



## THE IMPACT OF HYDROQUINONE ON KIDNEY HEALTH

**Possible Okikiola Akanbi<sup>1</sup>, Ogechi Cecilia Ofor<sup>2</sup>, Ekaba Samson Gift<sup>3</sup>,  
Chinedum Okafor<sup>4</sup>, Oluwanifemi Omodara Ogunleye<sup>5</sup>,  
Margaret Seun Ijibadejo<sup>6</sup>, Oghenefejiro Dorcas Efeurhobo<sup>7</sup>,  
Deborah Olufunmilayo Adejumo<sup>8</sup>, and Leah Titilayo Olawumi<sup>9</sup>.**

<sup>1</sup>Division of Diagnostic Assay Development and Medical Artificial Intelligence,  
Helix Biogen Institute.

Email: [popoolapossible@gmail.com](mailto:popoolapossible@gmail.com)

<sup>2</sup>Ebonyi State University.

Email: [oforogechi2@gmail.com](mailto:oforogechi2@gmail.com)

<sup>3</sup>University of Port Harcourt.

Email: [sekaba025@uniport.edu.ng](mailto:sekaba025@uniport.edu.ng)

<sup>4</sup>Louisiana State University Health System, Shreveport, Louisiana, USA.

Email: [cojinaka17@gmail.com](mailto:cojinaka17@gmail.com)

<sup>5</sup>Department of Biological Sciences, Covenant University, Ogun State, Nigeria.

Email: [oluwaniifemi.ogunleyepgs@stu.cu.edu.ng](mailto:oluwaniifemi.ogunleyepgs@stu.cu.edu.ng)

<sup>6</sup>Department of Biochemistry, Babcock University Illishan Remo.

Email: [ijibadejomargaret@gmail.com](mailto:ijibadejomargaret@gmail.com)

<sup>7</sup>Department of Pharmacology, Delta State University, Abraka.

Email: [efeurhobodorcas120@gmail.com](mailto:efeurhobodorcas120@gmail.com)

<sup>8</sup>University of Ibadan.

Email: [deborahpelutimi@gmail.com](mailto:deborahpelutimi@gmail.com)

<sup>9</sup>Department of Science Laboratory Technology, Ladoké Akintola University of Technology.

Email: [leahtitilayoolawumi@gmail.com](mailto:leahtitilayoolawumi@gmail.com)

**Cite this article:**

Akanbi, P. O., Ofor, C. O., Ekaba, S. G., Okafor, C., Ogunleye, O. O., Ijibadejo, M. S., Efeurhobo, D. O., Adejumo, D. O., Olawumi, T. L. (2025), The Impact of Hydroquinone on Kidney Health. International Journal of Public Health and Pharmacology 5(2), 75-87. DOI: 10.52589/IJPHP-UMHXM0ON

**Manuscript History**

Received: 28 May 2025

Accepted: 16 Jul 2025

Published: 24 Oct 2025

**Copyright** © 2025 The Author(s).

This is an Open Access article distributed under the terms of Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0), which permits anyone to share, use, reproduce and redistribute in any medium, provided the original author and source are credited.

**ABSTRACT:** *Hydroquinone (1,4-dihydroxybenzene) is a chemical substance with widespread uses in cosmetics, industry, and medicine as a skin-lightening agent and antioxidant. In this article, the chemical nature of hydroquinone, metabolic transformations involved, and toxicological significance, with emphasis on its renal impact, have been addressed. Even though hydroquinone is anti-hyperpigmentary and anti-melasma, it has significant risks due to its ability to cause oxidative stress, nephrotoxicity, and systemic toxicity after prolonged exposure. The medication is metabolized into reactive metabolites with the ability to damage renal tissues and disrupt cellular redox homeostasis. Chronic exposure, especially via unregulated cosmetics, increases the likelihood of adverse health effects like kidney damage, exogenous ochronosis, and carcinogenicity. Hydroquinone product prevalence in locales of elevated skin lightening demand fueled by sociocultural constructions of beauty underscores the need for greater regulatory oversight and education. This article emphasizes the importance of cosmetovigilance as a public health approach and endorses global regulatory measures to minimize the risks in the application of hydroquinone while ensuring consumer safety.*

**KEYWORDS:** Hydroquinone, Public health Cosmetovigilance, Cosmetics, Kidney health.



## INTRODUCTION

In 1820, Pelletier and Caventou used the dry distillation of quinic acid to produce hydroquinone, also known as 1,4-dihydroxybenzene. Friedrich Wohler came up with the term around 1884. Hydroquinone has a melting point of 173 degrees Celsius; it is a white, crystalline powder (Bogels et al., 1997; Banodka et al., 2022), and it is the major benzene metabolite. It is a chemical everywhere in the environment due to its extensive use in industrial and human processes. In European Union nations, hydroquinone was used in commercially available skin-lightening cosmetic formulations from the 1950s to 2001. Since the 1960s, it has also been offered for sale as a medicinal medication. It can be found in cosmetic formulations of items used to coat hair dyes and fingernails (O'Donoghue et al., 2006). Additionally, it can be utilized as a stabilizer in paints, varnishes, lubricants, and motor fuels, as well as a developing agent in photography and a dye intermediate (Enguita & Leitão, 2013). A medication that is frequently used for melasma, post-inflammatory hyperpigmentation, and other conditions is hydroquinone. It is an aromatic chemical molecule and a strong inhibitor of melanin synthesis. The side effects that are frequently mentioned include exogenous ochronosis and irritating contact dermatitis. It may, however, in rare cases, result in irreversible leukoderma (Das et al., 2019). Hyperpigmentation, depigmentation, conjunctival melanosis, corneal degeneration, nail discoloration, exogenous ochronosis, and irritating contact dermatitis are hydroquinone's side effects. Erythema, pruritus, mild edema, burning, and scaling are the hallmarks of irritant contact dermatitis—the most prevalent side effect (Das et al., 2019). Berries that contain arbutin and the leaves of other plants are examples of natural sources of hydroquinone. Hydroquinone is produced from the hydrolysis of arbutin (Banodka et al., 2022).

