Oyana *et. al.,* Babcock Univ. Med. J.2025 8(2):435-443
https://doi.org/10.38029/babcockuniv.med.j..v8i2.1024
eISSN: 2756-4657

RESEARCH ARTICLE OPEN ACCESS

Exposure to surgical plumes induces histo-hepato-renal and ovarian disruptions, and the protective role of an antioxidant cocktail in experimental rats

Oyana EN[1,2]ID, Akpor OO[3]ID, Olaniyi KS[4]ID

[1]*Department of Nursing Sciences, College of Medicine and Health Sciences, Afe Babalola University, Ado-Ekiti, Ekiti State, Nigeria*
[2]*Faculty of Basic Medical Sciences, Department of Nursing Science, University of Benin, Benin City, Edo State, Nigeria*
[3]*Department of Nursing, Saint John Regional Hospital, Saint John, New Brunswick, Canada*
[4]*College of Medical Sciences, Department of Human Physiology, Afe Babalola University, Ado Ekiti, Ekiti State, Nigeria*

Submitted: 4th July 2025
Accepted: 22nd August 2025
Published: 31st December 2025
ID: Orcid ID

Abstract
Objective: Among the leading causes of liver/kidney disease and ovarian failure in reproductive-aged women, inflammation and oxidative stress have been reported to aggravate organ complications induced by several factors, including surgical plumes. Vitamins A, C and E are well documented to improve the antioxidant defence system. This study hypothesised the toxic exposure of ovaries to surgical plume and the therapeutic effect of antioxidants.
Method: Adult female Wistar rats were randomly assigned into four groups: control (CONT), surgical plume (SUPM), antioxidant (ANTO), and SUPM + ANTO groups. Surgical plume was simulated in an enclosed chamber, while administration of the antioxidant was for 28 days (20 mg/kg, *p.o.*). Thereafter, the animals were sacrificed, and the liver, kidney and ovaries were collected for histological examination.
Result: Histological analysis revealed the SUPM group exhibited severe expression of Kupffer cells (hepatic inflammation) as well as macrophage infiltration in the Bowman's space (renal inflammation. Similarly, ovarian tissue showed degenerated follicles when compared with control animals. Nevertheless, administration of an antioxidant cocktail decreased the Kupffer cells and macrophages in the liver and kidney, respectively, and reversed degenerated ovarian follicles when compared with the untreated SUPM group.
Conclusion: Surgical plume exposure compromises hepatic/renal and ovarian tissue to cellular inflammation and oxidative stress, which contributes to cellular apoptosis. The present study revealed that antioxidant administration elicited protective effects in mitigating plume-induced hepatorenal and ovarian toxicity. The findings further highlight the urgent need for plume evacuation systems, enhanced ventilation, and protective measures to safeguard reproductive health among healthcare workers.

Keywords: Antioxidants, Inflammation, Organotoxicity, Surgical plumes, ROS

Correspondence:
Oyana Elizabeth N
Department of Nursing Sciences, College of Medicine and Health Sciences,
Afe Babalola University, Ado-Ekiti
Nigeria
+234(0)8066643513, nwakaegooyana@gmail.com, elizabeth.oyana@uniben.edu

Plain English Summary

Surgical plumes are smoke substances that are emitted from surgical machines when performing surgical procedures. The surgical plumes pose a health threat to theatre doctors and nurses, and prolonged exposure can cause organ failure in these individuals, particularly female practitioners of childbearing age. The result from this study showed that the structure of the liver, kidney and ovaries of subjects in the surgical plume group was distorted, which could suggest an impairment in the normal function of these vital organs. However, when the antioxidant cocktail was administered, the morphological changes in these organs were averted.

Background

Over the decades, environmental factors/substances have been reported as a contributing factor to several health complications. Among these substances are surgical plumes (surgical smoke/vapour), which are often overlooked as an occupational hazard in surgical settings, emitted when biological tissue is disrupted by energy-based devices (1, 2). The use of electrosurgical instruments ruptures cellular membranes, releasing vaporised intracellular contents, including visible and invisible particulates, gases, and smoke (2, 3). Surgical plume is primarily composed of water in the form of steam, but approximately five per cent consists of cellular debris, which may contain carcinogens, toxins, blood, bacteria, viruses, and tissue particles (3). Aerosolisation of these plumes spread throughout the operating theatre, exposing both staff and patients to potentially harmful substances when they are inhaled into the body. This increases the metabolic demand on the liver and kidneys to eliminate the toxins from the body (4). However, these organs can be overwhelmed due to excessive exposure to toxins, and this can trigger cellular inflammation/oxidative stress, leading to complications such as hepatic carcinoma, liver cirrhosis, and chronic kidney diseases, among others (5, 6).

