Literature Search: 6PPD-Quinone Aquatic Effects, Testing and Treatment Efficacy, and Current Literature

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Contents

Summary of Results

The ultimate goal of this literature search is to keep abreast of current knowledge pertaining to the effects of 6PPD-Quinone (6PPD-Q) on aquatic life along with the development of best practices for the testing and treating of 6PPD-Q. In order to establish current knowledge, a search was conducted across multiple databases which resulted in a vast accumulation of materials. To help focus this results list, sixteen publications of existing research are included that list publications published up to 2024. Therefore, the remaining sixty results are filtered to publications after 2024 and focus on the types of testing to determine the effects on aquatic life and environmental testing and treatment of 6PPD-Q.

Alerts Setup

To remain abreast of this research beyond the publications listed here, alerts can be set on many of the databases searched. Since many of the results overlapped in the databases, three databases were selected for the most pertinent results along with their ability to allow for alerts on searches. The databases and queries are listed below. Once a login is established, saving and alerting the search will enable notifications of any future published research. If needed, further refining of the queries can narrow the alert results.

Recommended databases for alerts on this topic along with the search query linked to the results:

- PubMed
  - Query string: [(6PPD-Q OR 6PPD-quinone) AND fish AND test*]
- EBSCOHost (via OSL)
  - Query string: [(6PPD-Q OR 6PPD-quinone) AND fish AND test*]
- Google Scholar
  - Query string: [(6PPD-Q OR 6PPD-quinone) AND (tire OR auto) AND (fish OR aquatic) AND (testing OR treating)]
  - This one was filtered to 2025 and later due to the larger number of results and duplication of results with the other two search engines.

Annotated Bibliography

The following citations are arranged by three topics: ***Existing Research***, ***Effects on Aquatic Life***, and ***Environment Testing and Treatment***. While some citations may apply to more than one listed topic, it is listed under the topic that appears most prevalent in the abstract. Links and abstracts are listed with the citations; links open to full-text documents, pages that allow PDF downloads, or abstract pages. Articles and papers not openly available in full text can be obtained through interlibrary loans.

Existing Research

The following sixteen citations cover the vast majority of research conducted through 2024.

Aljohani, S., Engels, M. & Wallen, K.E. What research exists on the presence of 6PPD-Q in different environmental media? A systematic map protocol. *Environ Evid, 15*(2). https://doi.org/10.1186/s13750-026-00380-1

Automobiles are ubiquitous in the modern world, and chemicals leaching from car tires and from the tire wear particles produced during driving can be toxic to the environment, particularly in aquatic ecosystems. 6PPD-Quinone (6PPD-Q), a recently identified tire and tire wear particle leachate, has been identified as highly toxic to coho salmon and other aquatic species. Research on the distribution and impacts of 6PPD-Q in aquatic ecosystems is rapidly developing, while research on 6PPD-Q in other environmental media is just beginning. With research efforts developing on many fronts, there is a need to better map emerging knowledge about this toxin. To do that, we ask the question: "What research exists on the presence of the 6PPD-Q in different environmental media (water (freshwater), soil, sediment, and air, including dust)?" The ultimate purpose of this systematic map is to generate a literature catalog that serves as a searchable database about 6PPD-Q in different environmental media.

Brown, M. L.; Ivy, N.; Gonzalez, M.; Greer, J. B.; Hansen, J. D.; Kolodziej, E.; McIntyre, J. K. Roadway runoff induced acute mortality in juvenile coho salmon during spring storm events. *Environ. Sci. Technol.* *60*(2), 1723– 1732, DOI: 10.1021/acs.est.5c13992

Extensive mortalities of adult coho salmon (Oncorhynchus kisutch), often called "Urban Runoff Mortality Syndrome" (URMS), have been documented during the fall in creeks where water quality has been degraded by roadway runoff. The primary cause of mortality is 6PPD-quinone (6PPDQ; N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine quinone)–an ozone transformation product that forms on all vehicle tires. Laboratory studies have shown that juvenile coho salmon are highly sensitive to 6PPDQ exposure. Unlike adults, juveniles reside in impacted watersheds year-round, including during the spring when 6PPDQ concentrations can frequently exceed lethal thresholds during storms. To assess the potential incidence of URMS in springtime rearing habitats for juvenile coho salmon, we conducted a paired water quality and toxicology study at Miller Creek, a runoff-impacted watershed in Normandy Park, WA, USA. Using a small field facility, three naïve groups of juvenile coho salmon (N = 720) were exposed to either creek water or groundwater (N = 120 per treatment per storm), across three spring storms while comparing water quality and mortality end points. In creek water during exposures, peak 6PPDQ concentrations reached 73–110 ng/L, exceeding reported median lethal concentrations (LC50) for coho salmon. Over each 24–73 h storm exposure period, ∼80% of Miller Creek-exposed juvenile salmon died. No mortality occurred among control fish exposed to groundwater. These results indicate previously unidentified mortality risks for juvenile life stages of coho salmon during spring storms, suggesting substantial and year-round water quality impediments to coho salmon health and recovery across roadway runoff-impacted spawning, rearing, and migratory habitats.

Chaoju, L., et al. (2025). Residues of 6PPD-Q in the Aquatic Environment and Toxicity to Aquatic Organisms: A Review. *Fishes 10*(4). DOI: 10.3390/fishes10040146

N-(1,3-dimethylbutyl)-N′-phenyl-p-benzoquinone (6PPD-Q) is an emerging environmental contaminant that is widely distributed in aquatic environments and presents significant toxicological risks to aquatic organisms. As 6PPD-Q is primarily derived from oxidative transformation of the tire antioxidant N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD), its persistence and potential for bioaccumulation in aquatic organisms have raised

widespread concerns. This study reviews the environmental sources, spatial distribution, migration, and transformation behaviors of 6PPD-Q, as well as its degradation mechanisms in different environmental media. Additionally, this review systematically explores the toxicological effects of 6PPD-Q on aquatic organisms, including its physiological, biochemical, and molecular impacts on fish, crustaceans, mollusks, and algae, with a focus on potential toxicological mechanisms. Finally, we discuss the limitations of current research on 6PPD-Q and propose key directions for future studies, including long-term ecological risk assessments, mechanisms of bioaccumulation, metabolic pathway analysis, and optimization of pollution control strategies, aiming to provide a scientific basis for the ecological risk assessment and pollution management of 6PPD-Q.

Cocozza, A. (2024). Analysis of 6PPD-Q & Alternative PPD-Qs in Fish Using QuEChERS. *LCGC International*, *1*(9), 46. https://link-gale-com.slo.idm.oclc.org/apps/doc/A814422428/ITBC?u=sale38182&sid=ebsco&xid=9d1dbe05

This application outlines a QuEChERS extraction of a subset of PPD-Qs in salmon, combined with push-thru cartridge clean-up using UCT's Quick QuEChERS[R], C18, and LipiFiltr[R] in series to achieve the best level of sensitivity in the fatty matrix. The extracts are analyzed on UCT's SelectraCore[R] C18 HPLC column using LC-MS/MS.

González-Vázquez, M. A., et al. (2025). Organic pollutants leaching from tire waste: Ecotoxicity implications for aquatic species. *Aquatic Toxicology, 289*, 107613. https://doi.org/10.1016/j.aquatox.2025.107613

Tire-derived chemicals are emerging contaminants of environmental concern due to their continuous release from tire wear particles and their widespread occurrence in surface waters. This review synthesizes current knowledge on the environmental fate and ecotoxicological effects of key tire-associated compounds, with a focus on organic pollutants like N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine 6PPD, 6PPD-quinone (6PPD-Q), 1,3-diphenylguanidine (DPG), hexamethoxymethylmelamine (HMMM), and methyl isobutyl ketone (MIBK). These substances differ markedly in their physicochemical properties, transformation pathways, and toxicological profiles. A detailed literature review was conducted to identify peer-reviewed studies and safety data sheets addressing the occurrence, persistence, and biological impacts of these compounds across aquatic taxa. The analysis revealed that 6PPD-Q, a transformation product of the antioxidant 6PPD, is acutely toxic to sensitive fish species such as coho salmon (Oncorhynchus kisutch) at nanogram-per-liter concentrations, raising immediate ecotoxicological concerns. In contrast, DPG and HMMM show lower acute toxicity but are associated with sub-lethal effects under chronic exposure scenarios, including developmental and behavioral alterations. MIBK, though less persistent and more volatile, remains relevant due to its episodic presence in roadway runoff. The review highlights consistent patterns of species-specific sensitivity, with fish being particularly vulnerable, while significant data gaps persist for amphibians and for mixture toxicity. Emerging evidence of behavioral and developmental impacts underscores the potential for population-level consequences. The aim of this synthesis is to inform future research priorities and support the development of risk assessment frameworks and regulatory strategies to address the ecological risks posed by tire-derived contaminants in aquatic environments.

Interstate Technology Regulatory Council. (2026 January). *6PPD & 6PPD-quinone*. Retrieved April 24, 2026, from https://6ppd.itrcweb.org/.

> In the short time since 6PPD-quinone (6PPD-q) was isolated and characterized, scientists have been working to understand its prevalence and behaviors in the environment. This website provides in-depth guidance of the current understanding of 6PPD-q sources, exposure, fate, transport, toxicity, and mitigation strategies. While the intent of this document is to present the most salient and recently available information on 6PPD and 6PPD-q, interested readers are encouraged to search the scientific literature for newly available information.

Li, X., Zhou, S., Zhang, T. *et al.* Occurrence and environmental fate/behaviors of tire wear particles and their human and ecological health: an emerging global issue. *Arch Toxicol, 99*, 4353–4366. https://doi.org/10.1007/s00204-025-04147-4

> Rapid societal and urban development has driven a surge in tire production, generating tire wear particles (TWPs), a pervasive environmental pollutant. Upon environmental release, TWPs interact with physio-chemical factors, altering their fate and amplifying toxicity. Available evidence indicates that TWPs mainly enter the organism body through inhalation and dermal contact. Toxicokinetic studies reveal that TWPs and additives penetrate biological barriers, accumulating in the liver and metabolizing via P450 enzymes, producing detoxified compounds or more toxic derivatives like N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone(6PPD-q). Currently, the adverse ecological and health effects have gained increasing public concerns. A series of advances have been achieved to address their ecological and human risks across aquatic, terrestrial, and mammal models, even at population level. Their toxicological effects span across respiratory diseases, metabolic disorders, and reproductive toxicology. Considering the challenges arising from the large-scale production and mismanagement of TWPs, this review synthesizes the latest knowledge on their generation, environmental fate, toxicokinetic, and ecological and human health effects. However, critical gaps remain in long-term low-dose effects, cross-generational toxicity, and biomarker validation. This review highlights the urgent need for global regulatory frameworks and interdisciplinary strategies to mitigate the cascading ecological and health impacts originated from TWPs.

Nesseth, D. (2024). Canadian researchers take on 6PPD-quinone. *Environmental Science & Engineering, 37*(1): 56-58.

> The article focuses on Canadian researchers efforts to understand and mitigate the environmental impact of 6PPD-quinone, a toxic roadway runoff chemical associated with coho salmon die-offs. It discusses ongoing studies, monitoring initiatives, and mitigation strategies aimed at addressing the presence of 6PPD-quinone in aquatic environments and protecting vulnerable fish populations.

Seabrook, K.J., Adams, J.E., Robinson, S.A. *et al.* A systematic map protocol to identify evidence for the environmental occurrence and toxic effects of the tire additive 6PPD. *Environ Evid* (2026). https://doi.org/10.1186/s13750-026-00383-y

The tire additive N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD) is widely produced in large volumes as a rubber antidegradant. This chemical can be released into the environment throughout the lifecycle of rubber products. Recently, 6PPD has become the subject of regulatory interest in some jurisdictions due to its widespread environmental occurrence and the acute toxicity of its transformation product N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPDQ) to some salmonids. As research advances for these emerging contaminants, it is critical to understand whether 6PPD and 6PPDQ concentrations in the environment are high enough to pose a risk to living organisms. Here, we present a protocol to conduct two linked systematic evidence maps related to (1) the occurrence of 6PPD and 6PPDQ in the environment and (2) the effects of 6PPD and 6PPDQ on living organisms. Our objective is to collate information on quantification methods, occurrence data, studied species, and toxicity endpoints. This work will contribute to synthesizing a rapidly expanding body of literature and providing insight into knowledge gaps to direct future work.