Lightening of skin tones is common among women, young girls, and certain men worldwide (Shroff et al., 2018). Due to a significant demand for cosmetics that lighten skin tones, the skin-lightening sector is expanding quickly on a global basis. The belief that having light skin is a sign of beauty, prosperity, and power is largely responsible for society's excessive need for skin-lightening cosmetics (Bamidele et al., 2023). Additional justifications for applying skin-lightening cosmetics include enhancing one's appearance, adhering to current fashion trends, curing skin conditions like melasma or acne, enhancing one's body and facial complexion, and appeasing one's spouse (Olumide et al., 2008). These skin-lightening cosmetics, which are unfortunately being smuggled into countries, include dangerous metals and hydroquinone, among other prohibited ingredients (Dlova et al., 2015). The fact that many of these cosmetics contain dangerous ingredients, including hydroquinone, mercury (Hg), and arsenic (As), raises serious concerns (Alam et al., 2019; Chen et al., 2020; Balali-Mood et al., 2012). In skin-lightening cosmetics, hydroquinone and mercury have both been shown to be potentially harmful substances.

According to data from the National Toxicology Program (NTP), hydroquinone increased the incidence of renal tubule adenomas in male F344 rats but not in females, liver adenomas, and thyroid gland follicular cell hyperplasia in both male and female B6C3F1 mice (O'Donoghue et al., 2021). Hard et al.'s (1997) reexamination of renal pathology in rats used in the NTP hydroquinone bioassay revealed that male rats' renal adenomas colocalized with the more severe types of chronic progressive nephropathy (CPN). Additional research by Hard et al. (2012) using F344 rats and NTP cancer bioassays of 24 other chemicals demonstrated unmistakable proof of a qualitative and statistically significant correlation between the development of low-grade renal tubule tumors and atypical renal cell hyperplasia, which are

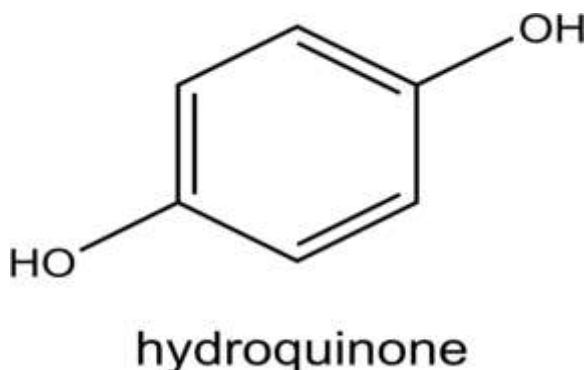
comparable to those observed in male F344 rats after exposure to hydroquinone, and advanced stages of CPN severity.

Human exposure to hydroquinone, a chemical that is widely used in many industrial, cosmetic, and pharmaceutical applications, is becoming more common. Because of its extensive use in skincare products, industrial processes, and even some foods, hydroquinone has sparked serious concerns about its possible health effects, especially on renal function. Understanding how it impacts renal health is crucial since the kidneys are particularly susceptible to damage from chemicals and are essential for detoxification. In addition to analyzing hydroquinone in detail, this review looks at its sources, applications, and frequency of human exposure. The chemistry, metabolism, and toxicokinetics of hydroquinone are also thoroughly explained. It further explains the process by which hydroquinone causes nephrotoxicity. This study has summarized recent studies to highlight the significance of investigating hydroquinone-related kidney toxicity, its consequences for public health, and potential mitigation.

### Chemical Structure and Properties of Hydroquinone

Hydroquinone is characterized by its aromatic benzene ring with two hydroxyl groups at the para position, making it a dihydroxybenzene derivative (Fónagy et al., 2021).

**Figure 1: Chemical structure of hydroquinone (C<sub>6</sub>H<sub>6</sub>O<sub>2</sub>)**



This structure allows it to participate in various chemical reactions, including oxidation and polymerisation (Koltzenburg et al., 2023).

### Properties of Hydroquinone

Hydroquinone exhibits interesting redox properties. It undergoes reversible oxidation to form quinones. This process involves the transfer of protons and electrons, which can be influenced by the presence of intramolecular hydrogen bonds (Ansari et al., 2025). Hydroquinone can also be polymerised through chemical oxidative reactions, forming poly(para-hydroquinone) (PPHQ). This polymerisation is facilitated by oxidants like titanium tetrachloride and involves the formation of a conjugated polymer with distinct vibrational and optical properties (Gregory et al., 2021). Additionally, hydroquinone can undergo electro-oxidative polymerisation to form poly(dihydroxyphenylalanine), which exhibits electroactive



properties (Pandikumar et al., 2021). It can form clathrates, which are crystalline structures that can encapsulate guest molecules, such as CO<sub>2</sub> and CH<sub>4</sub> (Englezos, 2024).