Moreover, studies have also shown that exposure to toxins and endocrine-disrupting chemicals (EDCs) from environmental contaminants, including surgical smoke could alter the female reproductive organ (ovaries) by contributing to impaired follicular development and estrogen/progesterone secretion needed to maintain/sustain female reproductive health, thus resulting in primary ovarian insufficiency and infertility, among others (7, 8, 9). Additionally, it has been documented that prolonged exposure to these toxins increases the risk of ovarian cancer in individuals who reside in regions where environmental pollution is common (10, 11), particularly individuals who are exposed to frequent inhalation of surgical smoke from surgical procedures. Similarly, the presence of toxins in circulation triggers the release of inflammatory cytokines to protect the cells from the invasion of foreign/harmful substances (12). However, during metabolic stress, the compensatory mechanism can be lost, leading to prolonged systemic inflammation (13). Likewise, chronic inflammatory response has been shown to alter cellular integrity, thus predisposing cells to neoplastic reformation (14, 15). In addition, chronic inflammation and oxidative stress may induce DNA mutation via the generation of excessive free radicals, which is a *sine qua non* for carcinogenesis (10, 16). Nevertheless, there is a paucity of studies showing the effect of surgical plume on the reproductive health of female workers who are exposed to frequent surgical procedures.

Moreover, several studies have shown that therapeutic agents, particularly agents with abundant antioxidants elicit their beneficial effects via modulation of inflammatory pathways and suppression of excessive free radicals in circulation which may arise as a result of metabolic stress induced by environmental factors/toxicants such as smokes from industries, water pollution, soil pollution, among others (17, 18, 19). Antioxidant-rich supplements have been reported to play a key role in improving immune responses and suppressing the excess free radicals in circulation by maintaining redox balance (20), which in turn protects cellular integrity from inflammation, oxidative stress and ultimately, apoptosis. Antioxidant-rich substances like citrus, green vegetables and high-fibre foods have been shown to improve gut health, which regulates the overall metabolic health of an individual (21, 22). Nevertheless, studies have reported that depleted levels of antioxidants contributed to systemic/tissue inflammation, oxidative stress and well as cellular apoptosis (21, 23, 24). Therefore, this study was designed to evaluate the hepatorenal/ovarian architecture aimed at investigating ovarian response to uncontrolled exposure to surgical plumes and the therapeutic effect of antioxidant administration in an experimental rat model.

Materials and methods

Experimental animals, grouping of animals and protocol

Adult female Wistar rats were purchased and housed in cages at the College of Medical Sciences, University of Benin, animal house. Rats were acclimatised for two weeks and had limitless access to standard rat chow and tap water. Thereafter, rats were randomised into four groups with a sample size, n=6. The groups include control (CONT), surgical plume only (SUPM), antioxidant only (ANTO), and SUPM + ANTO groups. Rats were maintained under standard environmental conditions, with normal room temperature, relative humidity of about 50-60% as well as a dark/light cycle of 12 hours respectively. This research was performed in adherence to guidelines from the National Institutes of Health Guide for the Care and maintenance of Laboratory Animals, and the protocol was approved by the Ethical Review Board of College of Medical Sciences, University of Benin animal house, Benin City, Edo State, Nigeria, with the protocol number CMS/REC/2024/787.

Exposure to surgical plumes

The exposure chamber, made of stainless steel and glass, measures 31 cm x 24 cm x 21 cm is prepared, and experimental rats are placed inside. An animal is used as the plume source and burned with a probe inside the chamber, generating a plume in the presence of the experimental rats. The rats are exposed to the plume for 30 minutes daily for a duration of 28 days (25, 26, 27). The chamber temperature and humidity are maintained within a specified range to ensure a controlled exposure environment.