Sivalingam, S., et al. (2026). Comprehensive review on environmental pollution caused by 6PPD-quinone and remediation strategies. *RSC Advances, 16*(2): 1943-1955. https://doi.org/10.1039/D5RA06263B

Recently the research concern, the environmental toxicity associated with 6PPD (N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine) and its by-products due to tyre wear. In the tyre manufacturing process, 6PPD is added to increase the durability of the tyre. 6PPD-Q (N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone) due to tyre wear and is emitted along with road particles into the air, resulting in a severe impact on the environment. It has been found that 6PPD increases the mortality rate of silver salmon, an aquatic fauna having rich omega-3 fatty acid content and many essential nutrients. Further, this 6PPD-quinone affects higher organisms by entering the aquatic food chain. Hence, this bioaccumulation severely affects the top predators, including humans. The scientific and regulatory bodies are working hard to find a safer alternative to 6PPD to eradicate the environmental pollution due to 6PPD-Q, a degradation product of 6PPD. The current review article addresses the various approaches to reducing 6PPD release in the environment, removing the existing 6PPD from the surrounding environment, and finding a safer alternative to 6PPD to increase the tyre lifetime. Remediation strategies involve potential substitutes for 6PPD and 6PPD-Q, including alternative PPDs such as IPPD, DPPD, 7PPD, and CCPD, as well as non-PPD options like specialized graphene, octyl gallate, lignin, and nano-calcium carbonate modified with gallic acid. These candidates offer protection against ozone, oxidation, and wear while maintaining tire performance and safety. In addition, the review provides details on potential alternatives and the mechanisms through which they protect the environment. The existing knowledge gaps and directing researchers in establishing research in the various fields to protect against this kind of pollution.

Somepalli, K. and G. Andaluri (2025). Transformation pathways, detection, removal, and sustainable alternatives of 6PPD and its quinone derivative (6PPDQ): A comprehensive review. *Emerging Contaminants, 11*(3). DOI:10.1016/j.emcon.2025.100547

N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD) is a widely used antioxidant and antiozonant in vehicle tires, commonly used to enhance rubber durability and performance.

However, its environmental transformation, particularly into 6PPD-Quinone (6PPDQ) through oxidative processes like ozonation, has emerged as a serious concern due to the acute toxicity in aquatic systems. 6PPDQ has been linked to significant mortality in sensitive fish species, including coho salmon, at 41 ng/L concentrations. This review synthesizes recent advances in the understanding of 6PPD degradation pathways, including ozonation, photodegradation, hydrolysis, microbial metabolism, and atmospheric reactions. This paper systematically examines the formation and characterization of various TPs and outlines the methods used for their detection across water, air, soil, sediments, and biota. It also evaluates the efficiency of treatment approaches such as advanced oxidation processes (AOPs), microbial degradation, adsorption, and membrane technologies for the removal of 6PPDQ from contaminated media. Additionally, emerging research on bio-based, synthetic, and engineered antioxidant alternatives to 6PPD is reviewed, with attention to their environmental compatibility and industrial feasibility. The paper concludes with a comprehensive outline of future research priorities focused on improving analytical detection, understanding long-term environmental fate and toxicity, optimizing treatment technologies, and guiding the development of safer alternatives. Collectively, this review provides a foundation for environmental risk assessment, regulatory policy development, and sustainable innovation in rubber manufacturing.

Transportation, C. D. o. (2024). Literature Review: 6PPD-Quinone Technical Information Relevant to Caltrans Stormwater Management Program C. D. o. Transportation. https://dot.ca.gov/-/media/dot-media/programs/environmental-analysis/documents/env/6ppd-q-literature-review-memo_a11y.pdf

This draft technical memorandum provides an overview of scientific literature of 6PPD-Q sources, transport, treatability, toxicity, and related information needed to guide monitoring design and evaluate stormwater treatment design guidance updates.

Transportation, C. D. o. (2024). Summary of Published Science, Caltrans Monitoring Results, and Treatment Approaches Related to 6PPD-Q in Stormwater. C. D. o. Transportation. https://dot.ca.gov/-/media/dot-media/programs/environmental-analysis/documents/env/ctws-6ppd-q-wp-a11y.pdf

This final technical memorandum provides an overview of scientific literature of 6PPD-Q sources, transport, treatability, toxicity, and related information needed to guide monitoring design and evaluate stormwater treatment design guidance updates.

Yin, T., et al. (2025). Environmental Occurrence, Influencing Factors, and Toxic Effects of 6PPD-Q. *Toxics, 13*(11): 906. https://doi.org/10.3390/toxics13110906

The antioxidant *N*-(1,3-dimethylbutyl)-*N*′-phenyl-*p*-phenylenediamine (6PPD) is widely incorporated into tires to extend their service life. However, in the presence of ozone, it is readily transformed into *N*-(1,3-dimethylbutyl)-*N*′-phenyl-*p*-benzoquinone (6PPD-Q). Owing to the large-scale production and widespread utilization of rubber-related products, 6PPD-Q is continuously released into the environment with tire and road wear particles, becoming ubiquitous across multiple environmental compartments. It possesses bioaccumulation potential and exhibits significant toxicity, while multiple exposure pathways enable it to enter human body, posing risks to public health. This review summarizes the environmental distribution of 6PPD-Q in

atmospheric, aquatic, and terrestrial systems, and examines key factors influencing its occurrence, including precipitation patterns, traffic characteristics, sunlight, and particle size. The toxicological effects of 6PPD-Q are also discussed. Based on these findings, a comprehensive management framework encompassing "source reduction—process regulation—end-of-pipe treatment" is proposed. Finally, current knowledge gaps are identified and future research directions are highlighted.

Zhang, S., et al. (2025). Environmental and Human Health Risks of 6PPD and 6PPDQ: Assessment and Implications. *Toxics*, *13*(10): 873. https://doi.org/10.3390/toxics13100873

This review aims to synthesize current knowledge on the environmental contaminants N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD) and its quinone derivative (6PPDQ) derived from tire wear particles (TWPs), focusing on their environmental distribution, transformation, human exposure pathways, toxicological effects, and health risks to ecological and human health. A comprehensive literature review was conducted, compiling and analyzing data from environmental monitoring studies, toxicological assessments on aquatic and mammalian models, and emerging human biomonitoring research. Key findings on concentrations, toxicological endpoints (e.g., LC50, oxidative stress, genotoxicity), and exposure pathways were evaluated. 6PPD and its transformation product 6PPDQ are ubiquitous environmental pollutants found in air, water, soil, sediment, and dust. 6PPDQ is notably highly toxic to aquatic organisms, with an acute LC50 of 790 ng/L for coho salmon. Human exposure to these compounds occurs through inhalation, ingestion, and dermal contact, and their presence has been confirmed in human matrices including blood, urine, and cerebrospinal fluid. Toxicological studies, primarily on model organisms, indicate that 6PPD and 6PPDQ can induce oxidative stress, cause DNA damage, and disrupt metabolic and neurological functions. Adverse outcomes such as intestinal toxicity, reproductive impairment, neurobehavioral changes, and potential carcinogenicity have been observed. However, direct evidence of their health impacts on humans remains limited. 6PPD and 6PPDQ pose significant and widespread ecological risks, with 6PPDQ representing a particularly potent aquatic toxicant. While human exposure is confirmed, the full scope of human health implications is not yet well understood. The review highlights the need for longitudinal environmental tracking, mechanistic studies, and refined exposure models to inform regulatory actions and mitigate risks. Addressing these challenges is essential to mitigate the ecological and health burdens posed by 6PPD and 6PPDQ. This study underscores the global societal importance of addressing 6PPD-related pollution—a pervasive and transboundary environmental challenge stemming from universal tire wear.

Zhang, X., et al. (2026). Ecotoxicity of 6PPD and 6PPD-Q in aquatic ecosystems: Mechanisms, influencing factors, and mitigation strategies. *Journal of Hazardous Materials, 501*: 140856. https://doi.org/10.1016/j.jhazmat.2025.140856

N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD) and its oxidation product 6PPD-quinone (6PPD-Q) are widely present in the environment due to their extensive use in rubber products, especially tires. Once released, 6PPD undergoes transformation to 6PPD-Q, which has been detected in air, dust, sediments, surface waters, and wastewater. Stormwater runoff and snowmelt act as key delivery

pathways, mobilizing a large fraction of 6PPD and 6PPD-Q into aquatic systems. Concentrations reported in surface waters overlap with biological effect thresholds, and 6PPD-Q has been identified as the proximate toxicant responsible for acute mortality in coho salmon (Oncorhynchus kisutch). Across multiple taxa (e.g. Chlorella vulgaris, Ceratophyllum demersum L., the rotifer Brachionus calyciflorus and zebrafish), exposure to 6PPD and 6PPD-Q is linked to oxidative stress and subsequent cardiotoxic, neurotoxic, intestinal, and developmental effects, modulated by species, life stage, dose, and co-exposures. This review integrates current knowledge along the full chain from environmental sources and cross-media transport to occurrence, toxic mechanisms, influencing factors, and risk. This review establishes a unified framework linking environmental sources, cross-media transport, occurrence patterns, toxic mechanisms, key modifying factors, and mitigation strategies for 6PPD and 6PPD-Q. It also highlights critical knowledge gaps in transformation kinetics and species-specific metabolic capacity that are essential for ecological risk assessment and future regulatory interventions.

Effects on Aquatic Life

The following thirty citations focus on specific types of testing on aquatic life or the results of aquatic life testing beyond basic toxicity levels. Results relating to more general toxicity testing of aquatic life have been removed but can be replaced upon request.

Ankley, P. J., et al. (2024). Biotransformation of 6PPD-quinone In Vitro Using RTL-W1 Cell Line. *Environmental Science & Technology Letters, 11*(7): 687-693. DOI: 10.1021/acs.estlett.4c00342

> Urban stormwater runoff contains the tire-derived transformation product N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-quinone), which poses significant environmental risks due to its high toxicity toward certain salmonids. 6PPD-quinone biotransformation has been investigated to explain some of the stark interspecies differences in sensitivity across different fishes; however, the primary mechanisms of 6PPD-quinone biotransformation remain unclear. This work aimed to explore the toxicokinetics of 6PPD-quinone in immortalized rainbow trout (Oncorhynchus mykiss) liver cells (RTL-W1) to identify transformation products, using coexposure with different enzyme inhibitors and inducers. Using high-resolution mass spectrometry, we identified three phase I 6PPD-quinone transformation products, with phenyl ring hydroxylation dominating, followed by hydroxylation of the alkyl side chain, and an unknown transformation product after 4 h of exposure. Co-exposing RTL-W1 cells with α-naphthoflavone and quercetin greatly inhibited the biotransformation of 6PPD-quinone, revealing that CYP1A is primarily involved in phase I biotransformation. Hepatic clearance predicted from in vitro results was further verified based on isolated perfused trout liver experiments. Further studies are necessary on the biotransformation and kinetics of 6PPD-quinone and the detoxification pathways involved in a wide phylogenetic space in fishes.

Ankley, P. J., et al. (2025). The Xenometabolome of Early-Life Stage Salmonids Exposed to 6PPD-Quinone. *Environmental Science & Technology***,** *59*(28): 14214-14225. https://doi.org/10.1021/acs.est.5c01442

> N-(1,3-Dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-Q) is a ubiquitous transformation product (TP) derived from the rubber tire antioxidant N-(1,3-dimethylbutyl)-N′-

phenyl-p-phenylenediamine (6PPD) and is acutely toxic to certain species of Salmonidae. Not all salmonids are sensitive to acute lethality caused by 6PPD-Q, with 6PPD-Q potency varying by several orders of magnitude among teleosts. The main driver(s) of species sensitivity differences is (are) a pressing question, with one area of interest examining whether differences in teleosts' ability to biotransform and detoxify 6PPD-Q could be a key factor. This study utilized liquid-chromatography high-resolution mass spectrometry (LC-HRMS) to assess biotransformation and metabolome-wide effects of 6PPD-Q on early life stage salmonids, including two sensitive species, rainbow trout (Oncorhynchus mykiss) and lake trout (Salvelinus namaycush), and one tolerant species, brown trout (Salmo trutta). Three phase I TPs and seven phase II TPs were detected, with differences in peak area ratios revealing that brown trout had the greatest ability to conjugate phase I TPs. Monohydroxylated TPs were verified using codeveloped analytical standards that will be of use for future biomonitoring and exposure assessment. Several endogenous metabolites were found to be dysregulated in rainbow and lake trout, indicative of mitochondrial dysfunction, altered metabolism, and disrupted membrane permeability. Results of this study indicate a potential difference in the biotransformation capability of 6PPD-Q among Salmonidae fish species, detection of a unique phase I TP in sensitive Salmonidae species, and subsequent unique metabolome responses.

