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Research Article

Histomorphological Studies on the Therapeutic Potential of *Cocos nucifera* (Coconut) on Selected Narcotics Induced Toxicity of the Lungs and Heart of Albino Rat

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Abstract

There have been reports of the protective and healing properties of coconut water in almost every bodily system and in a number of disease states, including those involving the heart and lung systems. The study is aimed at assessing the therapeutic potential of *Cocos nucifera* (coconut) on selected narcotics that induce toxicity in the lungs and hearts of albino rats. In this study, 50 rats were used; the rats were weighed before receiving the substances (Tramadol, Codeine, and Rohypnol) and were thus divided into five groups. Group A (Control) received only normal feed (growers' mash) and distilled water; Group B received 0.2 mg/kg bw of tramadol and 0.2 mg/kg bw of codeine, feed, and distilled water; Group C received 0.2 mg/kg bw of tramadol and 0.2 mg/kg bw of rohypnol; Group D received 0.2 mg/kg bw of codeine (6 weeks); and Group E received 0.2 mg/kg bw of tramadol and 0.2 mg/kg bw of rohypnohypnol; and 0.2 mg/kg bw of *C. nucifera* The animals were weighed, euthanized, and sacrificed, after which the serum was collected and the lungs, heart, and brain tissues were harvested and fixed for histological processing. Alterations were seen in the tested groups; there were congested vessels in the lungs, degenerative cells around the white matter, and congestive coronary arteries in the heart, aside from group five. *C. nucifera* was able to alleviate the damaging effects of the narcotics, possibly due to its rich phytonutrients.

Keywords: *Cocus nucifera*; Narcotics; Opioid; Lungs; Heart; Rats

Introduction

It is sometimes referred to as coconut, coco, coco-da-bahia, or coconut-of-the-beach [1]. *Cocos nucifera* is a member of the palm family [1]. The species is native to Southeast Asia, more specifically the islands between the Indian and Pacific Oceans (Malaysia, Indonesia, and the Philippines) [2]. The coconut palm fruit is thought to have originated in India before making its way to East Africa. This plant was introduced to West Africa with the discovery of the Cape of Good Hope, where it swiftly spread to other tropical parts of the world, including the continent of America [2].

All of the analogues of morphine, including norcodeine (15%), codeine-6-glucuronide (80%), and morphine-6-glucuronide, are made when CYP2D6 breaks down codeine. Codeine has only a very weak analgesic effect. Codeine is converted into morphine and codeine-6-glucuronide, which have analgesic properties [3]. The amount of active narcotics secreted in breast milk can vary depending on how CYP2D6 and UGT2B are impacted by genetic variation [3]. Compared to older babies and kids, newborns have a delayed clearance of morphine from their plasma [3]. The plasma half-life is about 3.35 hours. Other drugs can activate the benzodiazepine rohypnol (flunitrazepam). They interact with the benzodiazepine receptors BNZ1, which controls sleep, and BNZ2, which controls muscle relaxation, anticonvulsant activity, motor coordination, and memory [4,5].

There are numerous industrial and domestic uses for both solid albumen from ripe fruits and green coconut water. Furthermore, people in various countries have used various fruits and plant parts to treat various pathological conditions. Drug addiction has become a serious issue in many nations in recent years [7]. The issue is combined with those that affect society, the economy, domestic policy, and global politics [7]. The distribution of drugs like codeine, rohypnol, and tramadol has been steadily rising [7]. The various drug types that are currently available include methamphetamine, cannabis, volatile substances, opium, heroin, and club drugs (ice, ketamine, cocaine, and ecstasy are endemic in most parts of the world) [8]. Seventy-nine point one percent (79.1%) of the population lives in endemic regions. In spite of all efforts to prevent and manage it, addiction is a widespread issue today that poses a serious threat to public health. Narcotic alkaloids are one of the most commonly abused substances [9,10].

Drug abuse is seen as both a family issue and an issue for the family. [11] Given the evidence that traumatic family experiences, such as child abuse, homelessness, neglect, loss, and grief, increase a person's risk of developing drug problems, it may be classified as a family problem. Because 60-80% of drug users live with or regularly interact with their families, and 2%-3% of all children under the age of 16 have parents who use drugs, drug abuse can be viewed as a family problem. The consequences could be psychological, like depression and anxiety; physical, like elevated blood pressure and ulcers; or social, like loneliness and problems at work, home, finances, and in the community.