Treatment

The animals in the control group received olive oil, the SUPM group received distilled water, while rats in ANTO and SUPM+ANTO groups received a cocktail of antioxidants containing vitamins A, C, E and Selenium (20 mg/kg, *p.o.*) for 28 days as previously described by (27, 28). The antioxidant cocktail was purchased in powder form from Molychem India LLP (Code: 17785) and was dissolved in Olive oil (Goya Extra virgin Olive oil; Batch no: 0040, 8993379261771) as previously described by (27).

Sacrifice of animals

At the end of the 4 weeks of administration, the experimental animals were sacrificed. Chloroform was used to anaesthetise the animals. Organs were isolated for morphological evaluation.

Histological preparation

Hepatic tissue, renal tissue and ovarian tissue were isolated from rats and examined using H & E stains. For hematoxylin and eosin (H & E) stains, a section of the liver, kidney and ovary was fixed in 10% formalin saline overnight. The tissues were thereafter dehydrated, embedded in paraffin, and sectioned at 5-μm thickness. The various slides for each tissue were prepared and examined using an OPTO-Edu industrial camera light microscope and a computer (Nikon, Japan) as previously described by (29, 30, 31, 32).

Result

Antioxidant cocktail reversed hepato-morphological disruption in experimentally SUPM rats

There was a disruption in the liver morphology in the SUPM group when compared to the CONT group. However, this was reversed in the SUPM+ANTO group compared to the SUPM group (Figure 3.1).

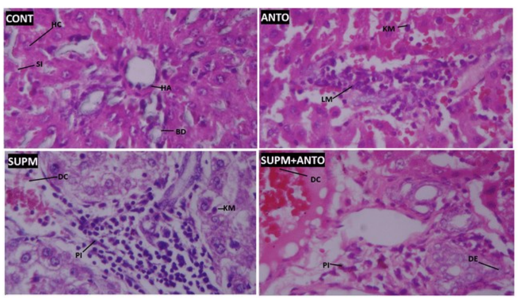

Figure 3.1: Photomicrograph of the sections of the Liver in the control and rats exposed to ANTO, SUMP, and ANTO+SUMP (H&E, X40).

Sections of hepatic tissue from control show normal architecture: HC, SI, HA and BD Sections of hepatic tissue from rats exposed to SUPM only show: DC, PI and KM. Sections from rats exposed to antioxidants only showed: active sinusoidal congestion, LM and KM, while sections from rats exposed to SUPM + ANTO showed: DC, PI and DE

Antioxidant cocktail improved renal morphology in the experiment SUPM-induced animal model
Renal morphology was distorted in the SUPM group when compared to the CONT group. However, this was reversed in the SUPM+ANTO group compared to SUPM only (Figure 3.2).

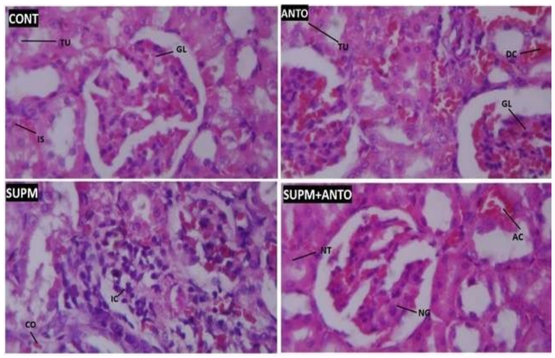

Figure 3.2: Photomicrograph of the sections of the Kidney in the control and rats exposed to ANTO, SUMP, and ANTO+SUMP (H&E, X40)

Sections of kidney from control animals showed: normal architecture: TU, IS and GL, while rats exposed to plumes only showed: CO and IC. Sections of kidney from rats exposed to antioxidants only showed: TU and GL, DC, and sections of kidney from rats exposed to plumes + antioxidants showed: NT and NG, AC

Antioxidant cocktail improved ovarian morphology in the experiment SUPM-induced animal model
Ovarian morphology was distorted in the SUPM group when compared to the CONT group. However, this was reversed in the SUPM+ANTO group compared to SUPM only (Figure 3.3).