Chae, Y., et al. (2026). Adverse effects of 6PPD-quinone bioaccumulation at environmentally relevant concentrations on Cyprinus carpio growth and development. *Sci Rep*, *16*(1): 6289. https://doi.org/10.1038/s41598-026-36900-9

With industrial advancements, the development and use of various additive substances have increased, leading to their potential release into the environment during use and disposal. As a primary tyre additive, the rubber additive, N(1)-(4-methylpentan-2-yl)-N(4)-phenylbenzene-1,4-diamine (6PPD) has recently become an environmental issue. Its oxidation by-product, 6PPD-quinone (6PPD-Q), which is released from tyre wear, has drawn considerable attention because of its potent toxicity to fish in aquatic environments, particularly to coho salmon. In this study, we aimed to determine whether 6PPD-Q is potentially toxic to non-salmonid fish species within aquatic ecosystems, using carp (Cyprinus carpio) as a representative model organism for toxicity assessment. We analysed the proteomic changes in carp following 8 weeks of chronic exposure to 6PPD-Q, and the effects were further evaluated using gene-based analyses. 6PPD-Q disrupts cytoskeleton-related pathways and induces oxidative stress in carp. This can potentially impair the antioxidant defence system and suppress gene expression related to growth and metabolism, contributing to a decline in health. These findings suggest that 6PPD-Q poses a serious threat to the growth and survival of aquatic organisms. Considering the escalating global tyre consumption and the resultant environmental runoff of 6PPD and 6PPD-Q, ongoing research into the ecotoxicology of these additives, along with the development of safer alternatives, is strongly warranted. SUPPLEMENTARY INFORMATION: The online version contains supplementary material available at 10.1038/s41598-026-36900-9.

Ding, Y. and W.-X. Wang (2026). Tissue-specific bioaccumulation and hepatotoxicity of 6PPD and 6PPD-Quinone in zebrafish. *Environmental Chemistry and Ecotoxicology, 8*: 307-318. DOI:

10.1016/j.enceco.2025.11.029

Tire additive 6PPD and its transformation product 6PPD-Quinone have garnered substantial attention due to their association with mass salmon mortality events. This study investigated the bioaccumulation and sublethal toxicological effects of 6PPD and 6PPD-Q using zebrafish (Danio rerio) as a model fish. Both 6PPD and 6PPD-Q exhibited distinct distribution and bioaccumulation potential in the fish. Specifically, 6PPD was enriched in the guts and liver, whereas 6PPD-Q was enriched in the brain, liver, and gills. Zebrafish liver exhibited strong absorption capacity for both compounds, while the eyes and gills showed selective absorption for 6PPD and 6PPD-Q, respectively. At elevated exposure concentrations, 6PPD demonstrated lower bioaccumulation potential but higher adsorption capacity. In contrast, 6PPD-Q displayed the opposite pattern. This suggested that depuration processes predominantly regulated the accumulation dynamics of these compounds. Exposure to both chemicals at concentrations ranging from environmentally relevant to high concentrations induced multi-levels of toxic responses in zebrafish. These included behavioral impairments with reduced swimming activity and histopathological damages of inflammation, fat droplets, vacuoles, and cell gap enlargement in liver tissue. Transcriptomic analysis revealed that both compounds induced pathological liver damage in zebrafish through disruption of glycolysis and gluconeogenesis pathways. Additionally, effects on P450 metabolic systems led to differential bioaccumulation patterns between the two compounds. This study provides important toxicological evidence for assessing the ecological risks of tire-derived pollutants and emphasizes the necessity of monitoring both parent compounds and their transformation products in environmental surveillance.

Feng, Y., et al. (2025). Multi-omics insights into 6PPD- and 6PPDQ-induced gut-liver axis disruption and non-alcoholic fatty liver disease progression in zebrafish (Danio rerio). *Journal of Hazardous Materials, 495*: 138822. https://doi.org/10.1016/j.jhazmat.2025.138822

The pervasive environmental presence of N-(1,3-Dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD) and its transformation product, 6PPD-quinone (6PPDQ), has raised concerns about their potential toxicity, yet their interactions with the gut microbiota at environmentally relevant concentrations remain poorly understood. Here, we investigated the effects of 6PPD and 6PPDQ on the gut-liver axis in zebrafish (Danio rerio). Zebrafish larvae exposed to 0.01, 1, and 100 μg/L of 6PPD or 6PPDQ for five days exhibited intestinal and hepatic developmental toxicity, including hepatic lipid accumulation and hepatomegaly. Adult zebrafish exposed for 21 days displayed compromised intestinal barrier integrity, gut dysbiosis, and lipidomic disturbances in the liver. Statistical analysis using the multi-response permutation procedure confirmed significant shifts in gut microbial community structure. Dysbiosis was characterized by reduced beneficial bacteria and an increase in pathogenic taxa, accompanied by elevated circulating lipopolysaccharide (LPS) and upregulated hepatic expression of lbp (LPS-binding receptor). Hepatic lipid accumulation resulted from increased triglyceride (TG) and total cholesterol synthesis, with lipidomics revealing distinct disruptions: 6PPD impaired phosphatidylinositol phosphate synthesis, while 6PPDQ affected TG homeostasis. Correlation analysis linked gut microbial shifts to hepatic lipid dysregulation. These findings suggest that 6PPD and 6PPDQ exposure disrupts gut-liver axis

homeostasis, potentially driving non-alcoholic fatty liver disease development. This study underscores the need to integrate gut-liver-microbiota endpoints into environmental risk assessments for aquatic organisms.

Gamil, M. R., et al. (2025). Toxicity of 6PPD-quinone in European seabass (Dicentrarchus labrax) under baseline and Vibrio alginolyticus challenge conditions: Protective insights from astaxanthin mitigation. *Science of The Total Environment, 987*: 179821. https://doi.org/10.1016/j.scitotenv.2025.179821

6PPD-quinone, a widespread pollutant from tire wear, exhibits species-specific toxicity in aquatic organisms. This study examines the effects of 6PPD-quinone on European seabass (Dicentrarchus labrax) at an environmentally relevant concentration (2.85 µg/L, static renewal for five days) under both unchallenged conditions and bacterial challenge with Vibrio alginolyticus. The study also evaluates the hepatoprotective and immunomodulatory potential of astaxanthin supplementation (150 mg/kg diet for 30 days), derived from non-GMO Haematococcus pluvialis microalgae. While 6PPD-quinone exposure (up to 250 µg/L for 72 h) did not cause mortality, it induced neurological symptoms, including erratic swimming and respiratory distress. At 2.85 µg/L, biochemical analyses showed increased albumin, altered creatine kinase and blood urea nitrogen levels, and disrupted glucose homeostasis. Significant hepatic and gill lesions, such as diffuse hepatic necrosis and lamellar fusion, were observed. Astaxanthin mitigated liver necrosis, reduced inflammation, restored albumin/globulin ratios, and maintained glucose and triglyceride homeostasis. It improved gill integrity, reduced blood urea nitrogen, and enhanced lysozyme activity while preventing excessive immune activation. 6PPD-quinone disrupted immune gene expression in the head-kidney and spleen, which astaxanthin modulated. This study highlights 6PPD-quinone's physiological impact on seabass and astaxanthin's potential in mitigating pollutant toxicity, emphasizing that balanced immune responses are essential for maintaining health under environmental stressors.

Gao, Y., et al. (2025). Route-dependent toxicodynamics of 6PPD-quinone in mussels: Mechanical resilience trades off with subcellular injury and metabolic disruption. *Environmental Pollution, 385*: 127152. https://doi.org/10.1016/j.envpol.2025.127152

The byssal thread, a mussel-secreted proteinaceous anchor critical for underwater adhesion, represents a vital adaptation for survival in dynamic marine environments but faces vulnerability to pollutants. This study examines how the tire-derived contaminant 6PPD-quinone (6PPD-Q) impacts the byssal defense system of Mytilus coruscus via waterborne and dietary exposure. Experiments evaluated byssal production, mechanical traits, foot histology, and transcriptomic profiles. Waterborne 6PPD-Q induced a paradoxical enhancement: increased thread count/diameter and adhesion strength coexisted with progressive foot tissue damage, evidenced by histopathology and dysregulated ribosomal/DNA repair pathways. Dietary exposure, conversely, disrupted nutrient metabolism and immune responses, with transcriptomes diverging sharply from waterborne cases. KEGG analysis revealed route-specific toxicity: waterborne exposure activated nuclear DNA damage pathways, while dietary exposure triggered lysosomal/antigen-processing mechanisms. Solvent controls confirmed 6PPD-Q specificity. These findings unveil a dual paradox where 6PPD-Q simultaneously enhances mechanical resilience and

inflicts subcellular harm, with toxicodynamics governed by exposure route. The trade-off between structural fortification and physiological impairment highlights complex pollutant interactions in mussels, emphasizing the need for exposure pathway-specific assessments in managing aquaculture sustainability amid coastal contamination. This work advances understanding of anthropogenic pollutant impacts on marine bivalve adaptive strategies and ecosystem health.

Harris, F. R., et al. (2025). Phenotypic Profiling of 6PPD, 6PPD-Quinone, and Structurally Diverse Antiozonants in RTgill-W1 Cells Using the Cell Painting Assay. *Environmental Science & Technology Letters, 12*(6): 695-701. https://pubs.acs.org/doi/10.1021/acs.estlett.5c00327#

6PPD-quinone, a degradation product of the rubber antiozonant 6PPD that is frequently added to tires, has previously been identified as a causative agent of urban runoff mortality syndrome in salmonids. Previous high-throughput phenotypic profiling (HTPP) studies using the Cell Painting assay in the RTgill-W1 rainbow trout cell line have demonstrated that 6PPD-quinone toxicity occurs at much lower concentrations than the 6PPD parent molecule, which is consistent with available in vivo toxicity data in rainbow trout. Current research efforts include identifying alternative antiozonant compounds to potentially replace 6PPD in tire manufacturing. To fill bioactivity data gaps for potential 6PPD alternatives, 18 compounds including other substituted p-phenylenediamines (PPD) and PPD-quinones were assayed using HTPP in RTgill-W1 cells. 7PPD-quinone and 77PD-quinone produced changes in cellular phenotype similar to those of 6PPD-quinone at comparable concentrations. IPPD-quinone produced changes in cellular phenotype at higher concentrations than 6PPD-quinone, with a phenotypic profile that was most similar to its parent molecule IPPD. These findings suggest that 7PPD-quinone and 77PD-quinone may exhibit similar effects in rainbow trout and potentially other 6PPD-quinone sensitive salmonids. In contrast, IPPD may be less toxic to salmonids than 6PPD, given the relative lack of bioactivity of IPPD-quinone compared to 6PPD-quinone.

