Journal of International Medical and Health Sciences

scholixonline.org

Research Article

Cite this article: Elleh, B. I. (2026). Effects of herbal cosmetics on serum lipid profile and atherogenic coefficient in rabbit model: A toxicological study.

Received: 02 October 2026
Revised: 14 October 2026
Accepted: 15 October 2026

Keywords:
Herbal cosmetics, Serum lipid profile, Atherogenic coefficient and Rabbit model

Corresponding author:
Elleh, B.I

© The Author(s), 2026. Published by Scholix publishers. This is an Open Access article, distributed under the terms of the Creative Commons Attribution licence (https:// creativecommons.org/licenses/by/4.0/), which permits unrestricted re-use, distribution and reproduction, provided the original article is properly cited.

Effects of Herbal Cosmetics on Serum Lipid Profile and Atherogenic Coefficient in Rabbit Model: A Toxicological Study

Elleh, B.I[*1], Thompson, I.N[2]

[1]Department of Chemical Pathology, Federal University Otuoke, Bayelsa State, Nigeria
[2]Department of Chemical Pathology, Rivers State University, Port Harcourt, Nigeria

ABSTRACT

The aim of this study was to evaluate the effect of three commonly marketed herbal cosmetics in Port Harcourt on serum lipid profile and atherogenic coefficient in rabbit. Three herbal oils were named sample A, B, C and a total of 48 rabbits grouped into four were used for the study**.** Rabbits from each group were treated for thirty, sixty and ninety days respectively with 0.5ml/kg of Samples A, B, C and distilled water as control. At days thirty, sixty and ninety, respectively, four rabbits from each group were sacrificed under chloroform anesthesia. Blood samples were collected to determine biochemical parameters at intervals of, 30days, 60days and 90days respectively. GraphPad Prism v.7.0 was used for statistical analysis and $p<0.05$ was considered statistically significant. The result of lipid profile and atherogenic coefficient for day 30 and 60 showed no significant difference between sample A, B, C and control. While day 90 showed significant difference in concentrations of lipid profile and atherogenic coefficient in sample A, B and C compared to control. Also, the concentrations of heavy metals exceeded the maximum allowable concentrations limit set by WHO. The results reflected the impact of heavy metals in the cosmetics on lipid profile and atherogenic coefficient of the rabbits. Conversely, prolonged use of these different cosmetic brands would alter lipid profile and atherogenic coefficient of the recipients.

Introduction

Cosmetics are products used to improve or change the physical looks of the human face and skin by applying it on the face, hair, and skin. Cosmetics are generally a mixture of different synthethic or natural compounds, produced from either synthetic or plant sources. There are two major types of cosmetic products, which are classified based on the source of the raw material used for production. They include synthetic and herbal cosmetics. When chemical compounds are used in the creation of the beauty product, it is known as a synthetic cosmetic. While herbal cosmetics involves the use of parts or whole of plant material in the manufacturing of the beauty product. There is an all-encompassing variety of herbal cosmetics that are manufactured and commonly used for everyday purposes. These herbal cosmetics are produced from a plant source [1].

The upsurge in the use of herbal cosmetics is because they are believed to be safer for health than the synthetic ones [2]. However, scientific research has discovered that not all herbal products are safe, as per general belief, some are quite toxic, having several adverse effects, polluted with heavy metals and contain phytotoxins or biotoxins [3]. Plant-based cosmetics are generally regarded as safe because they are produced from natural plants. Notwithstanding, there are cases of severe side effects after their use and the toxicity has been traced to contaminants and adulteration from poor manufacturing processes. This resulted from using plants contaminated with heavy metals from the environment or soil.

Exposure to heavy metals has been increasingly implicated in the disruption of lipid metabolism and the development of dyslipidemia. Heavy metals such as lead (Pb), cadmium (Cd), and mercury (Hg) are ubiquitous environmental and cosmetic contaminants that enter the human body through dermal absorption, inhalation, and ingestion. Once absorbed, these metals accumulate primarily in the liver and kidney,

organs that are central to lipid synthesis, metabolism, and clearance [4]. Several epidemiological and experimental studies have demonstrated a significant association between chronic heavy metal exposure and alterations in serum lipid parameters, including total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C).