Tramadol and rhypnol have been shown in histopathological studies to cause hepatotoxicity by altering biochemical markers and inflaming the liver, but they have no effect on the kidney at the dose and time of administration [12]. To fill in the gaps left by previous research on the combination of strong drugs, as well as to confirm it, is the goal of the current study. There is scanty information regarding the combination of these narcotics and the extraction of natural herbs to mitigate their effects. Reasons above informed this research; therefore, the study's aim is to evaluate histomorphological studies on the therapeutic potential of *Cocos nucifera (coconut*) on codeine, rohypnol, and tremadol-induced toxicity of the lungs and hearts of Albino rats.

Materials and Methods

Study Area

This research was carried out in two different locations in Nigeria: the University of Nigeria and the University of Benin Teaching Hospital, respectively. Both institutions are located in the eastern and southern geopolitical zones of the country.

Ethical consideration

The University of Nigeria Teaching Hospital in Etuku Ozuolla, Enugu, ethics and promotion committee granted authorization for the research to be conducted with clearance number 061/05/202. The guidelines for animal handling were strictly adhered to throughout the conduct of this research. [13]

Plant materials

The coconut plant purchased from New Benin, Benin City, was identified by Mr. H. Akinbosun from the Department of Plant Biology and Biotechnology, University of Benin, Nigeria.

Preparation of extracts

With an electric blender (Kenwood 1.6L, BL480 Prestons, Australia) for 7 minutes, 1.2 kg of dried coconut husks were uniformly ground up. A fine, uniform powder was produced by repeatedly grinding the coarse and rough mixtures. It was macerated in a maceration device for 24 hours at 390 °C with 1 litre of water provided by Samteck Extractions Technique GmbH, Austria. A Whatman® Grade 1 filter paper was used to filter the macerate, and after filtering, the macerate was allowed to settle. A brownish paste was produced by decanting the supernatant from the mixture and then baking it at 50 °C to dry it. [14]

Animals

Fifty (50) adult male rats weighing 200–220 g and aged five (5) weeks were obtained from the University of Nigeria (UNEC) campus animal farm. The animals were housed in wire mesh cages with a tripod that kept the animals away from their waste to avoid contamination. During the acclimatisation period, the rats were fed growers' mash, which contains ash, crude protein, crude fibre, crude fat, ether extract, nitrogen-free extract, metabolizable energy, calcium, and some essential minerals required for livestock growth, and were given free access to water.

Animal groupings

Five groups of experimental animals were created (A–E). Five (5) large cages are used to house the ten rats per group (n = 10) that are present in each group. The test groups were groups B through E, and group A served as the control group**.**

Study period

Five months were spent on the preliminary research, animal acclimatisation, drug or substance acquisition (dosage preparation and reconstitution), actual animal experiments, and result evaluation. However, the test animals received tramadol, codeine, and rohypnol for a total of 7 weeks.

Substances used during research

With the approval of the Chairman, Health Ethic Committee, University of Nigeria, 100 mg of codeine, 100 mg of swipha flunitrazepan, and 100 mg of tramadol capsules were acquired from God's Glory Pharmacy Ltd., located at 62 Chime Avenue, Enugu, Nigeria, and maintained in the refrigerator at a temperature below 30°C until use. The dosage was calculated using the formula: stock required/stock strength. The calculation and the minimal dose the animal can be exposed to, deliberately or accidentally, from previous research studies informed our dosage of 0.2 mg/kg per kilogramme of body weight.

LD_{50}s of substances

Tramadol has an oral LD_{50} of 427 mg/kg as a centrally acting analgesic [14]. Acute toxicity investigations have shown that Codeine LD_{50} values (rat, mouse, oral dosage) are approximately 300–350 mg/kg body weight. Flunitrazepam's estimated fatal dosage for people is 415 mg/kg [16].

Administration of Drugs and Extracts

Prior to the administration of the drugs (tramadol, codeine, and rohypnol), the rats were weighed. A similar weight measurement was also taken at the conclusion of every two weeks, and the average weight was noted accordingly. The following are the oral doses: Group A (the control) consumed only regular food (grower's mash) and distilled water every day for 42 days (6 weeks).