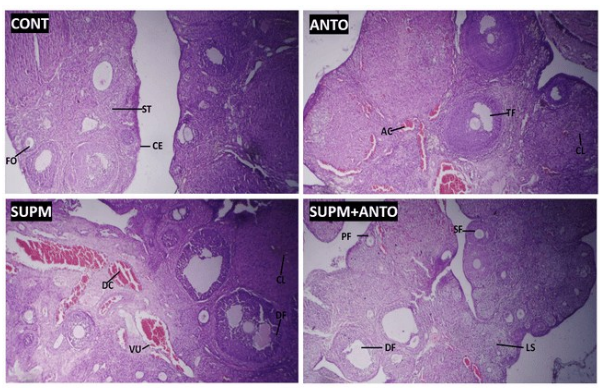

Figure 3.3: Photomicrograph of the sections of the Ovarian in the control and rats exposed to ANTO, SUMP, and ANTO+SUMP (H&E, X40).

Sections from control rats showed normal architecture, including CE, ST, and FO, whereas animals exposed to surgical plumes only showed severe DC, DF, VU, and CL. Ovarian sections from rats exposed to anti-oxidant only showed: AC, TF and CL, while the ovarian section of animals in the SUPM + ANTO group showed: LS, DF, PF and SF

Discussion

The effect of surgical plume (a byproduct of electrosurgical procedures) on the liver, kidneys and ovaries is understudied. Liver and kidney complications are among the leading causes of morbidity and mortality globally, particularly among women of childbearing age who reside in regions susceptible to environmental hazards (5, 33, 34). Similarly, these women are not only prone to hepatic/renal complications but infertility cases (10). Previous studies have shown that environmental toxins negatively affect metabolic health via suppression of the antioxidant system and upregulation of inflammatory mediators, as well as oxidative stress, which in turn disrupts cellular function and structure (13, 19). Results from the present study showed that rats in the SUPM group were characterised by distorted hepatic/renal morphology, as well as altered ovarian follicles when compared with the control group. Nevertheless, administration of an antioxidant cocktail attenuated these morphologic alterations in treated animals compared to the untreated surgical plume group.

The present finding showed an aberration in the hepatic and renal morphology of animals in the SUPM group when compared to the control animals. The liver is an important metabolic organ that plays several important roles, including glycogen storage, metabolism of proteins, fats and carbohydrates, detoxification of waste products, regulation of cholesterol level, and immune regulation, among others, under physiological conditions (34). However, the invasion of toxins into circulation triggers the release of abundant inflammatory cytokines into circulation (systemic inflammation). In response to the liver to aggravated circulating levels of inflammatory cytokines, glycogen is depleted to release ATP in order to cope during metabolic stress, which consequently triggers the influx of inflammatory cytokines into the liver as observed by the presence of abundant leucocytes in the hepatic tissue (35). This implies that under metabolic stress, the liver is susceptible to complications, including liver cirrhosis, liver carcinoma, among others, which in turn could release more reactive oxygen species (ROS) into circulation, thereby aggravating oxidative stress as well as cellular apoptosis (6, 34). Similarly, the renal tissue is susceptible to metabolic assault as a result of chronic inflammatory responses, which possibly disrupts the podocytes and makes it permeable to substances, including proteins and fats, leading to proteinuria and lipiduria, respectively, which are symptoms of kidney failure (36, 37). Nevertheless, when the antioxidant cocktail was administered, there was a significant improvement in the hepatic/renal morphology as observed by decreased expression of Kupffer cells in the liver and an improved Bowman's capsule/space, respectively, in the SUPM+ANTO group when compared to the untreated SUPM group. This suggests that the antioxidant cocktail suppresses inflammation and oxidative stress via upregulation of the antioxidant system, which possibly attenuates liver/renal complications associated with surgical plumes.