Hydroquinone and its derivatives, particularly in prenylated forms, exhibit antioxidant properties. These compounds can transform into para-quinones, which interact with enzymes and proteins, potentially offering neuroprotective effects and applications in preventing degenerative diseases (Giner et al., 2022). Hydroquinone's electron-rich nature makes it suitable for chemical sensing applications. It can be used in the synthesis of conjugated microporous polymers, which have been shown to have excellent fluorescence sensing performance for detecting compounds like 2,4-dinitrophenol and 2,4,6-trinitrophenol (Geng et al., 2021).

### **Metabolic Pathways of Hydroquinone in the Human Body**

Hydroquinone, a metabolite of benzene, is known for its potential toxic effects and involvement in various metabolic pathways in the human body (Valenzuela et al., 2024). Understanding these pathways is crucial for assessing the risks associated with hydroquinone exposure and its implications for human health.

Hydroquinone is metabolised by several cytochrome P450 enzymes, which play a significant role in its biotransformation. These enzymes include CYP2D6, CYP3A4, and CYP2C8, which are also involved in the metabolism of other compounds like hydroxychloroquine (Paludetto et al., 2022). The activity of these enzymes can influence the rate and extent of hydroquinone metabolism, affecting its toxicity and pharmacokinetics. Hydroquinone exposure can lead to cellular damage through various mechanisms. It has been shown to induce ferroptosis, a form of cell death characterized by iron-dependent lipid peroxidation, in Jurkat cells. This process involves increased levels of intracellular Fe<sup>2+</sup>, malondialdehyde, and reactive oxygen species, along with decreased glutathione levels (Liu et al., 2024). Additionally, hydroquinone can cause DNA damage and cell cycle abnormalities, potentially leading to malignant transformations through pathways involving JNK1 signalling (Yu et al., 2023).

### **Role of the Kidney in Excreting Hydroquinone and Its Metabolites**

The kidneys play a crucial role in the excretion of hydroquinone and its metabolites, primarily through processes involving conjugation and subsequent elimination via urine (Georgiou-Siafis et al., 2023). These metabolites can cause significant renal damage through oxidative stress and cellular toxicity. Hydroquinone is metabolised into various conjugates, which are then excreted by the kidneys, often contributing to nephrotoxicity (Potęga et al., 2022).

Hydroquinone and its derivatives, such as tert-butyl-hydroquinone (TBHQ), undergo glutathione (GSH) conjugation, forming nephrotoxic metabolites. These conjugates are excreted in urine and can cause kidney damage, as evidenced by increased urinary excretion of enzymes like gamma-glutamyl transpeptidase and alkaline phosphatase, indicating renal stress and damage (Khezerlou et al., 2022).

Hydroquinone is metabolized into sulfur-containing compounds, which are excreted in urine and bile. These metabolites are potentially nephrotoxic and contribute to the renal and bladder carcinogenicity observed in rats (Hard et al., 2021). The nephrotoxic effects of



hydroquinone metabolites are linked to their ability to induce oxidative stress and cell damage in kidney tissues.

This is partly due to the formation of reactive oxygen species (ROS) and the interaction of quinone-thioether metabolites with mitochondrial proteins, exacerbating oxidative damage (Bolton et al., 2017).

### **Effects of Prolonged Exposure**

Persistent, bioaccumulative, and toxic chemicals, such as heavy metals and organochlorines, have been linked to increased cancer risks, particularly when present in mixtures (Zamora et al., 2021). Long-term exposure to bioaccumulative substances can lead to significant biological effects, such as reduced growth rates, altered reproductive conditions, and increased DNA damage in organisms like fish and earthworms (Borgå et al., 2022). Bioaccumulation can lead to biomagnification, where the concentration of a substance increases up the food chain, potentially causing adverse effects in predators and entire ecosystems (Ali et al., 2019). As an environmental pollutant, hydroquinone can adversely affect cartilage homeostasis by activating the Aryl Hydrocarbon Receptor pathway. This activation leads to oxidative stress and degradation of cartilage, contributing to joint diseases (Fehsel et al., 2024).

### **The Role of Hydroquinone in Kidney Damage**

Significant injury to renal cells would occur when ROS levels increase, causing apoptosis and fibrosis, which ultimately contribute to the decline in kidney function (Verma et al., 2021). Oxidative stress induces kidney damage through several mechanisms, including mitochondrial dysfunction, inflammatory responses, and the upregulation of genes associated with oxidative phosphorylation. These processes aggravate ROS production (Verma et al., 2021; Piko et al., 2023).