The primary mechanism underlying metal-induced dyslipidemia is oxidative stress. Lead, cadmium, and mercury are known to generate reactive oxygen species (ROS), which initiate lipid peroxidation in hepatic cell membranes [5]. Lipid peroxidation leads to hepatocellular damage and impairment of key enzymes involved in lipoprotein metabolism, such as lecithin-cholesterol acyltransferase and lipoprotein lipase. Consequently, this results in elevated levels of TC, TG, and LDL-C, with a concomitant decrease in HDL-C [6]. In addition, heavy metals bind to sulfhydryl groups of antioxidant enzymes, thereby depleting glutathione and further exacerbating oxidative damage [7]. The dyslipidemic pattern associated with heavy metal exposure — elevated TC, TG, LDL-C and reduced HDL-C — translates to increased atherogenic risk. This is reflected in elevated cardiovascular risk indices such as the Atherogenic Index of Plasma (AIP), Cardiac Risk Ratio (CRI-I), and Atherogenic Coefficient (AC) [8].

[9] reported that several facial creams and skin-lightening products in circulation contained lead, cadmium, and mercury at concentrations exceeding WHO permissible limits. The authors emphasized that chronic dermal application of such products could result in systemic metal accumulation and subsequent liver and kidney toxicity, which may indirectly alter lipid profiles. Although direct human studies linking cosmetic use to lipid indices remain limited, evidence from occupational and environmental exposure studies provides biological plausibility for this relationship.

MATERIALS AND METHOD

PROCUREMENT OF HERBAL COSMETICS

Three (3) types of commonly used herbal hair oils namely Allthingsnatural by Emi Herbal Oil, Amal Botanical Shea Oil and Kakiva herbal oil were purchased from a Supermarket in Port Harcourt and labelled product A (Allthingsnaturalby Emiherbaloil), B (Amal Botanical Shea Oiland C (Kakiva Herbal Oil) respectively.

EXPERIMENTAL ANIMALS: A total of fifty-two (52), two-month-old, New Zealand white rabbits (*Oryctolagus cuniculus*) that weighed between 1.2 - 1.5 kg were used for this study. The rabbits were purchased from a breeder in Port Harcourt, Rivers State. Four (4) rabbits wereused asbaselinecontrol, while the remaining forty eight (48) rabbits were divided into three (3) groups (A, B and C) of twelve (12) rabbits each with matched control.

PLACE AND DURATION OF STUDY: This study was carried out at Animal House, Applied and Environmental Biology Department, Rivers State University, Port Harcourt, Rivers State, Nigeria, between April, 2020 and November, 2020.

STUDY DESIGN: Total of 48 rabbits grouped into four were used for the study**.** Rabbits from each group were treated for thirty, sixty and ninety days respectively with 0.5ml/kg of Samples A, B, C and distilled water as control. Blood sample of rabbits in all the groups was collected at intervals, 30, 60 and 90 days respectively.

SAMPLE COLLECTION

Prior to blood sample collection, the rabbits were anesthetized using diethyl ether then, surgical blade was used to cut the jugular vein of the rabbits, 5 moonblood samples were collected and dispensed into plain bottle. The samples were allowed to retract, then spun for 15 minutes to separate sera for determination of biochemical parameters.

BIOCHEMICAL ANALYSIS:

The heavy metals were measured using an atomic absorption spectrophotometer. Lipid profile was measured using Randox Laboratories United Kingdom reagent kits

STATISTICAL ANALYSIS

Data are expressed as mean ± SD. Statistical differences between groups were computed using Graph pad prism 7.0 versions. Results were analyzed using one-way analysis of variance (ANOVA) and significance between groups was taken at $p < 0.05$.

RESULTS

The concentration of heavy metals in the oil samples is presented in Table 1 The lipoprotein concentrations in serum of rabbits exposed for 30, 60 and 90 days to Sample A, B and C are presented in Table 2, 4 and 6.

Table 3,5 and 7 shows the results of cardioprotective and atherogenic risk ratios during 30, 60 and 90 days exposure.

