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The Effect of Tender Coconut Water on Cardiovascular Changes (Heart) Induced by Secondhand Cigarette Smoke in Male Wistar Rats

Aanuoluwapo Janet Adedokun[1] and Olulola Olutoyin Oladapo[1,2]*

[1]Department of Anatomy, Faculty of Basic Medical Sciences, University of Ibadan, Ibadan, Nigeria

[2]Cardiology Unit, Department of Medicine, Faculty of Clinical Sciences, College of Medicine, University of Ibadan, Nigeria

Abstract

Background: Second Hand Smoke (SHS) exposes nonsmokers to harmful chemicals that contribute to cardiovascular diseases, including endothelial dysfunction and increased oxidative stress. Tender Coconut Water (TCW), rich in antioxidants and essential nutrients, has been suggested as a potential natural intervention to mitigate SHS-induced cardiovascular damage.

Objective: This study aimed to evaluate the effects of TCW on structural and physiological changes in the heart and aorta of rats exposed to varying doses of SHS.

Methodology: Thirty-five male Wistar rats were divided into seven groups (n=5 per group). Group A served as the control, while Groups B, C, and D was exposed to 5, 10, and 15 Stick of Cigarette (SoC) smoke, respectively, twice daily, six days a week. Groups E, F, and G received the same SHS exposure as Groups B, C, and D but were additionally treated with 4 mL of TCW daily twice daily for four weeks. Blood pressure and body weight were monitored weekly, and at the end of the study, histological and biochemical, analyses were performed on the heart to assess collagen deposition and Nitric Oxide (NO) levels and structural changes in the heart.

Result: The results demonstrated that SHS exposure led to significant structural changes, including increased collagen deposition, and reduced NO bioavailability, contributing to hypertension and cardiovascular dysfunction. Rats in Groups E and F, which received TCW, exhibited partial improvements in blood pressure regulation, NO levels, and reduced fibrosis, particularly at lower SHS exposure levels. However, TCW was less effective in mitigating cardiovascular damage in rats exposed to high SHS doses (Group G), suggesting that its protective effects may be dose-dependent.

Conclusion: In conclusion, TCW shows potential as a natural cardioprotective agent against SHS-induced cardiovascular damage, particularly at lower exposure levels. However, its efficacy at higher SHS doses remains limited. Further research is needed to explore optimal dosing, long-term effects, and mechanisms underlying its protective properties.

Keywords: Tender Coconut Water; Second Hand Smoke; Cardiovascular Health; Nitric Oxide; Blood Pressure; Fibrosis

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Correspondence:
Aanuoluwapo Janet Adedokun, Department of Anatomy, Faculty of Basic Medical Sciences, University of Ibadan, Ibadan, Nigeria, E-mail: adedokunaanuoluwapo95@gmail.com

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Abbreviations

BP: Blood Pressure; DBP: Diastolic Blood Pressure7; SHS: Second Hand Smoke; HR: Heart Rate; TCW: Tender Coconut Water; NIBP: Non-Invasive Blood Pressure; SBP: Systolic Blood Pressure; SoC: Stick of Cigarette; WHO: World Health Organization

Introduction

Second Hand Smoke (SHS), also known as Environmental Tobacco Smoke (ETS), is the combination of smoke exhaled by smokers and smoke emitted from the burning end of tobacco products such as cigarettes, cigars, or pipes. SHS contains over 7,000 chemicals, with hundreds being toxic and approximately 70 identified as carcinogenic [1]. Exposure to SHS places nonsmokers at risk for numerous health complications, including respiratory infections, cardiovascular diseases, and various cancers4. SHS consists of two major components: side stream smoke and mainstream smoke. Side stream smoke is released directly from a burning tobacco product into the surrounding

air, making it the primary component of SHS [2].

Although active smokers receive significantly higher doses of smoke exposure than passive smokers, SHS still presents a considerable risk to cardiovascular health. The relative risk of coronary heart disease is estimated to be 1.78 for smokers compared to 1.31 for those exposed to SHS [3]. Evidence indicates that the cardiovascular system is highly sensitive to SHS toxins, leading to adverse effects even at low exposure levels. In the United States alone, SHS is responsible for nearly 34,000 premature deaths from cardiovascular disease among nonsmokers each year [4].