Additionally, the present study showed that SUPM animals were characterised by severe stromal vasodilatation, devitalized follicles, and vascular ulceration. These pathological changes suggest that surgical plume exposure induces oxidative stress, vascular injury, and inflammation, leading to follicular atresia and ovarian dysfunction. Previous studies have documented the toxic effects of environmental pollutants, including endocrine-disrupting chemicals (EDCs) and occupational exposures, on ovarian health. Additionally, chronic exposure to industrial air pollutants leads to follicular degeneration, stromal fibrosis, and impaired steroidogenesis, findings consistent with the observed ovarian damage in this study (38, 39). The implications of such pathological changes extend beyond reproductive dysfunction, as long-term oxidative stress in the ovary has been linked to premature ovarian failure, infertility, and an increased risk of ovarian cancer (40). Interestingly, animals in the SUPM+ANTO group showed improved ovarian morphology, as observed by improved primary and secondary follicles, suggesting some degree of follicular preservation. The presence of degenerating follicles indicates that antioxidant therapy alone was not fully protective against the toxic effects of surgical plume exposure. This finding is consistent with a previous study described by Porter et al. (41), who reported that antioxidants help reduce oxidative stress in the ovary, but are not sufficient in preventing all forms of ovarian damage induced by airborne toxins. The luteinisation of the stroma may reflect a compensatory response to oxidative stress, where altered steroidogenesis attempts to counteract follicular damage. This aligns with earlier findings suggesting that chronic exposure to oxidative stressors alters normal hormonal signalling, leading to disrupted ovarian function and suboptimal reproductive outcomes (17, 39).

Given these findings, therapeutic interventions should extend beyond antioxidant supplementation. While antioxidants provide a degree of ovarian protection, the persistence of follicular degeneration despite treatment suggests that additional strategies, such as plume evacuation systems, improved surgical ventilation, and the use of high-efficiency filtration masks, should be prioritised (41, 42). Furthermore, novel anti-inflammatory and cytoprotective agents may be explored to enhance hepatorenal/ovarian integrity against toxic insults. Future research should focus on dose optimisation, combination therapies, and long-term reproductive outcomes in individuals chronically exposed to surgical smoke.

Conclusion

Surgical plume exposure compromised hepatic/renal and ovarian tissue to cellular inflammation and oxidative stress, which contributed to cellular apoptosis. The present study revealed that antioxidant administration elicited protective effects in mitigating plume-induced hepatorenal and ovarian toxicity. The findings further highlight the urgent need for plume evacuation systems, enhanced ventilation, and protective measures to safeguard reproductive health among healthcare workers.

Limitations of the study

The mechanism of action was not investigated in this study. However, other studies have reported that plume-induced pathology could be due to oxidative stress and inflammation.

List of Abbreviations

AC: Active interstitial congestion
ANTO: Antioxidant
BD: bile ducts
CE: coelonic epithelium
CL: corpus luteum
CO: interstitial congestion
CONT: Control
DC: vasodilatation and congestion
DE: ductal epitheliosis
DF: degenerating mature follicles/devitalized follicles
DNA: Deoxyribonucleic acid
EDCs: Endocrine-disrupting chemicals
FO: follicles in different stages of maturation
GL: glomeruli
H & E: hematoxylin and eosin
HA: hepatic artery
HC: hepatocytes
IC: interstitial infiltrates of inflammatory cells
IS: the interstitial space
KM: Kupffer cell mobilisation
LM: lymphocytes
LS: luteinized stroma
NG: normal glomeruli
NT: normal tubules
p.o: per os
PF: primary follicles
PI: heavy periportal infiltrates of inflammatory cells
SF: secondary follicles
SI: sinusoids
ST: stroma
SUPM + ANTO: Surgical plume + Antioxidant
SUPM: Surgical plume
TF: tertiary follicles
TU: tubules
VU: vascular ulceration

Declaration

Ethics approval and consent to participate

The research was approved by the Research Ethics Committee of the College of Medical Sciences, University of Benin, with the ethical number CMS/REC/01/VOL.2/787. The research ethics committee's guidelines for animal handling and treatment at the University of Benin's College of Medical Sciences were fully implemented. Consent to participate is not applicable.

Consent for publication

Not applicable.

Availability of data and materials

The data supporting the present study will be made available on request from the corresponding author.

Competing interests

The authors have no conflict of interest to declare.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authors' contributions

ONE and OAA conceived, designed and conducted the experiment. ONE, OAA, and OKS contributed reagents. ONE, OAA, and OKS reviewed, revised and approved the final manuscript for submission.

Acknowledgement

Not applicable.