Oxidative stress is often defined as an imbalance between pro-oxidants and antioxidants (Jones D. P., 2006). A complex antioxidant defense mechanism generally maintains ROS attack in balance, since living cells are constantly under oxidative attack from ROS, causing “oxidative damage” (Burton & Jauniaux, 2011). Cell viability, activation, proliferation, and organ function are maintained by the regulation of redox (reduction and oxidation) state. A pathological change in that balance leads to growth in ROS concentrations, which results in adverse modifications to cell components, such as lipids, proteins, and DNA (Birben et al., 2012). Oxidative stress has been widely recognized as a risk factor for various detrimental events, including atherosclerosis and mortality in chronic kidney disease (CKD) patients. At the onset of the disease, oxidative stress is associated with the decline of renal function, which is further escalated by hemodialysis (Yari et al., 2020).

Lipids, proteins, carbohydrates, and nucleic acids in biological tissues are under constant oxidative attack due to the presence of Reactive Oxygen Species, which have an adverse effect on their structure and function. ROS plays an important role in the pathophysiology of renal impairment, facilitating chronic kidney disease progression as well (Coppolino et al., 2018).

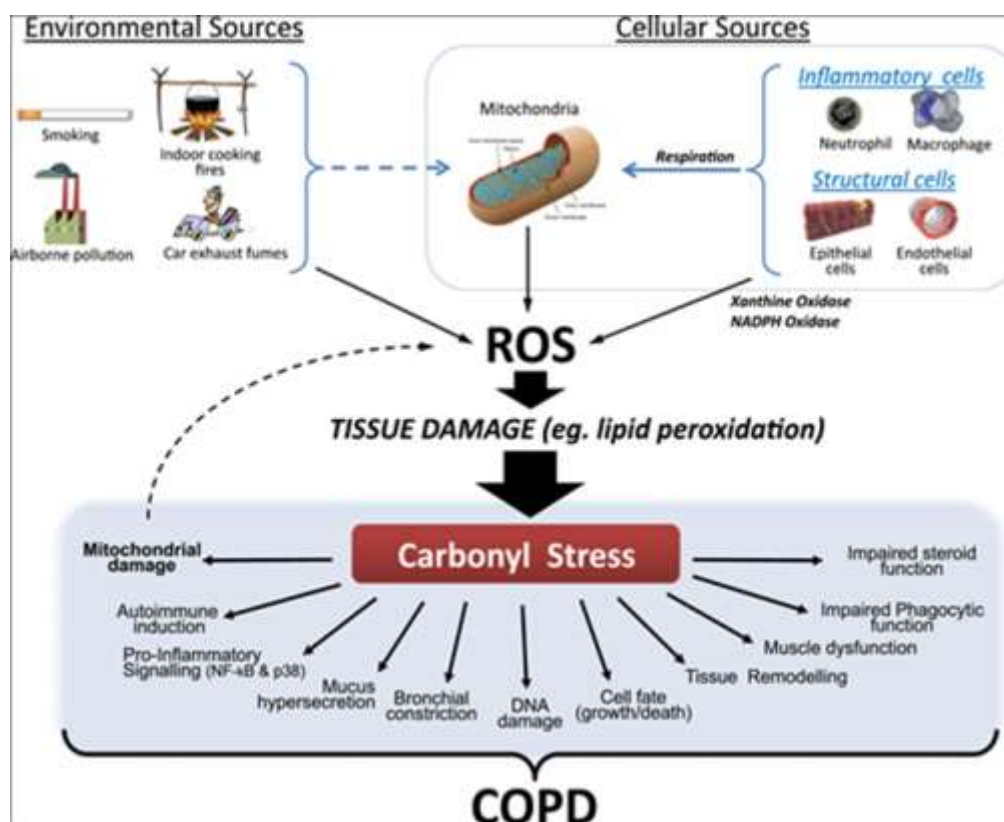
Oxidative stress and ROS production in the kidney interrupt the excretory function of each section of the nephron. Functions such as water–electrolyte and acid–base balance, kidney



regulatory mechanisms, tubular glomerular feedback, myogenic reflex in the supplying arteriole, and the renin–angiotensin–aldosterone system are impaired by ROS and oxidative stress (Podkowi et al., 2020). Podocyte damage (edema, apoptosis, and necrosis), depressed glomerular filtration rate, proteinuria (Snoeijs et al., 2010), and tubulointerstitial fibrosis have been linked to oxidative stress (Duni et al., 2019)

Hydroquinone has been found to stimulate genotoxicity, such as DNA damage, facilitated by oxidative stress in these cell models (Peng et al. 2012). It easily undergoes automatic oxidation to H<sub>2</sub>O<sub>2</sub> and semiquinone radicals, which are remarkably reactive molecules (Luo et al., 2008), causing oxidative stress (Rubio et al., 2011) and successive oxidative damage (Luo et al., 2008).

**Figure 2: Showing the impact of oxidative stress on the tissue from different sources (Podkowi et al., 2020)**



### Populations Exposed to Hydroquinone

Hydroquinone has been extensively utilized in various industries, including the manufacturing of rubber, paints, varnishes, motor fuels, oils, and as a reagent in photographic developers and in cosmetic products (McGregor, 2007). Furthermore, in industrial sources, hydroquinone exists in free form and as  $\beta$ -D-glucopyranoside conjugate (arbutin) in some bacteria, plants, coffee, red wine, and wheat cereals (Deisinger et al. 1996) as well as in cosmetics (Olumide et al. 2008). In some geographical regions, skin whitening is becoming prominent amongst men and young adults, although it is basically practised by women (Benn et al., 2016). Fair complexion is believed to be a synonym for beauty, and the widespread skin-lightening practices that result come from a complex interweaving of historical, cultural,



social, psychological, and economic factors. It is estimated that about 75% of women in Nigeria, 60% in Senegal, 50% in Mali and 30% in Ghana regularly use bleaching products (Pollock et al., 2021).