Exposure to SHS has immediate effects on the cardiovascular system, primarily due to its impact on Nitric Oxide (NO) bioavailability. SHS exposure leads to increased oxidative stress and catecholamine levels, which reduce NO bioavailability and contribute to endothelial dysfunction and hypertension [5].

Tender Coconut Water (TCW) is a natural beverage rich in potassium, magnesium, L-arginine, and antioxidants [6]. Its health benefits include antihypertensive and cardioprotective effects [7, 8]. TWC has shown potential cardioprotective effects, the majority of studies have been conducted on human subjects or *in vitro* settings. Animal studies assessing TCW's impact on cardiac oxidative stress, lipid metabolism, and myocardial function are scarce [9]. There is a need for controlled animal trials to evaluate TCW's role in mitigating SHS-induced cardiac damage, particularly in terms of reducing fibrosis, improving heart rate, and modulating inflammatory and apoptotic pathways. Furthermore, the dose-dependent effects of TCW in animal models require further exploration to determine its optimal therapeutic potential [8].

Second Hand smoke (SHS), a harmful combination of side stream and exhaled mainstream smoke, significantly reduces plasma Nitric Oxide (NO) levels, primarily through oxidative stress, endothelial dysfunction, and inflammation [10]. The reactive oxygen species in SHS rapidly degrade NO by forming peroxynitrite, a toxic compound that diminishes NO bioavailability and damages blood vessels. Additionally, SHS impairs the function of endothelial Nitric Oxide Synthase (eNOS), the enzyme responsible for NO production [11], and promotes systemic inflammation, both of which further suppress NO synthesis. Reduced NO levels compromise vascular relaxation, increase blood pressure, and contribute to the development of cardiovascular diseases [1]. Even short-term exposure to SHS has been shown to lower plasma nitrite - a stable indicator of NO levels - highlighting its immediate and harmful impact on vascular health. Vulnerable groups such as children, pregnant women, and individuals with preexisting conditions are especially at risk.

Materials and Methods

Animals and experimental design

Thirty-five male Wistar rats were randomized into seven groups (Table 1). SHS exposure was delivered in a specially designed chamber (Figure 1).

TCW was freshly extracted daily and administered orally according to methods by Bhagya *et al.,* [5].

The body weight was monitored, and blood pressure was measured using a non-invasive tail-cuff method [12]. Plasma nitric oxide levels were assessed using the Griess Reagent Assay [10]. Also, Hearts and aortas were processed and stained with Hematoxylin-Eosin and Masson's Trichrome stains for microscopic evaluation. Data were analyzed using one-way ANOVA. A p-value <0.05 was considered statistically significant.

Results

General observations

SHS-exposed rats showed signs of fatigue and decreased activity, consistent with prior findings [4].

Body weight

A significant reduction in average body weight was observed in all groups (Table 1) exposed to cigarette smoke compared to the control group as seen in Figure 2. By the second week, rats in the group 2, 3 and 4 (exposed to 5, 10, and 15 SoC respectively) exhibited a steady decline in weight, with group 4 (exposed to 15 SoC) experiencing the most pronounced weight loss (p=0.0023). Specifically, the average weights by week 2 were (164g±2.35, 158g±3.01, and 153±2.59g) for the groups, 2, 3 and 4, respectively, compared to (175±2.87g) in group 1 (control).