Supplementary files

Not applicable

References

1. Zhang F, Liu W, Zhou S, Jiang L, Wang K, Wei Y, Liu A, Wei W, Liu S. Investigation of environmental pollutant-induced lung inflammation and injury in a 3D coculture-based microfluidic pulmonary alveolus system. Analytical Chemistry. 2020 Apr 1;92(10):7200-8. https://doi.org/10.1021/acs.analchem.0c00759
2. Wang H, Xu T, Yin D. Emerging trends in the methodology of environmental toxicology: 3D cell culture and its applications. Science of The Total Environment. 2023 Jan 20;857:159501. https://doi.org/10.1016/j.scitotenv.2022.159501
3. Hamlin L. Perioperative nursing: An introduction. (No Title). 2016.
4. Wang H, Yin F, Li Z, Su W, Li D. Advances of microfluidic lung chips for assessing atmospheric pollutants exposure. Environment

International. 2023 Feb 1;172:107801. https://doi.org/10.1016/j.envint.2023.107801
5. Guo B, Guo Y, Nima Q, Feng Y, Wang Z, Lu R, Ma Y, Zhou J, Xu H, Chen L, Chen G. Exposure to air pollution is associated with an increased risk of metabolic dysfunction-associated fatty liver disease. Journal of hepatology. 2022 Mar 1;76(3):518-25. https://doi.org/10.1016/j.jhep.2021.10.016
6. Barouki R, Samson M, Blanc EB, Colombo M, Zucman-Rossi J, Lazaridis KN, Miller GW, Coumoul X. The exposome and liver disease-how environmental factors affect liver health. Journal of hepatology. 2023 Aug 1;79(2):492-505. https://doi.org/10.1016/j.jhep.2023.02.034
7. Schultz L. An analysis of surgical smoke plume components, capture, and evacuation. AORN journal. 2014 Feb 1;99(2):289-98. https://doi.org/10.1016/j.aorn.2013.07.020
8. Vabre P, Gatimel N, Moreau J, Gayrard V, Picard-Hagen N, Parinaud J, Leandri RD. Environmental pollutants, a possible etiology for premature ovarian insufficiency: a narrative review of animal and human data. Environmental Health. 2017 Apr 7;16(1):37. https://doi.org/10.1186/s12940-017-0242-4
9. Cui J, Wang Y. Premature ovarian insufficiency: a review on the role of tobacco smoke, its clinical harm, and treatment. Journal of Ovarian Research. 2024 Jan 9;17(1):8. https://doi.org/10.1186/s13048-023-01330-y
10. Dehghani S, Moshfeghinia R, Ramezani M, Vali M, Oskoei V, Amiri-Ardekani E, Hopke P. Exposure to air pollution and risk of ovarian cancer: a review. Reviews on Environmental Health. 2023 Sep 1;38(3):439-50. https://doi.org/10.1515/reveh-2021-0129
11. Evangelinakis N, Geladari EV, Geladari CV, Kontogeorgi A, Papaioannou GK, Peppa M, Kalantaridou S. The influence of environmental factors on premature ovarian insufficiency and ovarian aging. Maturitas. 2024 Jan 1;179:107871. https://doi.org/10.1016/j.maturitas.2023.107871