Huang, Z., et al. (2024). Protective role of ghrelin against 6PPD-quinone-induced neurotoxicity in zebrafish larvae (Danio rerio) via the GHSR pathway. *Ecotoxicology & Environmental Safety, 285*: 117031. https://doi.org/10.1016/j.ecoenv.2024.117031

The toxicity mechanisms of N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine quinone (6PPD-Q), an antioxidant derivative of 6PPD via ozone reaction commonly used in rubber and tire industries, were investigated in zebrafish larvae with concentrations ranging from 0 to 50 μg/L. Despite normal hatchability, 6PPD-Q exposure led to reduced body length and swimming distance in 120 hours post-fertilization (hpf) larvae. At the highest concentration (50 μg/L), 6PPD-Q significantly impaired dopaminergic neuron development and neurotransmitter levels, including dopamine, 5-hydroxytryptamine, and glutamate. Transcriptome profiling unveiled perturbations in growth and developmental gene expression, such as upregulation of runx2a , runx2b , and ghrl (ghrelin and obestatin prepropeptide), and downregulation of stat1b, auto1, and cidea. Notably, anamorelin, a growth hormone secretagogue receptor (GHSR) agonist, recovered the behavioral deficits induced by 6PPD-Q, implying a neuroprotective role of ghrelin possibly mediated via the ghrelin/GHSR pathway. Collectively, our findings indicate that ghrelin upregulation may counteract 6PPD-Q toxicity in zebrafish larvae, shedding light on potential

therapeutic avenues for mitigating the adverse effects of this antioxidant byproduct.

Jackson, M., et al. (2026). Metabolic disruption in salmonids following co-exposure to road runoff contaminants 6PPD-Quinone and 9,10-anthraquinone. *Science of The Total Environment, 1011*: 181172. https://doi.org/10.1016/j.scitotenv.2025.181172

Components of road runoff have been correlated to adverse health outcomes in fish and aquatic invertebrates residing in urban water bodies. Recently, a transformation product of the antioxidant chemical added to car tires, N-(1,3-Dimethylbutyl)-N'-phenyl-p-phenylenediamine (6PPD-quinone, 6PPD-q), was found to induce urban stormwater mortality syndrome (URMS) in salmonids. Exposure to 6PPD-q has the potential to increase the sensitivity of salmonids to other co-occurring contaminants commonly detected in surface waters, such as the polycyclic aromatic hydrocarbon 9,10-anthraquinone (AQ). To investigate the mechanisms of 6PPD-q toxicity, fish were exposed to established sublethal concentrations of 6PPD-q and AQ, both separately and in combination, over a five-day period. Non-targeted MS-based metabolomics analysis was conducted on liver samples. Using in-house and open-source spectral libraries, 260 metabolites were confidently annotated in liver tissues. This study provides mechanistic insights into 6PPD-q and AQ toxicity in salmonids, highlighting oxidative stress, mitochondrial dysfunction, and impaired detoxification as key drivers of sublethal effects. Findings also suggest that AQ may mitigate 6PPD-q toxicity by enhancing antioxidant defenses and xenobiotic metabolism. Additionally, quantitative RNA sequencing of liver tissues revealed species-specific transcriptomic responses to chemical exposures, with coho salmon exhibiting more pronounced differential gene expression relative to chinook and rainbow trout. Transcriptomic analysis identified key modulation of genes involved in apoptosis, immune response, vascular permeability, and energy metabolism, deepening mechanistic understanding of 6PPD-q toxicity.

Jankowski, M. D., et al. (2025). Bioactivity of the ubiquitous tire preservative 6PPD and degradant, 6PPD-quinone in fish- and mammalian-based assays. *Toxicol Sci, 204*(2): 198-217. https://doi.org/10.1093/toxsci/kfaf008

6PPD-quinone (N-(1,3-dimethylbutyl)-N'-phenyl-p-phenylenediamine quinone), a transformation product of the antiozonant 6PPD (N-(1,3-dimethylbutyl)-N'-phenyl-p-phenylenediamine) is a likely causative agent of coho salmon (Oncorhynchus kisutch) pre-spawn mortality. Stormwater runoff transports 6PPD-quinone into freshwater streams, rapidly leading to neurobehavioral, respiratory distress, and rapid mortality in laboratory-exposed coho salmon, but causing no mortality in many laboratory-tested species. Given this identified hazard, and potential for environmental exposure, we evaluated a set of U.S. Environmental Protection Agency's high-throughput assays for their capability to detect the large potency difference between 6PPD and 6PPD-quinone observed in coho salmon and screen for bioactivities of concern. Assays included transcriptomics in larval fathead minnow (FHM), developmental and behavioral toxicity in larval zebrafish, phenotypic profiling in a rainbow trout gill cell line, acute and developmental neurotoxicity in mammalian cells, and reporter transcription factor activity in HepG2 cells. 6PPD was more consistently bioactive across assays, with distinct activity in the developmental neurotoxicity assay (mean 50th centile activity concentration = 0.91 μM). Although 6PPD-quinone

was less potent in FHM and zebrafish, and displayed minimal neurotoxic activity in mammalian cells, it was highly potent in altering organelle morphology in RTgill-W1 cells (phenotype-altering concentration = 0.024 μM compared with 0.96 μM for 6PPD). Although in vitro sensitivity of RTgill-W1 cells may not be as sensitive as intact Coho salmon, the assay may be a promising approach to test chemicals for 6PPD-quinone-like activities. The other assays each identified unique bioactivities of 6PPD, with neurobehavioral and developmental neurotoxicity being most affected, indicating a need for further assessment of this chemical. Our results demonstrate that the common tire additive, 6PPD, is bioactive in a broader set of assays than the environmental transformation product 6PPD-quinone and that it may be a developmental neurotoxicant in mammals, whereas 6PPD-quinone was much more potent than 6PPD in altering the intracellular phenotype of rainbow trout gill cells. Application of the set of high-throughput and high-content bioassays to test the bioactivity of this emerging pollutant has provided data to inform both ecological and human health assessments.

Jiao, F., et al. (2025). Chronic toxicity mechanisms of 6PPD and 6PPD-Quinone in zebrafish. *Environmental Science and Ecotechnology, 25*: 100567. https://doi.org/10.1016/j.ese.2025.100567

N-(1,3-Dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD) and its oxidation derivative, 6PPD-quinone (6PPDQ), have been extensively detected in environmental and biological samples, raising significant concerns regarding their chronic aquatic toxicity at environmentally relevant concentrations. However, the underlying mechanisms driving this chronic toxicity remain largely unexplored. Here we show that zebrafish exposed to 6PPD and 6PPDQ exhibit distinct toxicokinetic profiles, with 6PPD preferentially accumulating in the liver and 6PPDQ predominantly targeting the brain. Exposure to both compounds impaired zebrafish growth, induced hepatic damage, and disrupted locomotor behavior. Transcriptomic analysis of liver tissue revealed disturbances in lipid and carbohydrate metabolic pathways in both treatment groups, with distinct differences in gene expression patterns and biochemical responses between 6PPD and 6PPDQ. Specifically, both compounds downregulated peroxisome proliferator-activated receptor gamma (PPARγ) and elevated the expression of pro-inflammatory cytokines (TNF-α and IL-6). Molecular dynamics simulations and surface plasmon resonance experiments further demonstrated that hepatotoxicity was associated with direct binding of these compounds to PPARγ, a critical regulator of lipid metabolism and inflammation. Our findings highlight the hepatotoxic risks of 6PPD and 6PPDQ to aquatic life. Importantly, 6PPDQ exhibited greater toxicity compared to 6PPD, emphasizing an urgent need for targeted environmental controls and regulatory actions to mitigate ecological harm and potential public health consequences.

Kuo, L.-J., et al. (2025). Analysis of 6PPD-Q in finfish, shellfish, and marine mammal tissues. *Chemosphere, 379*: 144418. https://doi.org/10.1016/j.chemosphere.2025.144418

6PPD-quinone (6PPD-Q), a transformation product of tire rubber anti-oxidant 6PPD, has been identified as the primary causal toxicant for the urban runoff mortality syndrome observed in coho salmon (Oncorhynchus kisutch) in the Pacific Northwest, USA. Several other fish species are also vulnerable to 6PPD-Q. However, monitoring efforts on 6PPD-Q have been focused on water, particulate matter, soils, and sediments, while that in tissues remains scarce. This study presents a

workflow for extraction and quantitative analysis of 6PPD-Q in complex tissues from shellfish, finfish, and marine mammals. A multi-residue extraction protocol was developed for quantitative analysis of 6PPD-Q and persistent organic pollutants (PCBs, PBDEs, organochlorine pesticides) and PAHs in tissues in a single extraction. A GC-MS/MS based 6PPD-Q measurement was also developed. The protocol was evaluated in tissues including fish fillets, whole fish homogenates, mussels, and whale blubber. Limits of quantification of 6PPD-Q were between 0.03 and 0.12 ng/g ww and the surrogate (6PPD-Q-d5) recoveries were ~60–100 % among matrices. We also conducted an initial biomonitoring study using caged mussels (Mytilus trossulus) and juvenile Chinook salmon (Oncorhynchus tshawytscha) from Puget Sound, WA. 6PPD-Q detection rates were at least 50 % but the concentrations were mostly <1 ng/g ww. Our protocol will aid 6PPD-Q biomonitoring in aquatic environments and also exposure assessments for improved understanding of 6PPD-Q bioaccumulation potential in these food webs.

Li, R., et al. (2025). Enantioselectivity in Metabolism and Toxicity of 6PPD-Quinone in Salmonids. *Environmental Science & Technology, 59*(25): 12878-12888. https://pubs.acs.org/doi/10.1021/acs.est.4c12384

The toxicity of *N*-(1,3-dimethylbutyl)-*N*′-phenyl-*p*-phenylenediamine quinone (6PPD-Q) in salmonids has been found to be sensitive to even minor structural changes on its alkyl side chain. Inspired by this, we herein isolated the enantiomers of 6PPD-Q and tested their *in vitro* metabolism and toxicity in rainbow trout (*O. mykiss*) and coho salmon (*O. kisutch*). (*R*)-6PPD-Q was found to be rapidly metabolized in rainbow trout liver S9 with a half-life ($t_{1/2}$) of 11.4 min, which was 2.59 times faster than that of (*S*)-6PPD-Q. Similarly, (*R*)-6PPD-Q was preferentially metabolized in coho salmon liver S9. This was further evidenced by the preferential formation of an (*R*)-aryl–OH-6PPD-Q metabolite. Supporting this, enantioselective accumulation of (*S*)-6PPD-Q was found in rainbow trout *in vivo*. To further distinguish between kinetics and intrinsic toxicity, we tested the toxicity of 6PPD-Q enantiomers in the CSE-119 cell line with a minimal metabolism of 6PPD-Q. (*R*)-6PPD-Q was found to strongly induce cytotoxicity in CSE-119 cells with a median effect concentration (EC_{50}) of 17.7 μg/L, which was 3.94 times stronger than that of (*S*)-6PPD-Q. Likewise, (*R*)-6PPD-Q was also the more toxic enantiomer in RTG-2 cells. In summary, this study reports the enantioselectivity of 6PPD-Q in both toxicity and metabolism.

Liang, L., et al. (2026). Comparative toxicity of 6PPD and 6PPD-quinone on the gut-liver axis in zebrafish: Insights into microbiota dysbiosis and oxidative stress. *Aquatic Toxicology, 295*: 107800. https://doi.org/10.1016/j.aquatox.2026.107800

The ubiquitous presence of the tire rubber antioxidant 6PPD and its toxic transformation product, 6PPD-quinone (6PPDQ), poses emerging risks to aquatic ecosystems. This study evaluated the comparative hepatotoxicity and enterotoxicity of environmentally relevant concentrations of 6PPD and 6PPDQ in adult zebrafish (Danio rerio), with a specific focus on the gut-liver axis. Histopathological analysis revealed that both compounds induced dose-dependent liver damage, characterized by hepatocyte vacuolation and nuclear degeneration; notably, 6PPDQ elicited distinct, and in some aspects comparable, tissue injury relative to the parent compound. Biochemical assays confirmed that oxidative stress is a key mechanism of toxicity, evidenced by

elevated malondialdehyde (MDA) levels (increased by up to 62.2%) and glutathione peroxidase (GSH-Px) activity (increased by up to 257.1%), alongside inhibited superoxide dismutase (SOD) and catalase (CAT) activities (reduced by up to 15.2% and 25.3%, respectively). In the intestine, exposure compromised barrier integrity and triggered inflammation, indicated by villus structural damage and the upregulation of NF-κB p65 expression. 16S rRNA sequencing unveiled distinct dysbiosis patterns: 6PPD exposure reduced the abundance of Firmicutes and enriched Proteobacteria, whereas 6PPDQ significantly altered the abundances of Bacteroidetes and Proteobacteria. Functional prediction analysis indicated hypothetical functional shifts, suggesting that 6PPDQ may specifically impair immune-related pathways while enhancing energy metabolism. Collectively, these findings demonstrate that 6PPD and 6PPDQ induce concurrent multi-organ toxicity via the gut-liver axis, with the transformation product 6PPDQ presenting a distinct ecological risk profile. This study highlights the critical need to incorporate transformation products into the risk assessment of tire-derived contaminants.