In contrast, group 5, 6 and 7 supplemented with 4 mL of TCW alongside cigarette exposure, (5 SoC+4 mL TCW, 10 SoC+4 mL TCW, 15 SoC+4 mL TCW respectively) showed slight improved weight retention compared to their non-TCW counterparts. Specifically, group 5 (exposed to 5 SoC+4 mL TCW) exhibited a lesser degree of weight reduction, with an average weight of (166g±1.24g) by week 2 as seen in Figure 2, though the difference was not statistically significant (p=0.0654). Group 6 (exposed to 10 SoC+4 mL TCW) had an average weight of (160g±2.43g), while group 7, (exposed to 15 SoC+4 mL TCW) weighed (155g±2.45g) at week 2. Notably, by week 4, group 7 still showed a significant weight reduction (p=0.0041) compared to

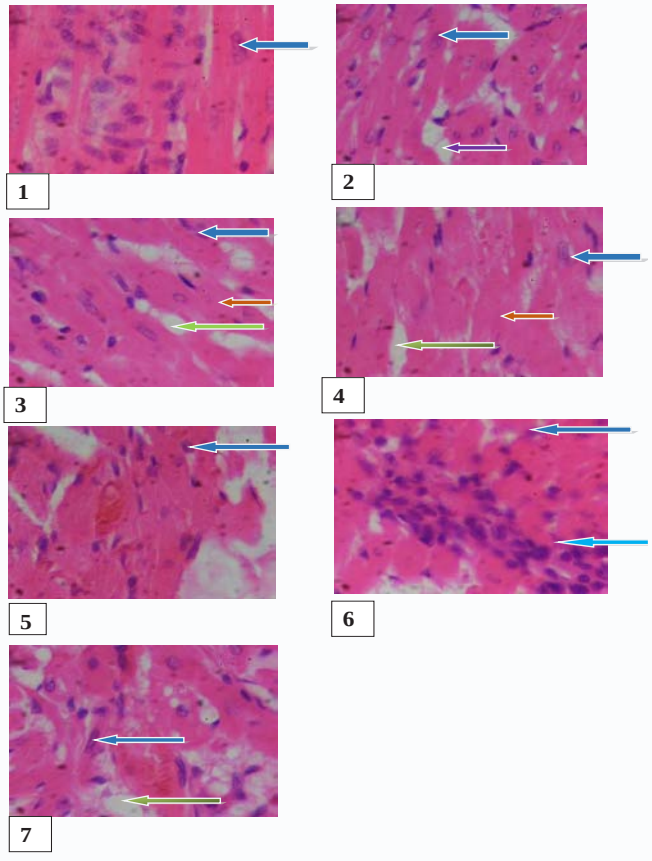

Figure 1:

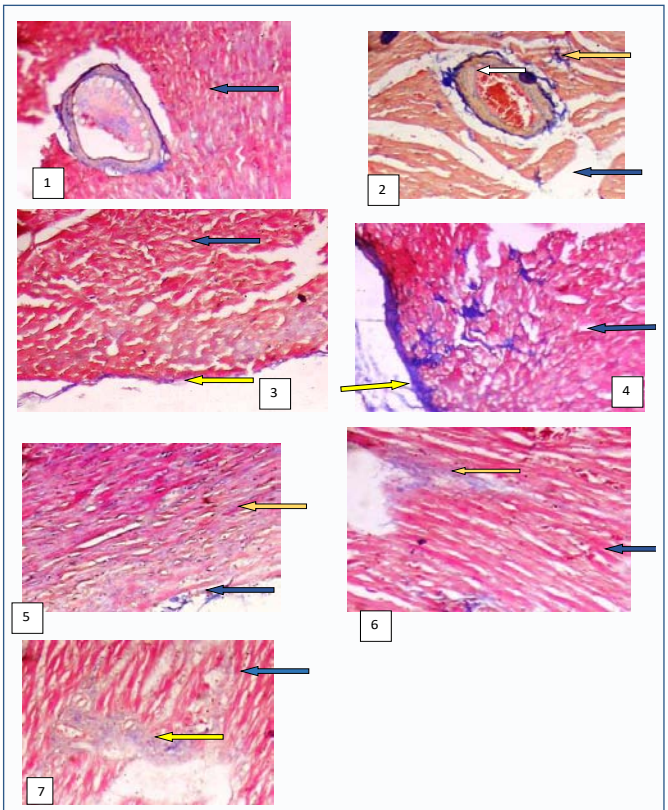

Figure 2:

the control group, weighing (148g±3.42 *versus* 182g±2.99 in control group 1).