### **Cosmetic Use and Its Potential Systemic Effects**

The use of cosmetic products is increasing around the world, and the variety of chemical compounds used in the manufacture of these products is growing at the same time. In this way, the risk of intoxication, allergic processes, prolonged chemical exposure, side effects, and indiscriminate use is also increased. The side effects derived from the use of cosmetics pose health risks mainly due to exposure to numerous chemical substances. Its consequences can range from a simple mild hypersensitivity reaction to an anaphylactic process or even a lethal intoxication.

Cosmetics and skin care products are currently consumed worldwide, with frequent use, increasing the exposure of the human body to the various chemical compounds that make up its formulas. The prevalence of cosmetic side effects is challenging, as those with mild reactions often do not seek medical advice (Draelos, 2015). The side effects of cosmetics can indeed pose significant health risks, largely due to the extensive range of chemical substances present in their formulations. The outcomes of such exposure can vary widely—from mild hypersensitivity reactions, such as skin redness or itching, to severe cases like anaphylactic shock or, in rare situations, lethal intoxication. These risks emphasize the need for stringent regulation, proper labeling of ingredients, and increased consumer awareness to minimize potential harm (Alani J. I. et al., 2013).

Cosmetic ingredients are increasingly being recognized as emerging pollutants, with environmental monitoring still in its infancy. These substances enter the environment through various pathways, such as wastewater discharge, and often accumulate in aquatic ecosystems. Their presence can disrupt marine and freshwater biodiversity, affecting organisms at multiple trophic levels. Moreover, the persistence of certain chemicals, like microplastics, UV filters, and parabens, poses long-term health risks not only to aquatic ecosystems but also to humans, as these pollutants can enter the food chain (Nicolopoulou-Stamati P et al., 2015). In public health science, the term "cosmetovigilance" has become known as a kind of health surveillance aimed at ensuring the safety of cosmetic products for commercial purposes. Cosmetovigilance is very important to control the potential risk posed by hazardous ingredients present in cosmetics; by assessing adverse reactions and ensuring compliance with safety regulations, this practice plays a crucial role in preventing consumer health. (Vigan & Castelain, 2014).

### **Observation Associations with Kidney Diseases or Dysfunction**

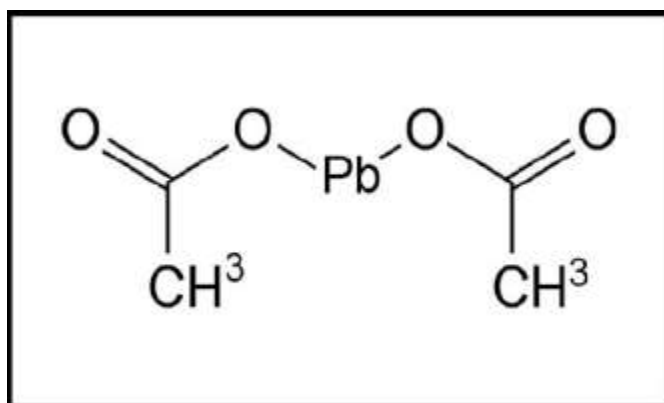
Hazardous substances in the manufacture of cosmetics are toxic heavy metals, such as lead (Pb), cadmium (Cd), nickel (Ni), arsenic (As), and mercury (Hg). Some cosmetics may contain aluminum (Al), classified as a light metal.

Cadmium, a heavy metal, is found in body and hair creams, which have the potential to pose significant health risks due to its ability to penetrate the body through dermal contact. Cadmium (Cd) may accumulate in the kidneys, with possible damage. Chronic exposure to low levels of cadmium can also cause bone fragility and consequent bone fractures. Also, acute ingestion of high levels of cadmium can result in severe stomach irritation, nausea, and



vomiting. Prolonged exposure, at lower levels, increases the risk of chronic conditions, especially kidney damage, due to its accumulation in the body (Khan et al., 2019). Cobalt (Co) and nickel (Ni) can cause allergies such as contact dermatitis, and these metals are commonly present in cosmetics such as eye shadow, face paint, hair cream, and lipstick. Lead (Pb), when ingested in large quantities, may interfere with the synthesis of hemoglobin and calcium channels, whose functions are important for nerve conduction. Lead is found in dyes for hair (such as lead acetate, whose chemical formula is shown in Figure 2) and lipsticks, eyeliners, eye pencils, and hair creams in their inorganic form, and it can be minimally absorbed by the skin.

**Figure 3: Chemical formula of Lead acetate**



Mercury is a significant ingredient in many skin-lightening creams, soaps, and eye makeup, appearing in both organic and inorganic formulations. The inorganic form of mercury, found in products, accumulates in the kidneys, leading to kidney damage over a long period of exposure. Cases of inorganic mercury poisoning have been reported globally, including in the USA, Mexico, Africa, and Europe (Mehrdad et al., 2014). Due to the toxicity, the US FDA has banned the use of nine specific ingredients in cosmetics, while the EU has taken a stricter stance by banning over 1,000 potentially harmful ingredients (Jadhav V. et al., 2016).