Lin, J., et al. (2026). Molecular Toxicity Pathways and Transcriptomic Points of Departure (tPODs) of 6PPD-Quinone in Early-Life Stage Lake Trout (*Salvelinus namaycush*). *Environmental Science & Technology, 60*(15): 11324-11336. https://pubs.acs.org/doi/10.1021/acs.est.5c17198

N -(1,3-Dimethylbutyl)-*N* '-phenyl-p-phenylenediamine-quinone (6PPD-quinone) has been linked to acute mortality in select salmonid species at concentrations found in surface waters after stormwater runoff events. However, little is known about the specific mechanism underlying the highly species-specific sensitivity to 6PPD-quinone, limiting our ability to predict effects across fishes and develop protective guidelines. This study aimed to characterize the molecular toxicity pathways of 6PPD-quinone in lake trout (*Salvelinus namaycush*), a species of significant ecological, cultural, and economic importance in North America that is among the most sensitive fishes to 6PPD-quinone exposure. Whole-transcriptome analysis of alevins exposed to graded 6PPD-quinone concentrations (0.22-7.6 μg/L) for 96 h revealed concentration-dependent dysregulation of pathways related to inflammation, vascular integrity, oxidative stress, protein homeostasis, and skeletal and craniofacial development. Notably, exposed alevins showed early activation of proinflammatory cytokine signaling and programmed cell death processes, including efferocytosis, necroptosis, and ferroptosis. Benchmark concentration (BMC) modeling of the transcriptomic data identified a transcriptomic BMC_{10th} (the concentration affecting the most sensitive 10th percentile of genes) of 0.211 μg/L, closely aligning with the 45-day apical BMC_{10th} and BMC_{20th} values (0.161 and 0.252 μg/L, respectively). These findings highlight that short-term transcriptomic responses in nonregulated embryonic life stages offer mechanistic insights and provide predictive, quantitative benchmarks that closely correspond with traditional apical toxicity thresholds.

Lv, M., et al. (2026). 6PPD impairs liver growth through inflammatory pathways: Insights from zebrafish and human cell models. *Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology, 302*: 110454. https://doi.org/10.1016/j.cbpc.2026.110454

N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD), a widespread tire-derived contaminant, has drawn increasing concern for its environmental persistence and toxicity.

However, its specific effects on early liver development remain poorly understood. In this study, we investigated the hepatotoxicity of 6PPD using zebrafish larvae and human L02 hepatocyte models. A novel exposure strategy was employed, initiating 6PPD treatment at 48 h post-fertilization, after liver budding, to minimize interference from early-stage developmental defects. 6PPD exposure led to a marked reduction in liver size without obvious morphological abnormalities, alongside downregulation of hepatocyte marker genes. Importantly, liver growth gradually recovered after 6PPD removal, suggesting acute and reversible toxicity. Mechanistically, 6PPD induced DNA damage and apoptosis in hepatocytes, as evidenced by elevated γ-H2AX, baxa, and casp3a expression, while hepatocyte proliferation remained unaffected. Transcriptomic and qPCR analyses revealed activation of inflammatory pathways and increased macrophage infiltration. Co-treatment with the anti-inflammatory agent dexamethasone rescued liver size and reduced DNA damage, indicating inflammation as a key mediator of 6PPD-induced toxicity. Similarly, 6PPD exposure in human hepatocytes reduced viability and increased apoptotic markers, which were alleviated by dexamethasone. These results demonstrate that 6PPD causes acute, inflammation-mediated liver toxicity during embryogenesis, with conserved mechanisms across species.

Meng, L., et al. (2026). [Comparison on neurotoxicity and immunotoxicity between 6PPD and 6PPD-Q in zebrafish under equimolar exposure conditions]. *Sheng Wu Gong Cheng Xue Bao, 42*(1): 112-132. DOI: 10.13345/j.cjb.250566

N-(1,3-dimethylbutyl)-N'-phenyl-p-phenylenediamine (6PPD) and its derivative N-(1,3-dimethylbutyl)-N'-phenyl-p-benzoquinone (6PPD-Q) have been widely detected in the environment and pose potential threats to ecosystems and human health. Given that 6PPD can be equimolarly converted to 6PPD-Q in the environment and organisms, the toxicity differences and molecular mechanisms of action between the two under equimolar exposure remain unclear. This study systematically compared the neurotoxicity and immunotoxicity of the two substances under equimolar conditions. The results from the zebrafish model showed that both 6PPD and 6PPD-Q induced excessive production of reactive oxygen species (ROS) and inhibited the expression of antioxidant enzymes, leading to oxidative damage and immune dyshomeostasis. Meanwhile, they activated the innate immune system and caused an increase in immune cells. In terms of neural development, both disturbed the expression of neurofunctional genes and induced malformations such as pericardial edema, delayed swim bladder closure, and spinal curvature. Notably, at environmentally relevant concentrations, the two showed similar acute toxicity. However, at sublethal levels, 6PPD-Q exhibited stronger toxicity, with oxidative damage, immunotoxicity, teratogenicity, and neurotoxicity being 1-3 times higher than those of 6PPD, and it significantly impaired the sensory and motor abilities of larval fish. The results of real-time quantitative PCR (RT-qPCR) indicated that both substances regulated the expression of neuro- and inflammation-related genes in a dose-dependent pattern. The gene ontology (GO), Kyoto encyclopedia of genes and genomes (KEGG), disease ontology (DO), and hub gene analyses further revealed differences in the molecular-level action focuses between 6PPD and 6PPD-Q. This study provides new evidence for the identification and early warning of the potential risks of 6PPD and 6PPD-Q at environmental and sublethal levels.

Michelangeli, M. E., et al. (2026). Tyre-derived ecotoxicity: Differentiating the effects from particles and chemical leachates on the blue mussel Mytilus edulis. *Environmental Chemistry and Ecotoxicology, 8*: 934-951. https://doi.org/10.1016/j.enceco.2026.01.003

Tyre particles contain complex chemical additives that can leach out into the aquatic environment, posing potential risks to marine organisms. Despite growing evidence of adverse effects, the relative importance of particle-driven versus chemically mediated toxicity remains poorly explored, especially under environmentally relevant exposure scenarios. This study used the blue mussel (Mytilus edulis) as a model to differentiate these effects by exposing individuals to cryomilled tyre particles (TP), their leachates (L) and pre-leached particles (TPL) over 36 days at the environmentally relevant concentration of 0.1 g/L. Chemical analysis confirmed uptake of key organic additives such as poly(1,2-dihydro-2,2,4-trimethylquinoline) (TMQ), N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD), and 6PPD-quinone (6PPDQ), with certain compounds persisting after depuration. Particle-exposed mussels accumulated higher additive concentrations than those exposed to only leachates, indicating enhanced chemical release from particles. Biomarker responses revealed signs of oxidative stress and neurotoxicity in exposed mussels across all treatments, with earlier responses in leachate exposure and delayed responses during particle exposures. These results demonstrate that chemical additives are key toxicity drivers alongside physical particles, highlighting the importance of considering both pathways in environmental risk assessments. To our knowledge, this study is among the first to experimentally separate particle and leachate specific effects in mussels by using tyre particles before and after leaching to create contrasting chemical loads, thereby providing novel insights into their distinct and combined impacts on marine biota.

Moody, A. H., et al. (2025). Targeted quantitation of 6PPD-quinone in fish tissue samples with liquid chromatography–tandem mass spectrometry. *Environmental Toxicology and Chemistry, 44*(10): 2807-2817. https://doi.org/10.1093/etojnl/vgaf151

The tire additive transformation product *N*-(1,3-dimethylbutyl)-*N*′-phenyl-*p*-phenylenediamine-quinone (6PPD-quinone) has recently garnered global attention due to its acute toxicity to some salmonids, such as coho salmon (*Oncorhynchus kisutch*), and its ubiquitous presence in urban stormwater systems. In this study, we developed and compared the extraction efficiency of two sample preparation methodologies for quantification of 6PPD-quinone among two fish tissue sample types that included fillet of smallmouth bass (*Micropterus dolomieu*) and whole-body samples of *O. kisutch* fry subjected to in vivo exposure tests with 6PPD-quinone. The two sample preparation methods tested included an accelerated solvent extraction (ASE) approach and a sonication extraction approach. Both sample preparation methods included identical purification steps for the crude sample extracts with enhanced matrix removal cartridges. The purified sample extracts were subjected to targeted analysis of 6PPD-quinone using ultra-performance liquid chromatography–tandem mass spectrometry. The results showed that extractions made with the reported ASE method demonstrated significantly higher absolute recovery (80%–96%) of the extracted internal standard, [$^{13}C_6$]-6PPD-quinone, than sonication-based extractions (74%–80%) in both fish tissue sample types. The proposed ASE method shows acceptable limits of

quantification (0.37–0.67 ng g^{-1}), linearity (R^2 > 0.996), and repeatability (relative standard deviation ≤ 9%). This work advances research capabilities for investigations on the toxicokinetic processes of 6PPD-quinone in biological samples.

Nair, P., et al. (2025). Structure and Toxicity Characterization of Alkyl Hydroxylated Metabolites of 6PPD-Q. *Environmental Science & Technology, 59*(15): 7474-7484. https://pubs.acs.org/doi/10.1021/acs.est.4c11823

Distinct from other nontoxic phenyl-p-phenylenediamine (PPD) quinones, N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-Q) was recently discovered to be regioselectively metabolized to alkyl hydroxylated metabolites (alkyl–OH-6PPD-Q) in rainbow trout. It remains unknown whether the unique alkyl–OH-6PPD-Q contributes to the toxicity of 6PPD-Q. To test this, we herein synthesized chemical standards of alkyl–OH-6PPD-Q isomers and investigated their metabolic formation mechanism and toxicity. The predominant alkyl–OH-6PPD-Q was confirmed to be hydroxylated on the C4 tertiary carbon (C4–OH-6PPD-Q). The formation of C4–OH-6PPD-Q was only observed in microsomal but not in cytosolic fractions of rainbow trout (O. mykiss) liver S9. A general cytochrome P450 (CYP450) inhibitor fluoxetine inhibited the formation of hydroxylated metabolites of 6PPD-Q, supporting that CYP450 catalyzed the hydroxylation. This well-explained the compound- and regio-selective formation of C4–OH-6PPD-Q, due to the weak C–H bond on the C4 tertiary carbon. Surprisingly, while cytotoxicity was observed for 6PPD-Q and C3–OH-6PPD-Q in a coho salmon (O. kisutch) embryo (CSE-119) cell line, no toxicity was observed for C4–OH-6PPD-Q. To further confirm this under physiologically relevant conditions, we fractionated 6PPD-Q metabolites formed in the liver microsome of rainbow trout. Cytotoxicity was observed for the fraction of 6PPD-Q, but not the fraction of C4–OH-6PPD-Q. In summary, this study highlighted the C4 tertiary carbon as the key moiety for both metabolism and toxicity of 6PPD-Q and confirmed that alkyl hydroxylation is a detoxification pathway for 6PPD-Q.