12. Yang Y, Huang W, Yuan L. Effects of environment and lifestyle factors on premature ovarian failure. InEnvironment and Female Reproductive Health 2021 Feb 2 (pp. 63-111). Singapore: Springer Singapore. https://doi.org/10.1007/978-981-33-4187-6_4
13. Mishra B, Tiwari A, Mishra S. Metabolic Changes and Immunity Suppression Parameters as Biomarkers of Environmental Pollutants. InBiomonitoring of Pollutants in the Global South 2024 Jun 6 (pp. 693-719). Singapore: Springer Nature Singapore. https://doi.org/10.1007/978-981-97-1658-6_20
14. Anuja K, Roy S, Ghosh C, Gupta P, Bhattacharjee S, Banerjee B. Prolonged inflammatory microenvironment is crucial for pro-neoplastic growth and genome instability: a detailed review. Inflammation Research. 2017 Feb;66(2):119-28. https://doi.org/10.1007/s00011-016-0985-3
15. Danforth DN. The role of chronic inflammation in the development of breast cancer. Cancers. 2021 Aug 3;13(15):3918. https://doi.org/10.3390/cancers13153918
16. Dabravolski SA, Orekhova VA, Baig MS, Bezsonov EE, Starodubova AV, Popkova TV, Orekhov AN. The role of mitochondrial mutations and chronic inflammation in diabetes. International journal of molecular sciences. 2021 Jun 23;22(13):6733. https://doi.org/10.3390/ijms22136733
17. Jiang D, Chen S, Sun R, Zhang X, Wang D. The NLRP3 inflammasome: Role in metabolic disorders and regulation by metabolic pathways. Cancer letters. 2018 Apr 10;419:8-19. https://doi.org/10.1016/j.canlet.2018.01.034
18. Farzaei MH, Singh AK, Kumar R, Croley CR, Pandey AK, Coy-Barrera E, Kumar Patra J, Das G, Kerry RG, Annunziata G, Tenore GC. Targeting inflammation by flavonoids: novel therapeutic strategy for metabolic disorders. International Journal of Molecular Sciences. 2019 Jan;20(19):4957. https://doi.org/10.3390/ijms20194957
19. Charles-Messance H, Mitchelson KA, Castro ED, Sheedy FJ, Roche HM. Regulating metabolic inflammation by nutritional modulation. Journal of Allergy and Clinical Immunology. 2020 Oct 1;146(4):706-20. https://doi.org/10.1016/j.jaci.2020.08.013
20. Ramos-Lopez O, Milagro FI, Riezu-Boj JI, Martinez JA. Epigenetic signatures underlying inflammation: An interplay of nutrition, physical activity, metabolic diseases, and environmental factors for personalized nutrition. Inflammation Research. 2021 Jan;70(1):29-49. https://doi.org/10.1007/s00011-020-01425-y
21. Martemucci G, Costagliola C, Mariano M, D'andrea L, Napolitano P, D'Alessandro AG. Free radical properties, source and targets, antioxidant consumption and health. Oxygen. 2022 Apr 12;2(2):48-78. https://doi.org/10.3390/oxygen2020006
22. Talaie A, Kamyab H, Mediavilla DZ. Environmental Factors Affecting the Gut Microbiome. Journal of Environmental Treatment Techniques. 2023;11(3):94-108.