## CONCLUSION

Due to increasing consciousness about beauty, people are using more cosmetic products, which results in increasing adverse events. Cosmetic and beauty products are not that safe for health, as they contain more than 10,000 ingredients that can be linked to disease. Due to the continued use of cosmetics, people are exposed to adverse effects such as contact dermatitis, allergy, redness of skin, type IV hypersensitivity, etc. Cosmetic ingredients used in cosmetic products have a direct effect on the hormonal systems of the human body. The public should be warned about the presence of toxic ingredients in cosmetic products. It is important to implement a global cosmetovigilance system to encourage changes in cosmetic product manufacturing, marketing, and use by the general population. Such a system would increase the safety of these products and their ingredients and prevent the adverse effects of these products. It is important to carry out studies investigating the adverse effects of cosmetics. We suggest that researchers across the globe, especially from developing and underdeveloped countries, perform more and more novel studies, particularly on the adverse effects of cosmetics.



## REFERENCES

- Alam, M. F., Akhter, M., Mazumder, B., Ferdous, A., Hossain, M. D., Dafader, N. C., ... & Atique Ullah, A. K. M. (2019). Assessment of some heavy metals in selected cosmetics commonly used in Bangladesh and human health risk. *Journal of Analytical Science and Technology*, 10(1), 1-8.
- Alani, J. I., Davis, M. D. P., & Yiannias, J. A. (2013). Allergy to cosmetics: A literature review. *Dermatitis*, 24(6), 283-290.
- Ali, H., & Khan, E. (2019). Trophic transfer, bioaccumulation, and biomagnification of non-essential hazardous heavy metals and metalloids in food chains/webs—Concepts and implications for wildlife and human health. *Human and Ecological Risk Assessment: An International Journal*, 25(6), 1353-1376.
- Ansari, S. P., & Dar, R. A. (2025). Quinones as redox-active materials for energy applications. In *Quinone-Based Compounds in Drug Discovery* (pp. 209-228). Academic Press.
- Balali-Mood, M., Naseri, K., Tahergorabi, Z., Khazdair, M. R., & Sadeghi, M. (2021). Toxic mechanisms of five heavy metals: Mercury, lead, chromium, cadmium, and arsenic. *Frontiers in Pharmacology*, 12, 643972.
- Bamidele, O. D., Kayode, B. A., Eniayewu, O. I., Adegbola, A. J., Olatoye, R. S., Njinga, N. S., ... & Bakare-Odunola, M. T. (2023). Quality assessment of hydroquinone, mercury, and arsenic in skin-lightening cosmetics marketed in Ilorin, Nigeria. *Scientific Reports*, 13(1), 20992.
- Banodkar, P. D., & Banodkar, K. P. (2022). History of hydroquinone. *Indian Journal of Dermatology, Venereology and Leprology*, 88(5), 696-699.
- Benn, E.K.T.; Alexis, A.; Mohamed, N.; Wang, Y.-H.; Khan, I.A.; Liu, B. Skin bleaching and dermatological health of African and Afro-Caribbean populations in the US; new directions for methodologically rigorous, multidisciplinary, and culturally sensitive research. *Dermatol. Ther.* **2016**, 6, 453–459.
- Birben, E.; Sahiner, U.M.; Sackesen, C.; Erzurum, S.; Kalayci, O. Oxidative stress and antioxidant defense. *World Allergy Organ. J.* 2012, 5, 9-19.
- Bögels, G., Pot, T. M., Meekes, H., Bennema, P., & Bollen, D. (1997). In *Proceedings of the International Symposium on Silver Halide Imaging*.
- Bolton, J. L., & Dunlap, T. (2017). Formation and biological targets of quinones: Cytotoxic versus cytoprotective effects. *Chemical Research in Toxicology*, 30(1), 13-37.
- Borgå, K., et al. (2022). The influence of global climate change on accumulation and toxicity of persistent organic pollutants and chemicals of emerging concern in Arctic food webs. *Environmental Science: Processes & Impacts*, 24(10), 1544-1576.
- Burton, G.J.; Jauniaux, E. Oxidative stress. *Best Pract. Res. Clin. Obs. Gynaecol.* 2011, 25, 287–299. [CrossRef]
- Chen, J., Ye, Y., Ran, M., Li, Q., Ruan, Z., & Jin, N. (2020). Inhibition of tyrosinase by mercury chloride: Spectroscopic and docking studies. *Frontiers in Pharmacology*, 11, 81.
- Coppolino, G.; Leonardi, G.; Andreucci, M.; Bolignano, D. Oxidative Stress and Kidney Function: A Brief Update. *Curr. Pharm. Des.* 2018, 24, 4794-4799.
- Das, A., Ghosh, A., & Kumar, P. (2019). Chemical leukoderma due to hydroquinone: An unusual phenomenon. *Indian Journal of Dermatology, Venereology and Leprology*, 85, 567.