Nair, P., et al. (2025). Synthesis and Toxicity Evaluation of p-Phenylenediamine-Quinones. *Environmental Science & Technology, 59*(15): 7485-7494. https://pubs.acs.org/doi/10.1021/acs.est.4c12220

N-(1,3-Dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-Q), the tire rubber-derived transformation product of 6PPD, was recently discovered to cause the acute mortality of coho salmon (Oncorhynchus kisutch). Aiming to identify potential replacement antiozonants for 6PPD that do not produce toxic quinones, seven PPD-quinones with distinct side chains were synthesized to investigate their structure-related toxicities in vivo using rainbow trout (Oncorhynchus mykiss). While 6PPD-Q exerted high toxicity (96 h LC50 = 0.35 μg/L), toxicity was not observed for six other PPD-quinones despite their similar structures. The fish tissue concentrations of 6PPD-Q after sublethal exposure (0.29 μg/L) were comparable to the other PPD-quinones, which indicated that bioaccumulation levels were not the reason for the selective toxicity of 6PPD-Q. Hydroxylated PPD-quinones were detected as the predominant metabolites in fish tissue. Interestingly, a single major aromatic hydroxylation metabolite was detected for the alternate PPD-quinones, but two abundant OH-6PPD-Q isomers were detected for 6PPD-Q. MS2 spectra confirmed that hydroxylation occurred on the alkyl side chain for one isomer. The structurally selective toxicity of 6PPD-Q was also observed in a coho salmon (CSE-119) cell line, which further supports its intrinsic toxicity. This study reported the selective toxicity of 6PPD-Q

and pinpointed the possibility for other PPDs to be applied as potential substitutes of 6PPD.

Qiu, X., et al. (2024). A transcriptomics-based analysis of mechanisms involved in the neurobehavioral effects of 6PPD-quinone on early life stages of zebrafish. *Aquatic Toxicology, 276*: 107129. https://doi.org/10.1016/j.aquatox.2024.107129

> As an emerging pollutant frequently detected in aquatic ecosystems, the toxicity of N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-quinone) on fish has been confirmed, but insight into the mechanisms underlying those adverse effects is still limited. Thus, we exposed zebrafish embryos to 6PPD-quinone at 0, 0.25, 2.5, and 25 μg/L until 120 h post-fertilization (hpf), and investigated the variations in their development, behavior, monoamine neurotransmitter levels, and transcriptional profile. Exposure to 6PPD-quinone notably elevated the heart rate of zebrafish at 48 hpf (at 2.5 and 25 μg/L) and 72 hpf (at 0.25, 2.5, and 25 μg/L). In the dark-light transition test, the locomotor activity of zebrafish larvae exposed to 6PPD-quinone significantly increased, especially in the dark periods. Exposure to 6PPD-quinone also altered the dopamine level and its turnover in zebrafish, which exhibited significant correlations to their locomotor activity. RNA sequencing identified 394 differentially expressed genes (DEGs), most of which have the molecular function of binding and catalytic activity. Five DEGs were predicted as the key driver genes in the protein-protein interaction networks associated with circadian rhythm (i.e., npas2), protein processing in endoplasmic reticulum (i.e., hsp90b1 and pdia4), and estrogen signaling pathway (i.e., hsp90aa1.1 and hsp90aa1.2). Our findings provide more insights into mechanisms underlying the toxicity of 6PPD-quinone to teleosts and highlight the necessity to assess its potential risks to aquatic ecosystems.

Rao, C., et al. (2026). Effects of 6PPD-Q and Its Hydroxylated Metabolites on function in the Rainbow Trout Cell Line RTgill-W1[Preprint]. *ChemRxiv, 2026*(0403). https://doi.org/10.26434/chemrxiv.15001535/v2

> The tire rubber-derived contaminant 6PPD-quinone (6PPD-Q) exhibits high acute toxicity to salmonid species, yet the toxicological profiles of its hydroxylated metabolites and the underlying mitochondrial mechanisms remain poorly characterized. This study investigated the cytotoxicity and mitochondrial function in rainbow trout gill cells (RTgill-W1) exposed to 6PPD-Q and three mono-hydroxylated metabolites (p-hydroxy 6PPD-Q, 6PPD-Q-1-OH, and 6PPD-Q-4-OH). Using OECD Test Guideline 249 in combination with complementary physiological assays, we demonstrate a clear structure–toxicity relationship (STR). Among the tested compounds, p-hydroxy 6PPD-Q exhibited the greatest toxicity, exceeding that of 6PPD-Q, whereas the alkyl-hydroxylated metabolite showed little to no detectable toxicity. Mechanistic analysis revealed that the toxic variants induced a concentration-dependent collapse of mitochondrial membrane potential (MMP) and severe ATP depletion, concurrent with a significant disruption of intracellular calcium homeostasis. Although no statistically significant differences in total oxygen consumption were observed, likely due to uncoupling-like effects or compensatory cellular dynamics, these results demonstrate that 6PPD-Q and its active transformation products compromise rainbow trout cell viability primarily by targeting the mitochondrial bioenergetic axis.

Ren, S., et al. (2026). Evaluating 6PPD-Q bioavailability and biotransformation in zebrafish by diffusive

gradients in thin-films. *Environmental Research, 294*: 123851. https://doi.org/10.1016/j.envres.2026.123851

> The bioavailability assessment of highly environmentally concerned substance 6PPD-Q for aquatic organisms is of great significance for studying its bioaccumulation potential and environmental risks. Traditional bioanalytical methods face limitations in efficiency and field applicability. This study represented the first to evaluate the novel application of diffusive gradients in thin-films (DGT) technology for predicting the bioavailability of 6PPD-Q in zebrafish (Danio rerio) by simultaneously exposing zebrafish and DGT devices to solutions containing different 6PPD-Q concentrations (5−100 μg L−1). Results showed that DGT could accurately determine the time-weighted averaged (TWA) concentration of 6PPD-Q despite the strong concentration fluctuations caused by degradation and biological uptake. Although the in vivo bioaccumulation and biotransformation of 6PPD-Q in zebrafish are likely complex, a strong positive correlation ($R2 = 0.786−0.996$, $p < 0.05$) between DGT enrichment and zebrafish accumulation was observed at environmentally relevant concentrations (1.90−20.8 μg L−1), confirming DGT's reliability in predicting 6PPD-Q bioavailability. Through high-resolution liquid chromatography-mass spectrometry, four metabolites (hydroxylated, dihydroxylated, sulfated, and glucuronidated derivatives) of 6PPD-Q were identified to be produced by zebrafish and were revealed to be continuously and rapidly excreted into the solution. Notably, DGT effectively captured trace-level metabolite dynamics, yielding superior detection sensitivity and clearer kinetic profiles than direct biological sampling. This study pioneers the application of DGT for assessing the bioavailability of hydrophobic organic pollutants and their metabolites, demonstrating that DGT is a valuable tool for assessing the bioavailability of 6PPD-Q in aquatic organisms and for characterizing the in-situ release kinetics of its metabolites, providing novel insights into 6PPD-Q's environmental behavior.

Selinger, S., et al. (2025). Sublethal 6PPD-quinone exposure impairs swimming performance and aerobic metabolism in juvenile lake trout (Salvelinus namaycush). *Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology, 294*: 110166. https://doi.org/10.1016/j.cbpc.2025.110166

> 6PPD-quinone, an environmental oxidation product of the rubber tire antioxidant 6PPD, has recently gained recognition as a chemical of concern. Frequently detected in road runoff and surface waters, studies have reported this compound to cause acute lethality in several salmonid species at extremely low concentrations, including lake trout (Salvelinus namaycush; 24-h LC50 = 0.51 μg/L). Following exposure, species experiencing acute lethality show characteristic symptoms such as gasping, spiraling, increased ventilation, loss of equilibrium, erratic movements, and tumbling. However, there is a deficit of research targeted at understanding sublethal toxicities of 6PPD-quinone exposure, particularly concerning swimming capability and metabolic function. To evaluate these effects, juvenile lake trout were exposed for 20 h to a measured concentration of 0.46 μg/L 6PPD-quinone in a swim tunnel respirometer to assess temporal changes in standard metabolic rate (SMR) compared to controls. Following exposure, fish underwent a swim trial to determine critical swimming speed (Ucrit), oxygen consumption rate (MO2), active metabolic rate (AMR), aerobic scope (AS) and energetic cost of transport (CoT), followed by analysis of muscle triglyceride and glycogen concentrations. Results showed that 6PPD-quinone exposure impaired swimming performance, evident by a decrease in Ucrit.

Additionally, exposure resulted in decreased AMR, although alterations in SMR were not observed. Decreased concentrations of muscle triglycerides of swam fish were also observed. These findings suggest that environmentally relevant concentrations of 6PPD-quinone disrupt aerobic metabolic capacity in juvenile lake trout, producing adverse effects that diminish endurance and maximum swim speeds, which may affect survival of fish populations.

Selinger, S. J., et al. (2025). Acute cardiorespiratory effects of 6PPD-quinone on juvenile rainbow trout (Oncorhynchus mykiss) and arctic char (Salvelinus alpinus). *Aquatic Toxicology, 280*: 107288. https://doi.org/10.1016/j.aquatox.2025.107288

N-(1,3-Dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-quinone) is an environmental transformation product of the widely used rubber tire antioxidant, 6PPD. Found in stormwater runoff, 6PPD-quinone has been reported to cause acute lethality at ≤1 µg/L in salmonids like coho salmon, rainbow trout, and brook trout. Conversely, other species such as Arctic char and brown trout are insensitive, even when exposed to significantly greater concentrations (3.8–50 µg/L). Sensitive species exhibit symptoms such as gasping, spiraling, increased ventilation, and loss of equilibrium, suggesting a possible impact on cardiorespiratory physiology. This study investigated sublethal 6PPD-quinone toxicities, focusing on cardiovascular and metabolic effects in two salmonids of varying sensitivity: a sensitive species, rainbow trout (Oncorhynchus mykiss) and a tolerant species, Arctic char (Salvelinus alpinus). Fish were exposed to measured concentrations of 0.59 or 7.15 µg/L 6PPD-quinone, respectively, in respirometry chambers for 48 h to assess temporal changes in resting oxygen consumption compared to unexposed controls. Following exposure, cardiac ultrasound and electrocardiography characterized cardiac function in vivo, while blood gas analysis examined blood composition changes. In both species, changes in resting oxygen consumption were observed. In rainbow trout only, a decrease in end systolic volume and an increase in passive ventricular filling, cardiac output, and PR interval length were observed, indicating cardiac stimulation. Cardiorespiratory symptoms observed following rainbow trout exposure might partly be driven by a significant increase in methemoglobin, resulting in an impaired ability to oxygenate tissues. This study is the first to examine the effects of 6PPD-quinone exposure on the cardiorespiratory system of salmonid fishes and provides information invaluable to a better understanding of the mechanism of 6PPD-quinone toxicity.

Showalter, M., & Kholi, N (2025, June 5). *Expanding the Science of 6PPD-Quinone: A new Highly Sensitive Species*. USGS.gov. Retrieved April 22, 2026, from https://www.usgs.gov/centers/western-fisheries-research-center/news/expanding-science-6ppd-quinone-a-new-highly.

This new report describes the results of a Western Fisheries Research Center study and similar work assessing impacts to fish and other species provide critical information that guides efforts to set limits on 6PPD-quinone in watersheds, evaluate technologies to remove the contaminant before it reaches rivers and streams, and to explore chemical alternatives to 6PPD less toxic to fish.

Soucek, D. J., et al. (2026). Time Course of Effects and Tissue Concentrations of 6PPDQ in Three Sensitive

Salmonids with Additional Data for Two Centrarchid Species. *Environmental Science & Technology, 60*(8): 6002-6010. https://pubs.acs.org/doi/10.1021/acs.est.5c12991

> Studies of 6PPD-quinone (6PPDQ) toxicity have identified both sensitive and resistant salmonid species at environmentally relevant concentrations. However, comparisons between species are complicated by varying testing conditions, which can limit the application to derivation of environmental quality guidelines. Furthermore, few studies have reported 6PPDQ tissue concentrations in fish with known exposure concentrations. Using standard methods with flow-through exposures to provide stable 6PPDQ concentrations, we compared time courses of mortality in three sensitive salmonid species (coho salmon Oncorhynchus kisutch, brook trout Salvelinus fontinalis, rainbow trout Oncorhynchus mykiss), evaluated the potential for latent (delayed) mortality, and measured tissue concentrations in both fish that died during exposure and those that survived an additional 7 days in clean water. Further experiments documented insensitivity in two centrarchids (smallmouth bass Micropterus dolomieu and largemouth bass Micropterus nigricans). Our study provided evidence for two mortality time courses: one in which nearly all mortality occurs in the first 24 h and one in which mortalities continue to accumulate throughout the exposure, and 96-h and 24-h LC50s are quite different. We did not observe evidence for the delayed onset of mortality due to 6PPDQ exposure. Most fish that survived exposure had undetectable 6PPDQ concentrations after 1 week in clean water, potentially informing biomonitoring strategies for wild populations.