By the fourth week, all groups exposed to cigarette smoke maintained significantly lower body weights compared to the control group. Group one; the control group exhibited a steady increase in weight, reaching an average of 182g±2.99g. In contrast, group 2, 3 and 4 *i.e.,* weighed 158g±3.21g, 150g±2.51g and 145g±, 2.44g respectively, with statistically significant weight reductions compared to the control (p=0.0008, p<0.001, and p=0.0023, respectively).

Similarly, while groups supplemented with 4mL of TCW showed slightly improved weight retention, their weights remained significantly lower than the control. By week 4, group 5, 6 and 7 weighed 160g±1.89g, 153g±3.41g, and 148g±2.67g, respectively. The weight reductions in these groups were also statistically significant when compared to the control (p=0.0012, p=0.0009, and p=0.0041, respectively).

The control group exhibited a progressive increase in body weight throughout the experiment, maintaining significantly higher weight levels than all cigarette-exposed groups (p<0.001 across multiple weeks).

Overall, the findings suggest that exposure to cigarette smoke induced weight loss in a dose-dependent manner, while TCW supplementation may provide partial but insufficient protection against weight reduction as seen in Figure 2.

Blood pressure

Systolic Blood Pressure (SBP): A significant increase in average Systolic Blood Pressure (SBP) was observed in all treatment groups exposed to SoC compared to the Control group throughout the experimental period According to Figure 3. By week 1, Groups 2, 3, and 4 (5 SoC, 10 SoC, and 15 SoC) showed an initial elevation in SBP compared to the Control group, with mean values of approximately 120.45±2.31 mmHg, 125.67±2.78 mmHg, and 130.23±2.92 mmHg, respectively.

By week 2, a further increase in SBP was noted in all exposure groups (Groups 2, 3, and 4), with Group 4 displaying the highest SBP (138.78±3.21 mmHg), significantly different from the Control group (p=0.0019). Group 5, 6, and 7 (exposed to 5 SoC+4mL TCW, 10 SoC+4mL TCW, and 15 SoC+4mL TCW) also showed a progressive increase, reaching 122.45±2.67 mmHg, 128.56±2.89 mmHg, and 133.45±3.11 mmHg, respectively (p=0.0032, p=0.0025, and p=0.0007).

At week 3, the SBP values remained elevated, with Group 4 exposed to 15 SoC reaching 145.23±3.45 mmHg, significantly higher than the Control group (p=0.0005), as seen in Figure 3 with Groups 2 and 3 following closely. Additionally, Groups 5, 6, and 7 (5 SoC+4mL TCW, 10 SoC+4mL TCW, and 15 SoC+4mL TCW) demonstrated increased SBP (130.67±3.02 mmHg, 135.78±3.18 mmHg, and 140.23±3.31 mmHg), with significant differences compared to the Control group (p=0.0020, p=0.0012, and p=0.0009, respectively).

By week 4, the highest SBP values were recorded in Group 4 (155.67±3.67 mmHg), followed by Group 7 (150.89±3.89 mmHg with p=0.0028,), Group 6 (147.23±3.57 mmHg with p=0.0016), Group 3 (148.45±3.42 mmHg), and Group 2 (142.34±3.19 mmHg). Similarly, Group 5 maintained elevated SBP values (140.89±3.23 mmHg with p=0.0004) which is statistically significant differences compared to the Control group.

Throughout the study period, no significant difference was observed between Groups 5, 6, and 7. Similarly, comparisons among Groups 2, 3, and 4 revealed no statistically significant differences despite their increasing SBP trends.

These findings suggest that exposure to SoC led to a sustained increase in systolic blood pressure over time. The administration of TCW did not significantly attenuate this rise, as SBP values in the TCW-supplemented groups remained comparable to their respective SoC groups (Figure 3). This indicates that TCW did not significantly counteract the hypertensive effect SoC chronic exposure.