- Deisinger PJ, Hill TS, English JC. Human exposure to naturally occurring hydroquinone. *J Toxicol Env Health*. 1996;47: 31-46.
- Dlova, N. C., Hamed, S. H., Tsoka-Gwegweni, J., & Grobler, A. (2015). Skin lightening practices: An epidemiological study of South African women of African and Indian ancestries. *British Journal of Dermatology*, 173(S2), 2-9.
- Draelos, Z. D. (2015). Cosmetics: The medicine of beauty. *Journal of Cosmetic Dermatology*, 14(2), 91.
- Duni, A.; Liakopoulos, V.; Roumeliotis, S.; Peschos, D.; Dounousi, E. Oxidative Stress in the Pathogenesis and Evolution of Chronic Kidney Disease: Untangling Ariadne's Thread. *Int. J. Mol. Sci*. 2019, 20, 3711.
- Englezos, P. (2024). Phase equilibrium in canonical cubic structure I (sI) and II (sII) and hexagonal (sH) gas hydrate solid solutions. *Fluid Phase Equilibria*, 578, 114005.
- Enguita, F. J., & Leitão, A. L. (2013). Hydroquinone: Environmental pollution, toxicity, and microbial answers. *BioMed Research International*, 2013(1), 542168.
- Fehsel, K. (2024). Metabolic side effects from antipsychotic treatment with clozapine linked to aryl hydrocarbon receptor (AhR) activation. *Biomedicines*, 12(10), 2294.
- Fónagy, O., Szabo-Bardos, E., & Horváth, O. (2021). 1,4-Benzoquinone and 1,4-hydroquinone based determination of electron and superoxide radical formed in heterogeneous photocatalytic systems. *Journal of Photochemistry and Photobiology A: Chemistry*, 407, 113057.
- Geng, T., Liu, M., Hu, C., & Zhu, H. (2021). The synthesis of conjugated microporous polymers via nucleophilic substitution of hydroquinone with cyanuric chloride and hexachlorocyclotriphosphazene for sensing to 2,4-dinitrophenol and 2,4,6-trinitrophenol. *New Journal of Chemistry*, 45, 3007-3013. <https://doi.org/10.1039/D0NJ06099B>
- Georgiou-Siafis, S. K., & Tsiftoglou, A. S. (2023). The key role of GSH in keeping the redox balance in mammalian cells: Mechanisms and significance of GSH in detoxification via formation of conjugates. *Antioxidants*, 12(11), 1953.
- Giner, R., Ríos, J., & Máñez, S. (2022). Antioxidant activity of natural hydroquinones. *Antioxidants*, 11. <https://doi.org/10.3390/antiox11020343>
- Gregory, S. A., et al. (2021). Vapor phase infiltration doping of the semiconducting polymer poly (aniline) with  $TiCl_4 + H_2O$ : Mechanisms, reaction kinetics, and electrical and optical properties. *ACS Applied Polymer Materials*, 3(2), 720-729.
- Hard, G. C. (2021). Comparative kidney carcinogenesis in laboratory rodents and humans. In *Carcinogenicity* (pp. 439-466). CRC Press.
- Hard, G. C., Betz, L. J., & Seeley, J. C. (2012). Association of advanced chronic progressive nephropathy (CPN) with renal tubule tumors and precursor hyperplasia in control F344 rats from two-year carcinogenicity studies. *Toxicologic Pathology*, 40, 473-481.
- Hard, G. C., Whysner, J., English, J. C., Zang, E., & Williams, G. M. (1997). Relationship of hydroquinone-associated rat renal tumors with spontaneous chronic progressive nephropathy. *Toxicologic Pathology*, 25(2), 132-143.
- Jadhav, V., Dhande, S., & Kadam, V. (2016). Cosmetic's side effects. *World Journal of Pharmaceutical and Pharmaceutical Sciences*, 6, 327-343.
- Jones, D.P. Redefining oxidative stress. *Antioxid Redox Signal*. **2006**, 8, 1865–1879.
- Khan, A., & Alam, M. (2019). Cosmetics and their associated adverse effects: A review. *Journal of Applied Pharmaceutical Science Research*, 2, 1-6.
- Khezerlou, A., et al. (2022). Alarming impact of the excessive use of tert-butylhydroquinone in food products: A narrative review. *Toxicology Reports*, 9, 1066-1075.