Stark, J. D. (2025). What Is the 6PPP-Quinone Concentration That Is Protective for Coho Salmon? *Environmental Science & Technology Letters, 12*(12): 1618-1623. https://pubs.acs.org/doi/10.1021/acs.estlett.5c00969

> The USEPA developed an Aquatic Life Screening Value (ASLV) of 11 ng/L for 6PPD Quinone (6PPD-Q), a breakdown product of 6PPD, an antiozonant in automobile tires. Because some coho populations are listed, "Take" requires that no individuals be harmed. The question asked in the present study is whether the ASLV for 6PPD-Q is protective for coho salmon. To answer this question, a concentration–response regression was developed for juvenile coho salmon from the raw data used to generate the three published acute mortality studies. The % mortality from the ASLV was read from the concentration–response regression resulting in a mean predicted mortality (95% confidence limits) of 2% (1–14%). Because of the Take restriction on harming a threatened or endangered species, the protective concentration of 6PPD-Q in coho-bearing streams should be set below the concentration–response curve. The LC1 and 95% CL are 8.5 (1.3–17.8) ng/L. The lower CL is 8.5 times lower than the EPA ASLV. A concentration of 6PPD-Q that does not result in mortality of one individual coho salmon should be below the lower 95% CL of the LC1 (approximately 1 ng/L). Results of this study show that the EPA ASLV is not protective for coho.

Environment Testing and Treatment

The following thirty citations focus on testing processes and potential treatments of environments that are most impacted by 6PPD-Quinone contamination.

Baker, J. A., et al. (2026). Toxicity identification evaluation techniques isolate zinc and 6PPD-Q as causes of acute lethality to rainbow trout in road runoff. *Environmental Toxicology and Chemistry, 45*(1): 184-194. DOI: 10.1093/etojnl/vgaf269

> The buildup of pollutants on impervious surfaces, and their subsequent flush into the environment within stormwater, could worsen with expected increases in prolonged dry periods and extreme rain events due to climate change. As such, the monitoring and treatment of urban stormwaters is becoming a high priority. Of particular interest is road runoff in urban areas, which has been found to be acutely lethal to salmonids and frequently contains elevated concentrations of metals and organic contaminants. In this study, samples of road runoff were collected in the Metro Vancouver area of British Columbia, Canada, and assessed for acute lethality to rainbow trout (Oncorhynchus mykiss). Three of the four stormwaters tested exhibited 100% mortality in the 96-hr test. Stormwater toxicity was demonstrated to be reduced by treatment in a rain garden. Phase I Toxicity Identification Evaluation (TIE) techniques initially identified a metal as the cause of toxicity in one stormwater, which was determined to be zinc after Phase II/III TIE testing. The second stormwater sample revealed an organic constituent to be responsible for toxicity, and subsequent TIE testing implicated N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-Q). The potential contribution of 6PPD-Q to toxicity was assessed by performing TIE techniques on a standard solution of 6PPD-Q in parallel with the stormwater. Chemical analysis of 6PPD-Q using Condensed-Phase Membrane Introduction Mass Spectrometry was used to support toxicity assessments. This is the first study to use the TIE approach to provide a toxicity profile for 6PPD-Q.

Du, M., et al. (2026). Covalent organic frameworks-derived magnetic solid phase extraction approach for enrichment and trace detection of 6PPD and 6PPD-quinone in aqueous matrices. *Talanta, 298*: 128991. https://doi.org/10.1016/j.talanta.2025.128991

> N-(1,3-Dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD), a widely used rubber antioxidant, and its ozonolysis derivative, 6PPD-quinone (6PPD-Q), have recently attracted significant attention due to their toxic effect in urban runoff mortality syndrome and widespread occurrence in aquatic environments. In this study, a magnetic covalent organic framework (Fe3O4@TpPa-NO2) was synthesized and applied as a magnetic solid-phase extraction (MSPE) adsorbent for the selective extraction of environmentally relevant levels of 6PPD and 6PPD-Q from road runoff, Pearl River water, tap water and human urine samples. The nanocomposite exhibited a uniform spherical morphology, high surface area, well-defined mesoporous structure and strong magnetic responsiveness, enabling rapid efficient analyte capture via synergistic hydrogen bonding and π-π stacking interactions. Critical adsorption parameters were systematically optimized to enhance the overall performance. Combined with ultra-performance liquid chromatography-tandem mass spectrometry (UHPLC-MS/MS), the developed method demonstrated good sensitivity (limit of detection: 0.004–0.005 μg L−1), high linearity (R2 ≥ 0.9942) and qualified recoveries (70.2−112.0 %) in real samples. The concentrations of 6PPD and 6PPD-Q ranged from 0.013 to 0.15 μg L−1, further highlighting the widespread occurrence in aquatic environments. Compared to traditional pretreatment methods, the Fe3O4@TpPa–NO2–based MSPE method offers a

sensitive and reliable approach for monitoring rubber-derived pollutants in environmental and biological samples.

Eckenberger, E., et al. (2026). About the Variability of Tire and Road Wear Marker Components in Air: From Emissions to Atmospheric Deposition. *Environmental Science & Technology, 60*(2): 2023-2036. https://pubs.acs.org/doi/10.1021/acs.est.5c12735

Particles originating from tire wear and road interactions, tire and road wear particles (TRWP), are an emerging class of nonexhaust emissions with growing environmental concern. Yet, little is known about their atmospheric abundance and variability due to emissions, transport and transformation processes. This study addressed this gap by quantifying six tire additives that serve as markers for the indirect detection of TRWP in complex environmental samples. Using a newly developed analytical workflow based on high-performance liquid chromatography–mass spectrometry (HPLC-MS), we traced three antiozonants, N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD), N-isopropyl-N′-phenyl-p-phenylenediamine (IPPD), and N,N′-diphenyl-p-phenylenediamine (DPPD), their oxidation products (6PPD quinone and IPPD quinone), and the vulcanization accelerator 1,3-diphenylguanidine (DPG) across a wide range of sample types. This single-method framework enabled us to observe marker variabilities, from pristine tire material to abrasion-related emissions from the testbed and road, airborne ultrafine particles (UFPs), and total atmospheric deposition. Marker composition varied strongly with source, emission conditions, and environmental exposure. During abrasion, 6PPD decreased by ∼90%, while 6PPD quinone increased along the emission pathway. In ambient UFP from six Bavarian (Germany) sites, mean 6PPD concentrations ranged from <0.01 to 0.55 ng m–3. In the particulate fraction in total deposition from an urban, semi-industrial site, the ratio of oxygenated and parent PPD varied seasonally, revealing a higher degree of oxidation during summer. 6PPD dominated in the autumn and winter with an average of 6.6 ± 0.91 ng m–2 day–1, while 6PPDq was highest in spring and summer with average concentrations of 5.6 ± 5.91 ng m–2 day–1 and reaching an estimated annual deposition of 1.7 ± 0.5 μg m–2 year–1. By linking source materials to atmospheric samples, this study demonstrated the traceability of TRWP along their emission pathway for the first time and highlighted the importance of accounting for the chemical transformation of dedicated marker components in assessing their environmental fate.

EnviroMail 30 Update Canada. (2025 24 Feb). *6PPD-Quinone – Mystery Salmon Killer Identified by UW Researchers*. Retrieved April 24, 2026 from https://www.alsglobal.com/sv/news-and-publications/2025/02/6ppd-quinone---mystery-salmon-killer-identified-by-uw-researchers.

ALS Canada first issued EnviroMail 30 in June 2021 to announce the first commercial testing service for 6PPD-quinone (6PPD-Q) in Canada. Since 2021 there has been extreme focus and activity on 6PPD-Q by the research community and by regulators in the US and Canada, due to its prevalence in the environment and its extreme toxicity to coho salmon at sub part per billion levels. This web page contains an overview of the toxicity of 6PPD-Q, discussion of long-term solutions and testing as well as sampling requirements.

EPA offers 6ppd measurement guidance (not law). (2024). *Rubber News, 54*(8): 34-34. Retrieved April 24,

2026, from https://research-ebsco-com.slo.idm.oclc.org/c/tzkni5/viewer/html/3lkfnbdqwz?route=details

The U.S. EPA has released guidance for measuring 6ppd and acute 6ppd-quinone levels in freshwater ecosystems, particularly in stormwater runoff, under EPA Method 1634. The screening values for acute exposures to 6ppd and 6ppd-q in freshwater are 8,900 nanograms per liter (ng/L) and 11 ng/L, respectively. While the guidance is not a law, it aims to help tribes, states, and local governments protect fish populations, especially coho salmon on the Pacific coast, from the harmful effects of 6ppd pollution in waterways. EPA acknowledges data gaps and discrepancies in testing approaches but emphasizes that the screening values are not regulations and do not replace existing laws or regulations.

Gonçalves, C., et al. (2026). Fluorine-bearing covalent organic framework as an efficient solid-phase extraction adsorbent for the analysis of triazines and 6PPD-quinone in run-off waters. *Microchemical Journal, 224*: 117441. https://doi.org/10.1016/j.microc.2026.117441

The urban environment is a source of a wide variety of contaminants, which ultimately run-off or leach into water streams. Triazine herbicides have been extensively used in urban landscape management, leaving behind residues of both the parent chemicals and several degradation products. Another dangerous chemical frequently detected in run-off waters is the tire-wear compound 6PPD-quinone, derived from heavy traffic and use of recycled rubber products. Monitoring of these contaminants in water is enforced by EU legislation; therefore, the development of efficient solid-phase extraction adsorbents is of high importance. Covalent organic frameworks (COFs) have attracted considerable interest in recent years as adsorbents for sample preparation due to their large surface area and the possibility of tailoring their chemical features and pore size to absorb specific environmental pollutants. Herein, we demonstrate that these materials can be employed to prepare efficient and readily applicable solid-phase extraction (SPE) cartridges to monitor nine triazines and 6PPD-quinone in urban run-off waters. Three COF derivatives were tested, and the COF amount, adsorption mechanism, as well as several performance parameters, such as recoveries, breakthrough volume, carry-over, and reusability were investigated. The fluorinated derivative TpBD-(CF3)2 showed good performance with average recoveries of 87.6%, enabling concentration factors from 200 to 1000-fold and ng/L limits of quantification. The performance was comparable to that of the commercial cartridges which contain larger adsorbent amounts, highlighting the potential of COFs for use in miniaturized monitoring devices.

Han, L., et al. (2025). Biodegradation pathways and products of tire-related phenylenediamines and phenylenediamine quinones in solution – a laboratory study. *Water Research, 286*: 124235. https://doi.org/10.1016/j.watres.2025.124235

Para-phenylenediamines (PPDs) are antioxidants added to tires to protect the rubber. They are released from tire and road wear particles (TRWP) but the extent of their aerobic microbial degradation and the transformation products (TPs) formed are not known. Therefore, aerobic microbial degradation of seven tire-related PPDs, parent compounds as well as known transformation products, was studied for up to 28 days. Half-lives ranged from 0.2 ± 0.1 days (N-

(1,3-dimethylbutyl)-N'-phenyl-1,4-benzenediamine, 6-PPD) and 0.6 ± 0.1 days (N-isopropyl-N'-phenyl-1,4-phenylenediamine, IPPD) to 3 ± 0.1 days (N-(1,3-dimethylbutyl)-N'-phenyl-1,4-benzenediamine quinone, 6-PPDQ). A total number of 48 TPs was tentatively identified by liquid chromatography-high resolution-mass spectrometry for the seven study compounds. Of these TPs, only four did not decrease in concentration when the parent compounds were degraded completely. Biotransformation in aqueous solution forms several TPs not known for abiotic, photolytic or oxidative transformation. For the PPDs with aliphatic substituents (6-PPD, IPPD) hydrolysis to 4-HDPA was the major initial transformation. Formation of 6-PPDQ from 6-PPD was not detectable. For the fully aromatic DPPD aerobic microbial transformation, likely, proceeded via a quinone diimine intermediate, leading to products different to those of the aliphatic PPDs. From 6-PPDQ, 26 TPs were detected. A suspect screening for the TPs detected from the biodegradation experiments was performed in data of a soil degradation study over 23 months with TRWP and cryo-milled tire tread (CMTT) and in data from the influent and effluent of a municipal wastewater treatment plant during a rain event. In total, 10 TPs were found in those data with variable intensities, most of which originated from 6-PPDQ. While all seven test compounds were (primary) degraded under aerobic conditions, mineralization was not studied. A number of TPs remain as suspects to search for in the environment.