For six weeks, Group B received rat pellets, distilled water, 0.2 mg/kg bw of tramadol, and 0.2 mg/kg bw of codeine. Groups C and D received 0.2 mg/kg bw of tramadol and 0.2 mg/kg bw of rohypnol. Group E received 0.2 mg/kg bw of tramadol and 0.2 mg/kg bw of rohypnol, 0.2 mg/kg bw of codeine, and 0.2 mg/kg bw of coconut.

Collection of samples and analysis

Under chloroform anaesthesia, the serum was collected along with the lung, heart, and brain tissues after 2, 4, and 6 weeks. For histological processing, the tissues were subsequently fixed in 10% formalin. A tissue processor that automatically processed tissues was used [17].

Fixation of tissue

The tissues were cut into longitudinal and transverse slices, and they were dipped into a large amount of 10% formalin-fixed saline [17].

Processing of tissues

The fixed tissues were processed using an automatic tissue processor, targeting selected areas that showed signs of necrosis (grossing) or discoloration [17].

H and E staining protocol

The sections were dewaxed and hydrated appropriately. After being stained with hematoxylin, it was rinsed with Scot's water and dried. Slides were differentiated using 1% acid-alcohol, rinsed more, appropriately blued in running water, and counterstained with 1% eosin. The stained sections were dehydrated, cleared in xylene, and DPX mounted.

IHC staining protocol

To rinse the slides, phosphate buffered saline (PBS) was utilised. Following a 15-minute incubation with mouse plus rabbit linker and horseradish peroxidase from Genemed in the USA, sections were then washed twice for two minutes. Using an equal mixture of the substrate solution and 3,3'-diaminobenzidine hydrochloride (DAB), the chromogen was developed for 5 minutes (Genemed, USA). Each slide was drained, counterstained with hematoxylin (Jallica Scientific, Zaria), and mounted with DPX.

Biochemical analysis

Total cholesterol, triglycerides, high-density lipoprotein cholesterol in serum (HDL-C), and low-density lipoprotein cholesterol in serum (LDL-C**)** were carried out according to a technique used and described by Mohamed., *et al.* (2019) [18].

Data analysis

The obtained data were then subjected to statistical analysis using SPSS (version 20). (version 20). ANOVA (Scheffe) was used to compare the test groups' values to those of the control group with a 95% level of confidence.

Results and Discussion

In this study, the general tissue histology for group A shows that the lungs are normal. There are clear bronchioles and an alveolar ring with a clear alveolus. Group B exhibits lung histology with an

Parameters	group A	group b	p-Value	group c	P-Value	group d	P-Value	group e	P-Value
TC (Mmol/dl)	58.50±0.5	55.75±1.6	0.8	52.33±4.4	0.6	31.63±13.1	0.01*	53.33±4.6	0.10
TG (Mmol/dl)	26.50±2.5	32.50±3.2	0.1	21.33±1.3	0.2	38.00±9.8	0.05	26.67±1.9	1.02
HDL (Mmol/dl)	37.50±1.5	35.25±2.8	0.9	35.00±1.0	0.9	30.00±2.3	0.38	34.67±2.4	0.8
LDL (Mmol/dl)	15.70±0.5	16.75±1.3	0.9	11.67±4.1	0.07	11.73±3.2	0.06	13.33±5.5	0.8

Table 1: Effect of Narcotics and ameliorative of coconut on biochemical pararmeter.

* Means statistically significant ($p < 0.05$) with control and across the group

Significant reduction seen in the value of total cholesterol in group D

Keys: TC: Total Cholesterol; TG: Triglyceride; HDL: High Density Lipoprotein; LDL: Low Density Lipoprotein