- LH, Jiang LP, Geng CY, Cao J, Zhong LF. Hydroquinone-induced genotoxicity and oxidative DNA damage in HepG2 cells. *Chem Biol Interact.* 2008;173:1-8.
- Liu, N., Liu, G., Li, Q., Hu, Y., & Wang, H. (2024). Effects on iron metabolism and System Xc<sup>-</sup>/GPX4 pathway from hydroquinone suggest ferroptosis of Jurkat cells. *Toxics*, 12. <https://doi.org/10.3390/toxics12090644>.
- McGregor D. Hydroquinone: an evaluation of the human risks from its carcinogenic and mutagenic properties. *Crit Rev Toxicol.* 2007;37:887–914.
- Mehrdad, R. R., Mehravar, R. R., Sohrab, K., & Moghadamnia, A. A. (2014). Current approaches to the management of mercury poisoning: Need of the hour. *DARU Journal of Pharmaceutical Sciences*, 22, 22-46.
- Nicolopoulou-Stamati, P., Hens, L., & Sasco, A. J. (2015). Cosmetics as endocrine disruptors: Are they a health risk? *Reviews in Endocrine and Metabolic Disorders*, 16(4), 373-383.
- O'Donoghue, J. (2006). Hydroquinone and its analogues in dermatology—a risk-benefit viewpoint. *Journal of Cosmetic Dermatology*, 5(3), 196-203.
- Olumide, Y. M., Akinkugbe, A. O., Altraide, D., Mohammed, T., Ahamefule, N., Ayanlowo, S., ... & Essen, N. (2008). Complications of chronic use of skin lightening cosmetics. *International Journal of Dermatology*, 47(4), 344-353.
- Paludetto, M., Kurkela, M., Kahma, H., Backman, J., Niemi, M., & Filppula, A. (2022). Hydroxychloroquine is metabolized by cytochrome P450 2D6, 3A4, and 2C8, and inhibits cytochrome P450 2D6, while its metabolites also inhibit cytochrome P450 3A in vitro. *Drug Metabolism and Disposition*, 51, 293-305. <https://doi.org/10.1124/dmd.122.001018>.
- Pandikumar, A., & Devi, K. S. S. (Eds.). (2021). Disposable electrochemical sensors for healthcare monitoring: Material properties and design (Vol. 21). Royal Society of Chemistry.
- Peng D, Jiaying W, Chunhui H, Weiyi P, Xiaomin W. Study on the cytogenetic changes induced by benzene and hydroquinone in human lymphocytes. *Hum Exp Toxicol.* 2012;31:322-35.
- Pereira, J. X., & Pereira, T. C. (2018). Cosmetics and its health risks. *Global Journal of Medical Research*, 18, 63-70.
- Piko, N., Bevc, S., Hojs, R., & Ekart, R. (2023). The role of oxidative stress in kidney injury. *Antioxidants*, 12(9), 1772. <https://doi.org/10.3390/antiox12091772>.
- Podkowińska, A.; Formanowicz, D. Chronic Kidney Disease as Oxidative Stress- and Inflammatory-Mediated Cardiovascular Disease. *Antioxid* 2020, 9.
- Pollock, S.; Taylor, S.; Oyerinde, O.; Nurmohamed, S.; Dlova, N.; Sarkar, R.; Galadari, H.; Manela-Azulai, M.; Chung, H.S.; Handog, E.; et al. The dark side of skin lightening: An international collaboration and review of a public health issue affecting dermatology. *Int. J. Women Dermatol.* **2021**, 7, 158-164.
- Potęga, A. (2022). Glutathione-mediated conjugation of anticancer drugs: An overview of reaction mechanisms and biological significance for drug detoxification and bioactivation. *Molecules*, 27(16), 5252.
- Rubio V, Zhang J, Valverde M, Rojas E, Shi ZZ. Essential role of Nrf2 in protection against hydroquinone- and benzoquinone-induced cytotoxicity. *Toxicol in Vitro.* 2011;25:521-9.
- Shroff, H., Diedrichs, P. C., & Craddock, N. (2018). Skin color, cultural capital, and beauty products: An investigation of the use of skin fairness products in Mumbai, India. *Frontiers in Public Health*, 5, 365.



- Snoeijs, M.G.J.; van Heurn, L.W.E.; Buurman, W.A. Biological modulation of renal ischemia-reperfusion injury. *Curr. Opin Organ. Transplantation* 2010, 15, 190-199.
- Tabriziani, H., Lipkowitz, M. S., & Vuong, N. (2018). Chronic kidney disease, kidney transplantation and oxidative stress: A new look to successful kidney transplantation. *Clinical Kidney Journal*, 11(1), 130–135. <https://doi.org/10.1093/ckj/sfx091>
- Valenzuela, A., et al. (2024). Hydroquinone ecotoxicity: Unveiling risks in soil and river ecosystems with insights into microbial resilience. *Toxics*, 12(2), 115.
- Verma, S., Singh, P., Khurana, S., Ganguly, N. K., Kukreti, R., Saso, L., Rana, D. S., Taneja, V., & Bhargava, V. (2021). Implications of oxidative stress in chronic kidney disease: A review on current concepts and therapies. *Kidney Research and Clinical Practice*, 40(2), 183-193. <https://doi.org/10.23876/j.krcp.20.163>
- Vigan, M., & Castelain, F. (2014). Cosmetovigilance: Definition, regulation and use “in practice.” *European Journal of Dermatology*, 24(6), 643-649.
- Yari, Z.; Tabibi, H.; Najafi, I.; Hedayati, M.; Movahedian, M. Effects of soy isoflavones on serum systemic and vascular inflammation markers and oxidative stress in peritoneal dialysis patients: A randomized controlled trial. *Phytother. Res.* 2020, 34, 3011-3018.
- Yu, L., Qiu, W., Gao, Y., Sun, M., Chen, L., Cui, Z., ... & Luo, H. (2023). JNK1 activated pRb/E2F1 and inhibited p53/p21 signaling pathway is involved in hydroquinone-induced pathway malignant transformation of TK6 cells by accelerating the cell cycle progression. *Environmental Toxicology*, 38, 2344-2351. <https://doi.org/10.1002/tox.23870>.
- Zamora, R., & Hidalgo, F. J. (2021). Formation of naphthoquinones and anthraquinones by carbonyl-hydroquinone/benzoquinone reactions: A potential route for the origin of 9,10-anthraquinone in tea. *Food Chemistry*, 354, 129530.