Table 1: Concentration of Heavy Metals in the Oil Samples

S/N	OIL SAMPLES	Cadmiun (mg/kg)	Lead (mg/kg)	Arsenic (mg/kg)	Copper (mg/kg)	Zinc (mg/kg)
1	A	1.580	11.38	30.088	1.080	5.760
2	B	2.260	18.060	45.660	1.700	5.910
3	C	2.370	13.390	21.090	1.190	5.110
USEPA LIMIT		0.5	0.5	2.5	0.5	18
WHO LIMIT		0.3	10	10	1	20

Key: Sample A= Allthingsnatural by Emi Herbal Oil, Sample B = Kakiva Herbal Oil, Sample C= Amal Botanical Herbal Oil

Table 2: Mean ± SD of Lipoproteins during 30 days Exposure

Samples	TC (mmol?L)	HDL-C (mmol/L)	LDL-C (mmol/L)	TG (mmol/L)	VLDL-C (mmol/L)
Control	2.45±0.10	0.26±0.02	1.65±0.15	1.18±0.13	0.54±0.06
A	2.49±0.13	0.27±0.01	1.66±0.16	1.25±0.17	0.57±0.08
B	2.65±0.14	0.27±0.02	1.79±0.13	1.33±0.13	0.60±0.06
C	2.56±0.06	0.27±0.02	1.73±0.14	1.23±0.17	0.58±0.08
F-value	2.381	0.12	0.7629	0.6881	0.6384
P value	0.1206	0.9466	0.5363	0.5765	0.6046

Keys: TC= total cholesterol, HDL-C= high density lipoprotein cholesterol, LDL-C= low density lipoprotein cholesterol, VLDL-C= very low-density lipoprotein cholesterol, TG= triglycerides.

Table 3: Mean ± SD of Cardioprotective and Atherogenic Risk Ratios during 30 days Exposure

Samples	CRR-1	CRR-2	AIP	AAI	AC	TG/HDL
Control	9.36±0.73	6.31±0.77	0.65±0.05	11.50±1.29	8.35±0.73	4.49±0.52
A	9.31±0.32	6.19±0.58	0.77±0.14	11.25±0.96	8.31±0.32	4.88±1.10
B	9.95±1.10	6.78±0.91	0.69±0.06	11.25±0.96	8.95±1.10	4.98±0.65
C	9.58±0.77	6.50±0.83	0.66±0.06	11.25±0.96	8.58±0.77	4.59±0.63
F-value	0.5708	0.4283	1.462	0.0566	0.5751	0.3740
P value	0.6449	0.7364	0.2741	0.9815	0.6423	0.7728

Keys: CRR-1=cardiac risk ratio 1, CRR-11= cardiac risk ratio 11, AIP= atherogenic index of plasma, AC=atherogenic coefficient, AAI- anti-atherogenic index

Table 4: Mean ± SD of Lipoproteins during 60 days Exposure

Samples	TC (mmol/L)	HDL-C (mmol/L)	LDL-C (mmol/L)	TG (mmol/L)	VLDL-C (mmol/L)
Control	2.43 ± 0.06^{a}	0.28 ± 0.01^{a}	1.62±0.03	1.18 ± 0.13^{a}	0.54 ± 0.06^{a}
A	2.56 ± 0.08^{ab}	0.25 ± 0.01^{b}	1.68±0.06	1.38 ± 0.10^{ab}	0.63 ± 0.04^{ab}
B	2.73 ± 0.07^{b}	0.24 ± 0.01^{b}	1.78±0.05	1.58 ± 0.10^{b}	0.72 ± 0.03^{b}
C	2.60 ± 0.06^{b}	0.25 ± 0.01^{b}	1.67±0.12	1.48 ± 0.17^{b}	0.67 ± 0.08^{b}
F-value	14.6	9.714	3.188	7.368	7.177
P value	0.0003	0.0016	0.0629	0.0046	0.0051

Keys: TC= total cholesterol, HDL-C= high density lipoprotein cholesterol, LDL-C= low density lipoprotein cholesterol, VLDL-C= very low-density lipoprotein cholesterol, TG= triglycerides. Mean ± SD with different superscripts is significantly different from each other at $p<0.05$.