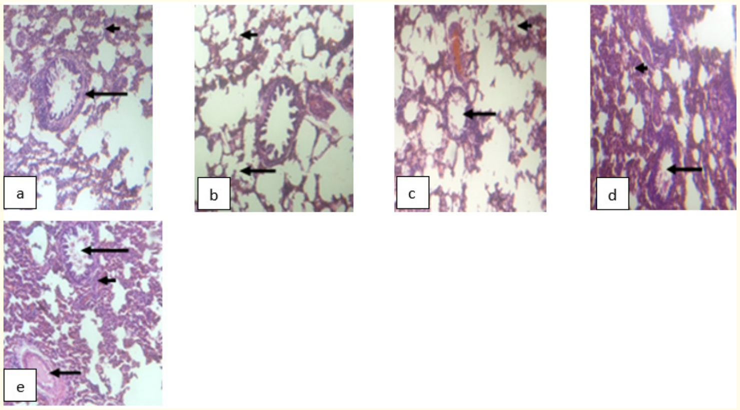

Figure a: Group A (Control): Photomicrograph of Sections of the lung of experimental animal showing prominent bronchiole (a) and alveolar ring. The alveoli appear distinct, Group B (0.2mg/kg tramadol and 0.2mg/kg codeine for 6 weeks): Photomicrograph of Sections of the lung of experimental animal showing prominent bronchiole (b) and alveolar ring. The alveoli appear distinct, Group C (0.2mg/kg tramadol and 0.2mg/kg rohypnol for 6 weeks): Photomicrograph of Sections of the lung of experimental animal showing prominent bronchiole (c) and alveolar ring. The alveoli appear slightly thickened. Group D (0.2mg/kg codeine and 0.2mg/kg rohypnol for 6 weeks): Photomicrograph of Sections of the lung of experimental animal showing prominent bronchiole (d) and alveolar ring. The alveoli appear slightly thickened (short arrow) and congested vessel (medium arrow). Group E (0.2mg/kg codeine, 0.2mg/kg rohypnol, 0.2 codeine for 6 weeks and *Cocus nucifera* extract): Photomicrograph of Sections of the lung of experimental animal showing normal bronchiole (e) and alveolar ring. X400.

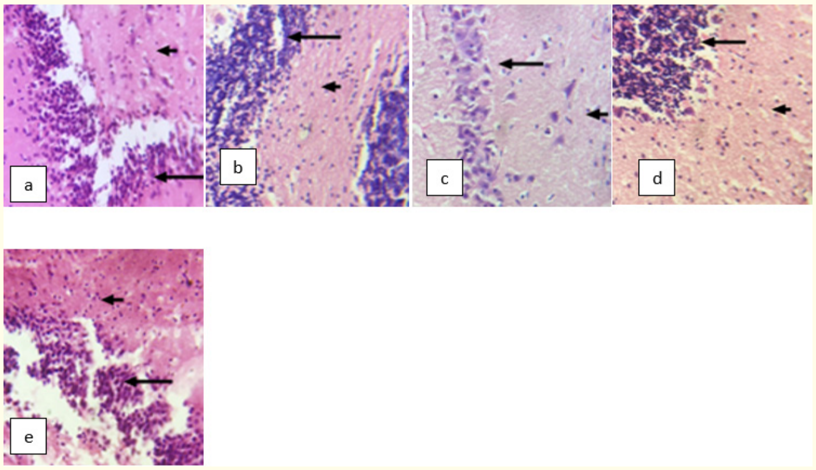

Figure 2: Group A (Control): Photomicrograph of Sections of the brain of experimental animal showing detailed features, molecular layer (a) and granule layer (long arrow) with white matter and prominent Purkinje cell layer, Group B (0.2mg/kg tramadol and 0.2mg/kg codeine for 6 weeks): Photomicrograph of Sections of the brain of experimental animal showing detailed features, molecular layer (b) and granule layer (short arrow) with white matter and reactive purkinje cell layer, Group C (0.2mg/kg tramadol and 0.2mg/kg rohypnol for 6 weeks): Photomicrograph of Sections of the brain of experimental animal showing detailed features, molecular layer (c) and granule layer (short arrow) with white matter and reactive purkinje cell layer, Group D (0.2mg/kg codeine and 0.2mg/kg rohypnol for 6 weeks): Photomicrograph of Sections of the brain of experimental animal showing detailed features, molecular layer (long arrow) and granule layer (d) with white matter and reactive purkinje cell layer, Group E (0.2mg/kg codeine, 0.2mg/kg rohypnol, 0.2 codeine for 6 weeks and *Cocus nucifera* extract): Photomicrograph of Sections of the brain of experimental animal showing normal detailed features, molecular layer (e) and granule layer (short arrow) with white matter and reactive purkinje cell layer. X400