Diastolic Blood Pressure (DBP): A significant increase in average Diastolic Blood Pressure (DBP) was observed in all treatment groups exposed compared to the Control group 1 throughout the experimental period as seen in Figure 4. By week 1, Group 2, 3 and 4 showed an initial elevation in DBP compared to the Control group 1, with mean values of approximately 95.23±2.15 mmHg, 100.56±2.48mmHg, and 105.12±2.63 mmHg, respectively.

By week 2, a further increase in DBP was noted in all exposure groups, group 4 with exposure to group 4 displaying the highest DBP (115.78±2.98 mmHg), significantly different from the Control group 1 (p=0.0021). Group 5 and 6 also showed a progressive increase (Figure 4), reaching 103.45±2.75 mmHg and 108.67±3.02 mmHg, respectively (p=0.0029 and p=0.0011).

At week 3, the DBP values remained elevated, with group 4 reaching 123.23±3.15 mmHg, significantly higher than the Control group 1 (p=0.0008), group 2 and additionally, group 5 and 6 respectively demonstrated increased DBP (110.89±2.94 mmHg and 118.45±3.12 mmHg), with significant differences compared to the Control group (p=0.0015 and p=0.0009, respectively).

By week 4, the highest DBP values were recorded in Group 4 (130.56±3.34 mmHg), followed by Group 7 (128.78±3.54 mmHg, p=0.0023), Group 3 (125.78±3.21 mmHg), and Group 6 (124.56±3.31 mmHg, p=0.0015). Group 5 maintained elevated DBP values (120.67±3.09 mmHg, p=0.0006), with statistically significant differences compared to group 1 (<000.1 respectively).

Throughout the study period, no significant difference was observed between the Group 5, 6 and 7. Similarly, comparisons among the Group 2, 3 and 4 revealed no statistically significant differences despite their increasing DBP trends. These findings suggest that exposure to cigarette smoke led to a sustained increase in diastolic blood pressure over time. The administration of TCW did not significantly attenuate this rise, as DBP values in the TCW-supplemented groups remained comparable to their respective SoC groups (Figure 4). This indicates that TCW did not significantly counteract the hypertensive effects of chronic cigarette exposure.

Significant increases in systolic and diastolic blood pressure were observed in SHS-exposed groups [13]. TCW intervention reduced blood pressure, particularly in low- and moderate-exposure groups.

Nitric oxide levels

This study in Fig 5 investigated the effect of cigarette smoking at various doses and the potential protective role of Tender Coconut Water (TCW) on plasma nitrite levels, a key indicator of Nitric Oxide (NO) availability and vascular function.

The control group exhibited the highest plasma nitrite concentration (≈14.5 nmol/ml), representing the baseline NO status in non-smoking conditions. In contrast, groups exposed to cigarette smoke showed a dose-dependent decline in plasma nitrite levels. Specifically, exposure to 5 stick SoC (Group 2) reduced nitrite levels to approximately 12.5 nmol/ml (p<0.05 *vs.* control), while 10 sticks (Group 3) and 15 sticks (Group 4) further decreased nitrite levels to about 11.5 nmol/ml and 10.5 nmol/ml, respectively (p<0.01 *vs.* control for both). These findings are consistent with the established detrimental effects of cigarette smoke, which impairs endothelial function and nitric oxide synthesis through oxidative stress and inflammatory pathways.

Interestingly, co-administration of 4 ml TCW alongside cigarette exposure yielded a protective effect. In Group 5 (5 SoC+TCW), plasma nitrite increased to approximately 13.2 nmol/ml, a significant improvement compared to Group 2 (p<0.05) and not significantly different from control (p>0.05). Similarly, Group 6 (10 SoC+TCW) showed a nitrite level of about 12.2 nmol/ml, significantly higher than Group 3 (p<0.05) but still lower than control (p<0.05). Group 7 (15 SoC+TCW) had a nitrite level of roughly 11.2 nmol/ml, also significantly improved compared to Group 4 (p<0.05) but lower than control (p<0.01).