23. Pisoschi AM, Pop A, Iordache F, Stanca L, Predoi G, Serban AI. Oxidative stress mitigation by antioxidants-an overview on their chemistry and influences on health status. European Journal of Medicinal Chemistry. 2021 Jan 1;209:112891. https://doi.org/10.1016/j.ejmech.2020.112891
24. Kıran TR, Otlu O, Karabulut AB. Oxidative stress and antioxidants in health and disease. Journal of Laboratory Medicine. 2023 Feb 23;47(1):1-1. https://doi.org/10.1515/labmed-2022-0108
25. Wong BA. Inhalation exposure systems: design, methods and operation. Toxicologic pathology. 2007 Jan;35(1):3-14. https://doi.org/10.1080/01926230601060017
26. Movia D, Bruni-Favier S, Prina-Mello A. In vitro alternatives to acute inhalation toxicity studies in animal models—A perspective. Frontiers in bioengineering and biotechnology. 2020 Jun 3;8:549. https://doi.org/10.3389/fbioe.2020.00549
27. Oyana NE, Akpor OA, Eze IG. The impact of surgical plume exposure on oxidative stress and respiratory Histopathology: evaluating the role of antioxidant interventions. Dutse Journal of Pure and Applied Sciences. 2025 Jun 11;11(2a):35-43.
28. Enoghase RJ, Osarinmwian IB, Osegbe ED, Abu J, Abukadiri M, Ajufoh CZ. Hepatoprotective Effects of Lawsonia inermis Aqueous Extract Against Lead Acetate-Induced Liver Injury in Wistar Rats. Medtigo Journal of Medicine. 2024;2(4):e30622418. https://doi.org/10.63096/medtigo30622418
29. Areloegbe SE, Olaniyi KS. Acetate mitigates cardiac mitochondrial dysfunction in experimental model of polycystic ovarian syndrome by modulating GPCR41/43 and PROKR1. Biochemical and Biophysical Research Communications. 2023 Nov 12;681:62-72. https://doi.org/10.1016/j.bbrc.2023.09.061
30. Bashir AA, Olaniyi KS. Butyrate alleviates renal inflammation and fibrosis in a rat model of polycystic ovarian syndrome by suppression of SDF-1. BMC Pharmacology and Toxicology. 2023 Oct 3;24(1):48. https://doi.org/10.1186/s40360-023-00692-9
31. Areloegbe SE, Oyekanmi OA, Ajadi IO, Ajadi MB, Atuma CL, Aturamu A, Olaniyi KS. Hepatic dysmetabolism in polycystic ovarian syndrome: impact of paraoxonase-1 modulation by butyrate. Comparative Clinical Pathology. 2024 Aug;33(4):623-32. https://doi.org/10.1007/s00580-024-03580-8
32. Olaniyi KS, Akintayo CO, Oladimeji TE, Areloegbe SE, Badejogbin OC, Bashir AA, Agan SU, Fafure AA, Adekeye AO, Ajadi MB, Owolabi OV. Butyrate ameliorates ovarian failure in experimental PCOS rat model by suppression of HDAC2. Comparative Clinical Pathology. 2025 Feb;34(1):155-67. https://doi.org/10.1007/s00580-025-03633-6
33. Zhang Y, Li K, Kong A, Zhou Y, Chen D, Gu J, Shi H. Dysregulation of autophagy acts as a pathogenic mechanism of non-alcoholic fatty liver disease (NAFLD) induced by common environmental pollutants. Ecotoxicology and Environmental Safety. 2021 Jul 1;217:112256. https://doi.org/10.1016/j.ecoenv.2021.112256
34. Matyas C, Haskó G, Liaudet L, Trojnar E, Pacher P. Interplay of cardiovascular mediators, oxidative stress and inflammation in liver disease and its complications. Nature Reviews Cardiology. 2021 Feb;18(2):117-35. https://doi.org/10.1038/s41569-020-0433-5
35. Ebert T, Neytchev O, Witasp A, Kublickiene K, Stenvinkel P, Shiels PG. Inflammation and oxidative stress in chronic kidney disease and dialysis patients. Antioxidants & redox signaling. 2021 Dec 10;35(17):1426-48. https://doi.org/10.1089/ars.2020.8184
36. Haidar Z, Fatema K, Shoily SS, Sajib AA. Disease-associated metabolic pathways affected by heavy metals and metalloid. Toxicology reports. 2023 Jan 1;10:554-70. https://doi.org/10.1016/j.toxrep.2023.04.010
37. Dutta S, Gorain B, Choudhury H, Roychoudhury S, Sengupta P. Environmental and occupational exposure of metals and female reproductive health. Environmental Science and Pollution Research. 2022 Sep;29(41):62067-92. https://doi.org/10.1007/s11356-021-16581-9
38. Dutta S, Sengupta P, Izuka E, Menuba I, Nwagha U. Oxidative and nitrosative stress and female reproduction: Roles of oxidants and antioxidants. Journal of Integrated Science and Technology. 2024;12(3):754-. https://doi.org/10.62110/sciencein.jist.2024.v12.754
39. Yan F, Zhao Q, Li Y, Zheng Z, Kong X, Shu C, Liu Y, Shi Y. The role of oxidative stress in ovarian aging: a review. Journal of ovarian research. 2022 Sep 1;15(1):100. https://doi.org/10.1186/s13048-022-01032-x
40. Zeliger HI. A Pound of Prevention for a Healthier Life: How and Why Avoiding Exposures to Toxic Chemicals and Other Sources of Oxidative Stress, the Cause of Most

Disease, Lowers the Odds of Getting Sick. Universal-Publishers; 2019 Apr 15.
41. Porter J, Blau E, Gharagozloo F, Martino M, Cerfolio R, Duvvuri U, Caceres A, Badani K, Bhayani S, Collins J, Coelho R. Society of robotic surgery review: Recommendations regarding the risk of COVID-19 transmission during minimally invasive surgery. BJU international. 2020 Aug;126(2):225-34. https://doi.org/10.1111/bju.15105
42. Watters DA, Foran P, McKinley S, Campbell G. Clearing the air on surgical plume. ANZ Journal of Surgery. 2022 Jan;92(1-2):57-61. https://doi.org/10.1111/ans.17340