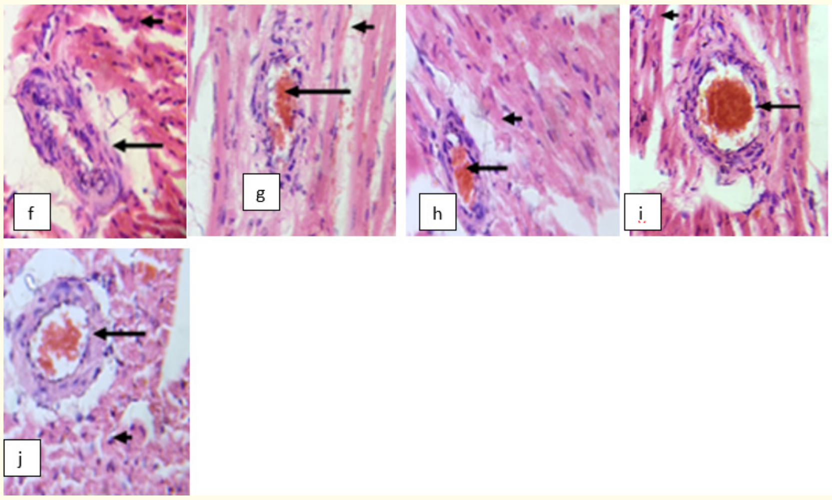

Figure 3: Group A (Control): Photomicrograph of Sections of the heart of experimental animal showing bundles of myocardial fibres (f), interstitial space and congested coronary artery (long arrow). Group B (0.2mg/kg tramadol and 0.2mg/kg codeine for 6 weeks): Photomicrograph of Sections of the heart of experimental animal showing bundles of myocardial fibres (g), interstitial space and congested coronary artery (long arrow). Group C (0.2mg/kg tramadol and 0.2mg/kg rohypnol for 6 weeks): Photomicrograph of Sections of the heart of experimental animal showing bundles of myocardial fibres artery with visible mononuclear infiltrate (h), interstitial space and congested coronary (long arrow). X400 magnification, Group D (0.2mg/kg codeine and 0.2mg/kg rohypnol for 6 weeks): Photomicrograph of Sections of the heart of experimental animal showing bundles of myocardial fibres (i), interstitial space and congested coronary artery (long arrow). Group E (0.2mg/kg codeine, 0.2mg/kg rophynol, 0.2 codeine for 6 weeks and 0.2 mg/kg bw of Cocos nucifera extract): Photomicrograph of Sections of the heart of experimental animal showing normal bundles of myocardial fibres (j), interstitial space and coronary artery (long arrow). X400

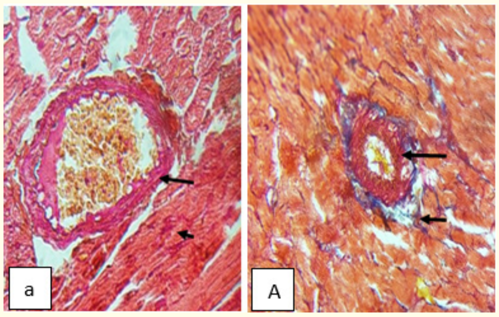

Figure 4: Group A (Control): Photomicrograph of Sections of the heart of experimental animal showing bundles of myocardial fibres coloured red reach in keratin (short arrow), interstitial space and coronary artery (a), Group E (0.2mg/kg codeine, 0.2mg/kg rohypnol, 0.2 codeine for 6 weeks and 0.2 mg / kg bw of *Cocos nucifera* extract): Photomicrograph of Sections of the heart of experimental animal showing coronary artery with fibers coloured red with reduced keratin surrounded by blue collagen deposition (long arrow) with visible myocardial fibres and deposits of collagen coloured blue (b) through the interstitial space. Heart Masson Trichrome Staining. X400.