Table 5: Mean ± SD of Cardioprotective and Atherogenic Risk Ratios during 60 days Exposure

Samples	CRR-1	CRR-11	AIP	AAI	AC	TG/HDL
Control	8.85±0.56^{a}	5.89±0.27^{a}	0.63±0.07^{a}	12.00±0.82^{a}	7.84±0.56^{a}	4.15±0.27^{a}
A	10.15±0.58^{b}	6.67±0.38^{b}	0.74±0.04^{b}	10.50±0.58^{b}	9.15±0.58^{b}	5.46±0.53^{b}
B	11.39±0.29^{c}	7.41±0.19^{c}	0.81±0.03^{b}	9.00±0.00^{bc}	10.39±0.29^{c}	6.11±0.76^{b}
C	10.28±0.04^{b}	6.62±0.37^{b}	0.77±0.05^{b}	10.00±0.00^{b}	9.28±0.04^{b}	5.85±0.76^{b}
F-value	23.42	15.94	8.744	25	23.68	8.103
P value	<0.0001	0.0002	0.0024	<0.0001	<0.0001	0.0032

Keys: CRR-1=cardiac risk ratio 1, CRR-11= cardiac risk ratio 11, AIP= atherogenic index of plasma, AC= atherogenic coefficient, AAI- anti-atherogenic index. Mean ± SD with different superscripts is significantly different from each other at p<0.05.

Table 6: Mean ± SD of Lipoproteins during 90 days Exposure

Samples	TC (mmol/L)	HDL-C (mmol/L)	LDL-C (mmol/L)	TG (mmol/L)	VLDL-C (mmol/L)
Control	2.46±0.08^{a}	0.27±0.01^{a}	1.65±0.10	1.20±0.14^{a}	0.55±0.67^{a}
A	2.67±0.09^{b}	0.25±0.01^{bc}	1.81±0.12	1.35±0.21^{b}	0.62±0.10^{b}
B	2.85±0.07^{b}	0.22±0.01^{b}	1.79±0.07	1.88±0.05^{c}	0.85±0.02^{b}
C	2.73±0.10^{b}	0.25±0.01^{c}	1.74±0.16	1.65±0.13^{bc}	0.75±0.06^{b}
F-value	13.55	21.92	1.416	17.73	16.99
P value	0.0004	<0.0001	0.2863	0.0001	0.0001

Keys: TC= total cholesterol, HDL-C= high density lipoprotein cholesterol, LDL-C= low density lipoprotein cholesterol, VLDL-C= very low-density lipoprotein cholesterol, TG= triglycerides. Mean ± SD with different superscripts is significantly different from each other at p<0.05.

Table 7: Mean ± SD of Cardioprotective and Atherogenic Risk Ratios during 90 days Exposure

Samples	CRR-1	CRR-2	AIP	AAI	AC	TG/HDL
Control	9.23±0.64^{a}	6.18±0.56^{a}	0.65±0.06^{a}	11.50±1.00^{a}	7.84±0.56^{a}	4.50±0.62^{a}
A	10.79±0.19^{b}	7.30±0.40^{b}	0.75±0.09^{a}	9.75±0.50^{b}	9.15±0.50^{b}	5.45±0.82^{ac}
B	13.12±0.54^{c}	8.21±0.36^{b}	0.94±0.04^{bc}	7.75±0.50^{c}	10.39±0.2^{c}	8.63±0.57^{b}
C	11.16±0.55^{b}	7.10±0.73^{b}	0.83±0.02^{ac}	9.75±0.50^{b}	9.28±0.04^{b}	6.73±0.48^{c}
F-value	39.75	9.747	17.58	21.48	23.68	31.7
P value	<0.0001	0.0015	0.0001	<0.0001	<0.0001	<0.0001

Keys: CRR-1=cardiac risk ratio 1, CRR-11= cardiac risk ratio 11, AIP= atherogenic index of plasma, AC= atherogenic coefficient, AAI- anti-atherogenic index. Mean ± SD of experimental groups with different superscripts are significantly different from each other at p<0.05.

DISCUSSION

Cosmetics are important but often overlooked source of heavy metal exposure. The results of heavy metals analysis of the all-threes oil samples are above the WHO acceptable limits of cadmium, lead, arsenic, and copper while zinc is within the acceptable limit [10], [11]. This result agrees with [12] who stated that some cosmetics used in Nigeria seem to be laced with heavy metals beyond the acceptable limit established by Who Health Organization.

The results of lipid profile and atherogenic coefficients in 30 and 60 days was not significant in sample A, B and C, compared to control. However, in 90 days the lipid profile and atherogenic coefficient results was significantly ($p<0.005$) higher in sample A, B and C, compared to control. This result is in tandem with the work of [9], who reported that chronic exposure to heavy metals in cosmetics induces oxidative stress and hepatotoxicity leading to a pro-atherogenic lipid profile characterized by elevated total cholesterol, triglyceride low density lipoprotein and reduced high density lipoprotein.

