0ml/kg to 10.0ml/kg coconut oil twice daily, an increase of 0.5ml daily for 19 days (Table 1).

Groups 2: (A repeat of Group 1 experiment with 2 rats for confirmation). Each of the 2 rats in the group received from 1.0ml/kg to 9.5ml/kg coconut oil twice daily, an increase of 0.5ml daily for 18 days (Table 1).

Group 3 (Control): There was one rat in this group, and it wasn't ingested with coconut oil but had water and feeds regularly.

The animals were weighed daily. At death, the organs were removed, weighed and compared with the control.

## Results

Group 1: Two rats died on the 18<sup>th</sup> day after receiving a cumulative dosage of 9.5ml/kg. The other 2 rats in the group died on the 19<sup>th</sup> day after receiving a cumulative dosage of 10.0ml/kg

Group 2: All two rats died on the 19<sup>th</sup> day after receiving a cumulative dosage of 10.0ml/kg coconut oil.

Group 3: This group had only one rat which was used as a comparison with the other rats. There was no significant change in the total body weight and weight of the organs when compared with the test rats.

$$LD_{50} = \sqrt{(D_0 \times D_{100})}$$

$D_0$  = Highest dose that gave no mortality,  
 $D_{100}$  = Lowest dose that produced mortality.  
 $LD_{50} = \sqrt{9.0 \times 9.5}$   
 = 9.2ml/kg b.w.

**Table 1. Lethal dose of coconut oil on Wistar rats**

Day	Coconut Oil Given Twice Daily (ml/Kg)	Number of Deaths
1	1.0	0
2	1.5	0
3	2.0	0
4	2.5	0
5	3.0	0
6	3.5	0
7	4.0	0
8	4.5	0
9	5.0	0
10	5.5	0
11	6.0	0
12	6.5	0
13	7.0	0
14	7.5	0
15	8.0	0
16	8.5	0
17	9.0	0
18	9.5	4
19	10	2

### Discussion

Certain chemicals have a wide range of effects on animals and humans as well as our environment (Enevide *et al.*, 2013; Lorke, 1983). The toxic effects of chemicals on our bodies depend on the type of chemical, duration of exposure and the quantity administered. Lethal dose is the amount of a chemical required to cause death in a living being. The extent to which people use pharmacological substances and chemicals on the human body has increased to a high level (Sperling, 1979). Some of these chemicals are often constituents of food substances, medicines, beverages, other industrial and household products. They may result in chronic toxicity when used or taken over a long period of time or acute toxicity when large quantities capable of eliciting immediate toxic effects are taken. The effects may be mild or severe, depending on the nature and quantity of the substance taken (Walum, 1998). Lethal dose (LD<sub>50</sub>) (the dose that kills 50% of test animals population) is a major parameter for measuring toxicity and also as an initial procedure for general screening of chemicals and pharmacological substances for toxicity. It gives

information about the degree of safety of a pharmacological agent (Akhila *et al.*, 2007) before it is allowed to enter the market for sale. Several methods have been developed for toxicity testing (Bruce, 1985; Lorke, 1983; Turner, 1965). Some methods make use of few animals while others use more animals. (18) in chronic and sub chronic toxicity testing observed that the LD<sub>50</sub> for the Nufera® Virgin Coconut Oil was more than 5000 mg/kg body weight. They did not observe significant differences in the general condition, growth, organ weights, hematological parameters, clinical chemistry values, or gross and microscopic appearance of the organs from the treatment groups as compared to the control group after 28 days of oral administration of the coconut oil. In our experiment, we observed that the chronic LD<sub>50</sub> of coconut oil was 9.2ml/kg b.w. Those who take coconut oil drink between one and 4ml table spoonful, about 5 to 20ml at a time. This translates to about 0.2ml/kg b.w. at the maximum.

However, it was observed in this research that intakes of coconut oil up to 8.5ml/kg did not kill the experimental rats, although its effects on

the biochemical parameters have not been studied. The result of this experiment will be useful in clinical sciences in cases of drug overdose and in the pharmaceuticals.

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