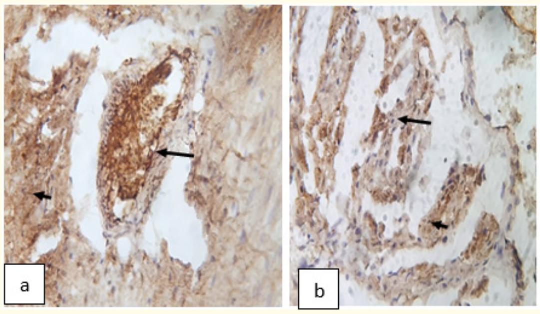

Figure 5: Group D ((0.2mg/kg codeine, 0.2mg/kg rohypnol, 0.2 codeine for 6 weeks) : Photomicrograph of Sections of the heart of experimental animal showing bundles of myocardial fibres (a), interstitial space and coronary artery (long arrow) with visible background reaction and loss of specific expression of p53, Group E (0.2mg/kg codeine, 0.2mg/kg rohypnol, 0.2 codeine for 6 weeks and *Cocos nucifera* extract): Photomicrograph of Sections of the heart of experimental animal showing Heart diffused immunoexpression of varying mild (b) to strong (short arrow) staining reaction. Heart P53 Immunohistochemistry. X400.

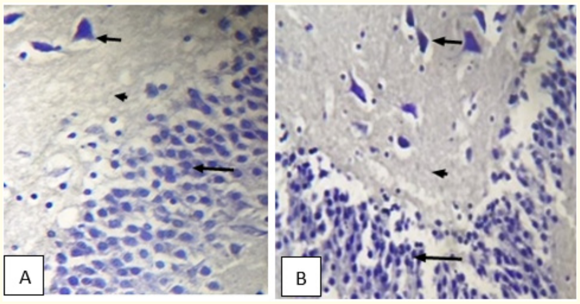

Figure 6: Group A (Control): Photomicrograph of Sections of the brain of experimental animal showing detailed features, molecular layer cells are faintly stained (short arrow) and granule layer (long arrow, A) with white matter and prominent Purkinje cell layer staining deep violet, Group D (0.2mg/kg codeine and 0.2mg/kg rohypnol for 6 weeks): Photomicrograph of Sections of the brain of experimental animal showing detailed features, molecular layer that appears deeply stained and smaller (long arrow) and granule layer (short arrow, B) with white matter and slightly enlarged pink violet stained purkinje cell layer. Brain Masson Cresyl Fast Violet Staining. X400.

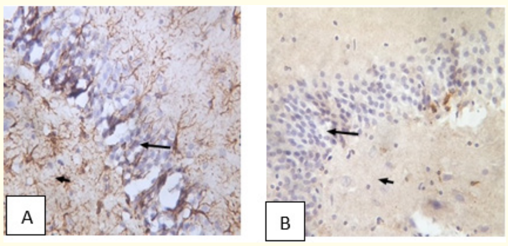

Figure 7: Group A (Control): Photomicrograph of Sections of the brain of experimental animal showing detailed features, molecular layer cells (short arrow) and granule layer (A) with total loss of bcl2 immunoexpression.-Group D (0.2mg/kg codeine and 0.2mg/kg rohypnol for 6 weeks): Photomicrograph of Sections of the brain of experimental animal showing detailed features of granule layer (long arrow) and molecular layer (short arrow, B) with visible dispersed membranous immunoexpression Bcl2immunohistochemistry Staining. X400.

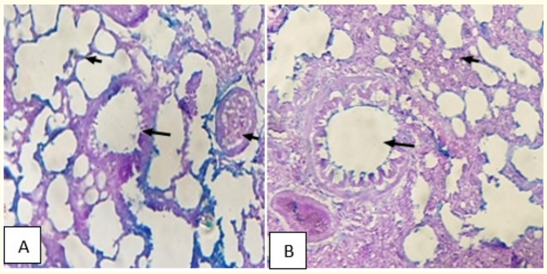

Figure 8: Group E (0.2mg/kg codeine, 0.2mg/kg rophynol, 0.2 codeine for 6 weeks and C. nucifera extract): Photomicrograph of Sections of the lung of experimental animal showing prominent bronchiole (long arrow (A) and alveolar ring. The alveolus and alveolar vein appears distinct with glycogen deposition across thickened alveoli (short arrow), Group D (0.2mg/kg codeine and 0.2mg/kg rohypnol for 6 weeks): Photomicrograph of Sections of the lung of experimental animal showing prominent dilated bronchiole (long arrow (B) and alveolar ring. The alveoli appear thickened with visible glycogen deposition and congested veins. Pas Staining .X400.