Several studies have reported significant increase in total cholesterol, triglyceride and low-density lipoprotein with concomitant decrease in high density lipoprotein following chronic exposure. These alterations culminate in elevated atherogenic indices, suggesting a plausible link between heavy metal burden and increased cardiovascular risk.

The present study revealed a significant association between the use of cosmetics containing heavy metals and alteration in lipid profile and atherogenic coefficient. This finding suggests that chronic exposure to heavy metals through cosmetics products might be a predisposing factor to the risk of cardiovascular disease. The observed pattern is consistent with previous report by [6], who linked heavy metal exposure to oxidative stress and hepatic dysfunction.

The dyslipidemic pattern associated with heavy metal exposure, elevated total cholesterol, triglyceride, low density lipoprotein and reduced high density lipoprotein, translates to increased atherogenic risk. This is reflected in elevated cardiovascular risk indices such as the Atherogenic Index of Plasma (AIP), Cardiac Risk Ratio (CRI-1), and Atherogenic Coefficient (AC) [8].

CONCLUSION

The result of this study suggests that heavy metals exert adverse effects on lipid profile through oxidative stress and hepatotoxicity. These mechanisms collectively promote a pro-atherogenic lipid profile and increase cardiovascular risk indices.

CONSENT

It is not applicable

ETHICAL APPROVAL

The Animal Welfare Act of 1985 of the United State of America for research and Institutional Animal Care and Use Committee (IACUC) protocol were strictly adhered to. All experiments have been examined approved by the appropriate ethic committee.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declares that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of this manuscript.

COMPETING INTERESTS

Authors have declared that no competing interest exist.

REFERENCES

1. Kapoor S. & Saraf, S. (2007). Risk Analysis Tools for Toxicological Profile of Cosmetics. *The Internet Journal of Toxicology,* 5-2.

2. Aburjai, T. & Natsheh F. M. (2003). Plants Used in Cosmetics. *Phytotherapy Research,* 7(9), 987-1000.

3. Campos-Da-Paz, M., Pereira, L. O., Bicalho, L .S., Dorea, J. G., Pocas-Fonseca, M. J. & Santos, M. F. (2008), Interaction of Bracken-Fern Extract with Vitamin C in Human Submandibular Gland and Oral Epithelium Cell Lines. *Mutation Research*, 652, 158–63.

4. Jaishankar, M., Tseten, T., Anbalagan, N., Mathew, B. B., & Beeregowda, K. N. (2014). Toxicity, mechanism and health effects of some heavy metals. *Interdisciplinary Toxicology*, 7(2), 60-72.

5. Ercal, N., Gurer-Orhan, H., & Aykin-Burns, N. (2001). Toxic metals and oxidative stress part I: mechanisms involved in metal-induced oxidative damage. *Current Topics in Medicinal Chemistry*, 1(6), 529-539.

6. Nwokocha, C. R., Ude, V. C., & Nwokocha, M. I. (2012). Possible antioxidant effect of _Moringa oleifera_ leaves on cadmium-induced hepatotoxicity and dyslipidemia. *Journal of Medicinal Food*, 15(9), 791-795.

7. Satarug, S., & Moore, M. R. (2004). Adverse health effects of chronic exposure to low-level cadmium in foodstuffs and cigarette smoke. *Environmental Health Perspectives*, 112(10), 1099-1103.

8. Nwagha, U. I., Ikekpeazu, E. J., Ejezie, F. E., Nebo, E. O., & Ibegbulam, O. G. (2010). Atherogenic index of plasma as useful predictor of cardiovascular risk among type 2 diabetic patients in Enugu, Nigeria. *African Health Sciences*, 10(3), 248-252.

9. Aolayan,T. O., Ogunyemi, O. O., & Adebayo, O. A. (2020). Assessment of heavy metals in some facial cosmetics sold in Nigeria. *Journal of Applied Sciences and Environmental Management*, 24(3), 455-460.

10. World Health Organization (2015). Guidelines on the safety assessment of cosmetic products. WHO Publication.

11. US EPA. (1996). Health Effect Test Guidelines: Dermal Penetration. US Environmental Protection Agency (EPA), Washington, DC: US EPA Publishing.

12. Orisakwe O. E. & Otaraku J. O. (2013). Metal concentrations in cosmetics commonly used in Nigeria. The Scientific World Journal, (1), 1-7.