These results suggest that tender coconut water mitigates the reduction in plasma nitrite caused by cigarette smoking, likely due to its antioxidant-rich composition - including ascorbic acid, L-arginine, phenolic compounds, and cytoprotective electrolytes. While TCW did not completely restore nitrite levels to those of the control group at higher smoking doses, its partial ameliorative effect indicates potential vascular protective benefits.

In conclusion, cigarette smoking significantly reduces plasma nitrite levels in a dose-dependent manner, reflecting impaired NO production or availability. Tender coconut water shows promise as a natural intervention, capable of counteracting the harmful vascular effectSoCigarette exposure, particularly at lower doses.

Histological findings

Histological analysis revealed collagen deposition, and vascular remodelling following SHS exposure [2]. TCW administration reduced these pathological changes, especially at lower SHS doses.

PLATE 1: H & E staining X400 shows group (1) cardiomyocytes of the myocardium (blue arrow), group (2) showed the cardiomyocytes of the myocardium appear enlarged (blue arrow) indicating mild hypertrophy also, fibrosis deposition is seen (Purple arrow) which indicate an onset of infarction. Group (3) showed the cardiomyocytes of the myocardium appears mildly enlarged (blue arrow), also necrosis of the nuclei (golden yellow arrow) and infarction of the myocardium seen (light green). Group (4) showed necrotic nuclei in the myocardial layer (golden yellow arrow), also, infarction of the myocardial is seen (light green arrow) and the cardiomyocy appeared mildly enlarged (blue arrow). Group 5 the cardiomyocyte appears moderately normal (blue arrow), group (6), the cardiomyocytes of the LV appear mildly enlarged (blue arrow) inducing ventricular hypertrophy, also, infilteration of cell to a focus indicate inflammation (light blue arrow). Group 7 showed enlarged cardiomyocytes indicating ventricular hypertrophy (blue arrow) there is evidence of myocardial infarction (light green).

Mason Trichrome (MT) staining of the heart

PLATE 2: MT staining X400 of heart sections stained with Masson's Trichrome, illustrating varying degree of collagen fiber deposition across groups. Mason Trichrome stains collagen fiber blue. Group 1 shows no collagen deposits, while Groups 2 show mild collagen deposition (yellow arrow). Group 3 exhibited mild to moderate deposits at the interspace of cardiomyocytes (yellow arrow). Group 4 exhibited high deposit of collagen fibers (yellow arrow). Group 5, 6, 7 that takes tender coconut water exhibit mild deposition of collagen fibers on the myocardium (yellow arrow), Normal cardiac muscle appears red in all groups (blue).

Discussion

This study examined the structural and physiological impact of Second Hand Smoke (SHS) on the heart of Wistar rats, while evaluating the potential protective role of Tender Coconut Water (TCW). SHS exposure led to significant weight loss, elevated systolic and diastolic blood pressure and plasma nitrite levels - markers of Cardiac hypertrophy and oxidative stress. Histological analysis confirmed dose-dependent collagen deposition in cardiac tissues of SHS-exposed rats, with Group 1 (control) showing none, and Groups 2–4 exhibiting mild to moderate fibrosis. These effects mirror findings from Wu *et al.,* and Wei-Wen *et al.,* confirming the link between SHS and cardiovascular remodelling.

Although TCW administration offered minor improvements - slightly lowering blood pressure, collagen deposition, and nitrite levels - these effects were not statistically significant at higher SHS exposures, suggesting limited protective efficacy. Findings from Heiss *et al.,* [3] also corroborate the reduction in Nitric Oxide (NO) bioavailability due to SHS, aligning with the observed nitrite decline.

Notably, Group 5 (lowest SHS dose+TCW) demonstrated marked reductions in collagen buildup and blood pressure, suggesting dose-dependent cardio protection, consistent with Bhagya *et al.,* [5] and Rini *et al.,* However, in Groups 6 and 7 (higher SHS doses), TCW's

effects diminished, supporting Syafriani *et al.,* and Sun *et al.,* who stress the importance of dose optimization in antioxidant therapy.