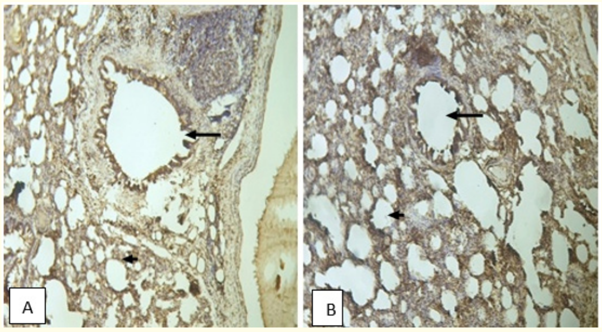

Figure 9: Group E (0.2mg/kg codeine, 0.2mg/kg rophynol, 0.2 codeine for 6 weeks and C. nucifera extract): Photomicrograph of Sections of the lung of experimental animal showing prominent bronchiole (A) and alveolar ring. The alveolus and alveolar vein appears thickened with varying TNF=alpha positive cytoplasmic staining immunoexpression (short arrow), Group D (0.2mg/kg codeine and 0.2mg/kg rohypnol for 6 weeks): Photomicrograph of Sections of the lung of experimental animal showing prominent dilated bronchiole (long arrow) and alveolar ring. The alveoli appear thickened with visible TNF=alpha positive cytoplasmic staining immunoexpression (B) Lung Tnf-Alpha Immunohistochemistry Staining.X400.

alveolar ring with a slightly thickened alveolus and a prominent bronchiole. Group C has a thickened alveolus and clogged blood vessels, as well as a prominent bronchiole and alveolar ring, when it comes to the histology of the lungs. In group D's lung histology, there is a clogged vessel, a prominent bronchiole, and an alveolar ring with a slightly thickened alveolus. None of these things can be seen in group E's lung histology, which was partly treated with Cocos nucifera.

Tramadol and codeine have been shown in animal studies to trigger respiratory stress. [19, 20] According to research by Ross., *et al.* [20]. the drawbacks of using morphine during the postoperative period include respiratory depression and histamine release. Tramadol has a very low tolerance, a little respiratory depressant effect, and the potential for physical dependence. In a similar study, tramadol (10 and 20 mg/kg) was used and demonstrated to have protective effects against transient forebrain ischemia in rats [20,21].

In a previous study that was in consonance with our findings, it was also observed that tramadol causes emphysema, which is expressed by irreversible airspace enlargement and helps promote degenerative lung function and is a major cause of chronic obstructive pulmonary disease. [22] The same source describes emphysema as an abnormal expansion of the lung, particularly the alveoli, with a loss of pulmonary elasticity [22]. Emphysema is also known to have extrapulmonary effects, including pulmonary arterial hypertension, alterations in the structure and function of the right ventricle, the wasting of skeletal muscle, and weight loss.

Similarly, according to Agusti, these systemic manifestations are linked to a higher risk of exacerbation and a lower rate of survival [23].

Tramadol and codeine can cause structural damage to the lungs, but they can also cause a long-lasting inflammatory process that includes electrolysis and fibrogenesis. According to research by Rocha., *et al*. [22], emphysema brought on by a particular hazardous chemical has been found to produce a reduction in total body mass. The sedative qualities of rohypnol may be related to the histological changes that occur after its administration; however, the exact process is still unknown [23].

In our current study, group A's photomicrograph shows a normal histomorphology of the heart, including bundles of myocardial fibres with mononuclear cells, interstitial space, and a blocked coronary artery. Heart histology in Group B shows hemorrhagic lesions, bundles of myocardial fibres with mononuclear cells, and clogged coronary arteries. Group C has myocardial fibre bundles with mononuclear cells, a mononuclear infiltrate, and interstitial space, as well as congested, dilated coronary arteries. A hemorrhagic lesion and dilated coronary arteries are seen in Group D, along with interstitial space and bundles of myocardial fibres filled with mononuclear cells. Group E shows normal myocardial fibre bundles, interstitial space, and coronary arteries. As demonstrated by Rudisil., *et al.* [24], it is well known that abuse of opioids and Rohypnol has a high incidence of cardiac complications. The findings were also in tandem with the work of Dobbe., *et al.* [25], where it was stated that opioids, especially tramadol, may result in arrhythmias, pulmonary edoema, and decreased cardiac output [25].

Rohypnol is a significant risk factor for sudden death and has significant effects on the cardiovascular system [24]. Like other stimulant addicts, those who use opioids on a regular basis have enlarged hearts [24]. Because of the actions of catecholamines, chronic opioid users may develop myocardial alterations like interstitial fibrosis, perivascular fibrosis, and small vessel dysfunction. The best technique to demonstrate it is with connective tissue stains. Using immunohistochemical markers, it has been observed that tramadol users have an increase in inflammatory cells in the myocardium, as stated in the work of Rudisil., *et al*. [24]. There are no fibrosis reports from our control studies. Previous research has shown that the presence of cardiac fibrosis in heroin users is likely due to the use of cocaine, methamphetamine, or other stimulants such as tramadol, codeine, and Rohypnol. Many drug addicts also abuse stimulants [25].

In this study, group A's general histology reveals a typical histomorphology of the brain with a prominent purkinje cell layer, molecular layer, and granule layer with white matter. Group B displays a molecular layer, granule layer, white matter, and reactive purkinje cell layer in the brain's histology. Group C displays brain histology with a reactive and slightly enlarged purkinje cell layer as well as a molecular layer, granule layer, and white matter. Group D displays brain histology with a reactive and slightly enlarged purkinje cell layer as well as a molecular layer, granule layer, and white matter. Group E displays normal brain histology, including the purkinje cell layer as well as white matter. In line with our findings, some researchers have also reported clogged submeningeal blood vessels and neuronal degeneration after tramadol administration [25].

It was also asserted that long-term morphine and other opioid use causes neuronal damage, with the various opioid effects on neuronal structure (the cytoskeleton) being recognised as markers. Again, our findings revealed that coconut was able to ameliorate the effects meted out on the brain by these substances. *C. nucifera's* curative effect could be attributed to its high phytochemical content [26].

Tramadol not only promotes oxidative stress in the brain, but it also lowers non-enzymatic antioxidants, intracellular reduced glutathione, enzymatic antioxidants, and glutathione peroxidase activity, according to previous research supporting our findings [27,28]. This finding was also coherent with that of Milardi., *et al.* [29], *wherein* the factors that contributed to the progressive loss of white matter were stated. However, the damaging effects were ameliorated by administering coconut oil along with narcotics. The effectiveness of the treatment using coconuts may have resulted from the antioxidant properties of some of the key components of coconuts, such as tannin, saponin, and phenol [30].

In addition to phenols, tannins, leucoanthocyanidins, flavonoids, triterpenes, steroids, and alkaloids, coconut extract also contains saponins, triterpenes, and condensed tannins [30,31]. Importantly, edible fruits, vegetables, and many herbs are rich in antioxidant active components like flavonoids, which may help explain how the effects of tramadol, royphnol, and codeine were reduced [32-35]. Proteins, carbohydrates, polyphenols, flavonoids, and tannins are examples of phytonutrients that are known to have antioxidant activities [35,37].

Coconuts could be recommended as supplements to people who are addicted to codeine, rohypnol, or tramadol or who intentionally abuse the aforementioned substances and are in need of treatment.

Conclusion

Our results demonstrate that chronic exposure to rohypnol, tramadol, and codeine causes progressive disruptions in brain, lung, and heart cellular architecture. These disruptions may be the underlying cause of some cerebellar lesions that result in clinical conditions like meningitis, headache, stroke, cerebellar edoema, vasculitis, and dementia, as well as ataxia, tremors, abnormal gait patterns, and speech abnormalities. This study further reveals the remedial or ameliorative effects of coconut on the aforementioned deleterious effects caused by narcotics. *C. nucifera* may provide relief by lessening the harmful effects that may have been generated from the abuse of rohypnol, codeine, and tramadol and, therefore, should be recommended as supplements.

Conflict of Interest

The authors declare that there is no conflict of interest.

Authors' declaration

The authors hereby state that the work they have presented in this article is original and that they will be responsible for any claims relating to its content.

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