The study concludes that while TCW shows promise as a cardioprotective agent, especially at lower SHS exposure, its efficacy is limited under prolonged or intense exposure. These results align partly with Alleyne *et al.,* though full reversal of SHS effects was not achieved. TCW moderately modulated blood pressure but failed to fully prevent SHS-induced damage, underscoring the need for SHS avoidance and further exploration of TCW's therapeutic potential [14].

Conclusion

Tender coconut water demonstrates cardioprotective effects against SHS-induced cardiovascular damage in Wistar rats, particularly at lower exposure levels. Further studies are needed to explore optimal dosing and long-term outcomes.

Recommendations

- Promote awareness of SHS dangers and support smoke-free environments.
- Incorporate natural antioxidants like TCW as a supplementary dietary measure.
- Conduct further research into TCW's molecular mechanisms and optimal dosing.
- Strengthen public health policies to protect non-smokers.
- Explore long-term TCW use in mitigating cardiovascular effects of SHS.

Limitations

Budgetary constraints restricted the study's scope, preventing comprehensive biochemical assays and limiting exploration of other antioxidants and therapeutic agents.

References

1. Lushniak BD, Samet JM, Pechacek TF, Norman LA, Taylor PA. The health consequences of smoking—50 years of progress: A report of the surgeon general. 2014.
2. Yong JW, Ge L, Ng YF, Tan SN. The chemical composition and biological propertieSoCoconut (Cocos nucifera L.) water. Molecules. 2009; 14(12): 5144-5164.
3. Heiss C, Amabile N, Lee A, Real W, Wassmann S. Secondhand smoke impairs endothelial function in healthy adults and decreases nitric oxide bioavailability. J Am College of Cardiology. 2015; 65(6): 606-615.
4. Wu JP. Secondhand Smoke Exposure Triggered Respiratory Cardiothoracic Diseases: Secondhand Smoke Exposure Induces Harmful Health. Partridge Publishing. 2021.
5. Bhagya D, Prema L, Rajamohan T. Therapeutic effects of tender coconut water on oxidative stress in fructose fed insulin resistant hypertensive rats. Asian Pac J Trop Med. 2012; 5(4): 270-276.
6. Burke H, Leonardi-Bee J, Hashim A, Pine-Abata H, Chen Y, Cook DG, et al. Prenatal and passive smoke exposure and incidence of asthma and wheeze: systematic review and meta-analysis. Pediatrics. 2012; 129(4): 735-744.
7. Wang R, LeLaurin J, Carrier A, Churba B, Karanth S, Yoon HS, et al. Trends and factors associated with secondhand smoke exposure among US cancer survivors, 2013–2020. Cancer. 2023; 129(19): 3053-3063.
8. Bryan NS, Bian K, Murad F. Discovery of the nitric oxide signaling pathway and targets for drug development. Front Biosci. 2009; 14(1): 1-18.
9. Tran N, Garcia T, Aniqa M, Ali S, Ally A, Nauli SM. Endothelial nitric oxide synthase (eNOS) and the cardiovascular system: in physiology and in disease states. Am J Biomed Sci Res. 2022; 15(2): 153-177.
10. Zhang Q, Zeng G, Wang X, Wu KH. Associations of exposure to secondhand smoke with hypertension risk and blood pressure values in adults. Environ Health Prev Med. 2021; 26: 1-11.
11. Cardiff RD, Miller CH, Munn RJ. Analysis of mouse model pathology: a primer for studying the anatomic pathology of genetically engineered mice. Cold Spring Harb Protoc. 2014; 2014(6): 561-580.
12. Agbafor KN, Elom SO, Ogbanshi ME, Oko AO, Uraku AJ, Nwankwo VUO, et al. Antioxidant property and cardiovascular effectSoCoconut (Cocos nucifera) water. Int J Biochem Res Rev. 2015; 5(4): 259.
13. Prabhakar P. Role of Indian fruits in the prevention and management of hypertension. J Pract Cardiovasc Sci. 2022; 8(3): 135-143.
14. Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking. Circulation. 2005; 111(20): 2684-2698.