Kazmi, S. S. U. H., et al. (2026). Pollutant-driven humification of dissolved organic matter fails to mitigate the chronic toxicity of tire-wear contaminants in freshwater bioindicator. *Chemical Engineering Journal, 529*: 173274. https://doi.org/10.1016/j.cej.2026.173274

Tire-wear chemicals 6PPD and its quinone derivative 6PPDQ are ubiquitous aquatic pollutants. Their environmental impact is governed by interactions with dissolved organic matter (DOM), a dynamic natural matrix central to chemical fate. This study provides the first evidence that 6PPD and 6PPDQ drive DOM humification, yet this process fails to mitigate their chronic toxicity to the freshwater clam Corbicula fluminea. Integrated analysis of water chemistry and biological responses revealed that despite negligible degradation, both pollutants, especially 6PPDQ, induced profound DOM shifts toward recalcitrance, evidenced by a rising Humification Index (HIX) and humic-like fluorescence, alongside declining microbial productivity indicators (BIX, protein-like components). This pollutant-driven transformation was insufficient for effective sequestration. Exposure caused significant neurotoxicity (AChE inhibition), oxidative stress (induction of CAT and GSH-Px), and metabolic disruption (increased LDH activity). These physiological impairments triggered substantial behavioral deficits, siphoning rates dropped by up to 65% and burrowing success fell to 39.2%. Microbiome analysis confirmed a stronger disruptive impact from 6PPDQ. Our findings reveal a critical paradox, while tire-wear chemicals catalyze DOM humification, this natural process is an ineffective remediation pathway, resulting in persistent multilevel toxicity. We therefore highlight the necessity of integrating DOM dynamics into risk assessment and the development of engineered solutions for tire-wear pollution.

Kim, M.-Y., et al. (2026). Environmental behavior and the degradation of tire-rubber-related pollutant 6PPD-quinone in soils. *Applied Biological Chemistry, 69*(1): 21. https://doi.org/10.1186/s13765-025-01064-z

N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-Q), a transformation product of the tire rubber antioxidant N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine, has recently been identified as a contaminant of emerging concern due to its acute aquatic toxicity and widespread occurrence. However, its behavior in terrestrial environments remains insufficiently understood. To address this gap, this study investigates the environmental degradation and behavior of 6PPD-Q in three soils with contrasting physicochemical properties through photodegradation, leaching, and adsorption–desorption experiments. Under simulated sunlight, 6PPD-Q exhibited varying degradation kinetics, with sandy soils showing the most rapid photodegradation. Organic- and clay-rich soils demonstrated slower breakdown and greater degradation driven by microbial activity under dark conditions. The soil column leaching test revealed extremely limited vertical mobility, with more than 90% of 6PPD-Q retained within the top 6 cm of soil after artificial rainfall treatment, suggesting a low potential for groundwater contamination. Adsorption–desorption experiments confirmed strong binding to soil organic matter and clay fractions, with less than 10% of the initially sorbed 6PPD-Q desorbed after 48 h. Collectively, these results demonstrate that 6PPD-Q persists primarily in surface soils, where its fate is governed by the interplay between photolytic processes and sorption-driven stabilization. From an applied perspective, the prolonged surface retention of 6PPD-Q raises ecological and agronomic concerns, including chronic exposure risks for soil invertebrates, potential plant uptake, and secondary mobilization during rainfall. This study provides critical mechanistic insights into the environmental fate of 6PPD-Q and highlights the importance of soil-specific assessments for the robust ecological risk evaluation of tire-derived pollutants.

King, M. D., et al. (2025). Tracking 6PPD-Quinone Dynamics in a Coho Salmon-Bearing Stream Following Rain Reveals Elevated Concentrations for Multihour Periods During High Flow. *Environmental Science & Technology Letters, 12*(8): 1026-1031. https://pubs.acs.org/doi/10.1021/acs.estlett.5c00477

Urban runoff mortality syndrome in coho salmon (Oncorhynchus kisutch) is attributed primarily to N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-Q), principally from car tires, that is delivered to streams by surface runoff. However, time-resolved stream 6PPD-Q concentration data remain scarce. We measured rainfall, discharge, and 6PPD-Q concentrations in an urban salmon-bearing stream over four rain events to capture the antecedent dry weather (baseflow) and the increased streamflow (stormflow). Portable autosamplers proved to be effective for continual water sampling. The resulting time series revealed 6PPD-Q flushing during stormflow, likely influenced by surface runoff from roads upstream. Following the initial rise in stream stage (+5% depth), mean 24 h time-averaged 6PPD-Q concentration was 45.0 ng/L, 25-fold higher than baseflow, and the mean 24 h load was 3.73 g, 220-fold higher than the preceding 24-h period. Measured 6PPD-Q concentrations, which peaked at 237.0 ng/L, exceeded the LC50 reported for juvenile coho salmon for periods ranging from 3.5 to 18.7 h during rain events, but never during the preceding dry period. Accordingly, coho salmon habitat can be subjected to repeated pulses of 6PPD-Q over the course of the wet season during elevated streamflow periods, which may expose spawning adults, juveniles, and hatching fry to toxic concentrations for considerable periods.

Lei, Y., et al. (2026). Exploring a green-approach for 6PPD and 6PPD-Q removal: Riverine plants in natural wetlands. *Journal of Hazardous Materials, 505*: 141511. https://doi.org/10.1016/j.jhazmat.2026.141511

> The tire-derived antioxidant 6PPD and its highly toxic transformation product (TP) 6PPD-Q represent an emerging threat to aquatic ecosystems. For example, 6PPD-Q is identified as the cause of acute mortality in coho salmon. This study investigates the phytoremediation potential of riverine plants in natural wetlands (e.g. Typha minima (T. minima)) to remove 6PPD and 6PPD-Q. T. minima accelerated the removal of 6PPD and 6PPD-Q from the medium compared to the unplanted controls, with a half-life of 21.31 min and 23.97 h respectively. Total 80 TPs were identified in roots and leaves. Multiple novel pathways were proposed to elucidate the biotransformation processes. The formation of hydrophilic TPs demonstrated the potential of T. minima in detoxifying 6PPD and 6PPD-Q. The occurrence of TPs was related to the activities of detoxification enzymes, such as cytochrome P450 monooxygenase. Although 6PPD and 6PPD-Q resulted in oxidative stress symptoms in T. minima, these stresses could be mitigated by their antioxidant defense system. The findings of the present study provide insights into the metabolic fate of 6PPD and 6PPD-Q in T. minima. This offers a foundation for natural wetlands in mitigating the ecological risks of tire-derived micropollutants in river basin management.

Li, W., et al. (2026). Reclaimed water in road cleaning: An unrecognized risk for toxic 6PPD-Q formation from tire wear waste. *Waste Management, 216*: 115466. https://doi.org/10.1016/j.wasman.2026.115466

> Tire wear particles (TWPs) are a major source of microplastic pollution, particularly due to toxic additives like N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD) and its transformation product, 6PPD-quinone (6PPD-Q). However, the mechanisms governing 6PPD transformation in TWPs are not yet fully understood. This study investigates photo-aging of TWPs as solid waste under simulated road conditions, focusing on how reclaimed water versus rainwater influences aging and 6PPD/6PPD-Q release. Sunlight and road exposure altered TWPs properties, characterized by surface fragmentation, increased hydrophilicity, and oxygen-containing functional groups. Following three weeks of aging in reclaimed water, the carbonyl index (CI) and hydroxyl index (HI) increased by 323.75% and 72.82%, respectively. The surface O/C ratio rose from 0.149 in pristine TWPs to 0.207 in the reclaimed water group and 0.161 in the rainwater group. This oxidation process was also accompanied by an increased abundance of reactive oxygen species. Under reclaimed water aging conditions, the concentrations of $O_2^{\bullet-}$, $^1O_2$, and •OH increased significantly by 82.95%, 33.72%, and 6.69%, respectively. Leaching experiments revealed a gradual decrease in 6PPD concentration during aging, accompanied by a significant increase in its conversion product 6PPD-Q. Pearson analysis revealed strong positive correlations between 6PPD-Q formation and C=O ($r = 0.65$, $p = 0.003$), $O_2^{\bullet-}$ ($r = 0.44$, $p = 0.031$), and •OH ($r = 0.65$, $p = 0.01$). These findings indicate that tire wear solid waste management is highly sensitive to external conditions, with reclaimed water in road cleaning promoting 6PPD-Q formation. This underscores the need to reassess water reuse practices in urban maintenance to mitigate hazardous emissions.

Li, Z., et al. (2025). A Rapid Testing Kit for 6PPD Screening in Rubber Products. *Analytical Chemistry, 97*(17): 9336-9344. https://pubs.acs.org/doi/10.1021/acs.analchem.5c00079

N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD) has received increasing attention due to its ubiquitous environmental occurrence and extreme aquatic toxicity of its oxidation product 6PPD-quinone. Given 6PPD's application as an antioxidant in a wide array of rubber products, cost-effective detection of 6PPD is important for product and waste management. We herein developed a rapid testing kit for the detection (<10 min) of 6PPD in rubber products with low costs (<$1/sample), which was inspired by the observation of a red-colored product after 6PPD exposure to oxidants. The selectivity of this reaction was evaluated by 14 structurally diverse phenols and anilines. Interestingly, the protonated N-1,3-dimethyl butyl-N′-phenyl quinone diimine (6QDI+H+), rather than its neutral or radical counterparts, appeared to be the red-colored product with λmax = 490 nm. We further developed a "green" and user-friendly 6PPD rapid testing kit, which was further applied to a wide array of 50 rubber products. 6PPD was detected in 22 out of 50 samples, with a strong agreement (R2 = 0.896) observed between the results of the rapid testing kit and LC-MS. In summary, this study introduces a 6PPD rapid testing kit for users without requiring expertise in chemistry.

Liu, B., et al. (2025). Advances in Analytical Determination Methods and Toxicity and Health Risk Assessment of 6PPD and Its Transformation Products in Food. *Toxics, 13*(12): 1076. https://doi.org/10.3390/toxics13121076

N-(1,3-Dimethylbutyl)-*N*′-phenyl-*p*-phenylenediamine (6PPD) is a member of the *p*-phenylenediamines (PPDs), recognized as a highly effective antioxidant. It has been extensively employed in the automotive tire manufacturing industry, and plays a critical role in enhancing the durability and service life of rubber materials. In recent years, significant research has demonstrated that 6PPD-quinone (6PPD-Q), the transformation product of 6PPD, is a toxic substance that causes the acute death of coho salmon (*Oncorhynchus kisutch*). The toxicity of its aquatic organisms has attracted great attention of scholars, and 6PPD-Q has been regarded as the emerging contaminant. It has been reported that 6PPD diffuses from rubber debris into environmental media such as air, soil, and water after the tires wear. 6PPD and 6PPD-Q have been widespread in the environment, and they migrate into food through the environment and enter the human body through exposure routes such as dietary intake and drinking water, posing potential risks to human health. This paper reviewed the current reports on the toxicity and health risks of 6PPD and 6PPD-Q, and compares the advantages and disadvantages of sample pretreatment methods and detection technologies of 6PPD and 6PPD-Q in different food matrices, and provides a scientific basis for food safety risk assessment. Evidence indicated that 6PPD-Q exhibits not only acute aquatic toxicity but also cytotoxicity, hepatotoxicity, neurotoxicity, and genotoxicity. Epidemiological data suggest a significant association between elevated 6PPD-Q levels and increased risks of colorectal cancer and liver abnormalities. There remains an urgent need to develop comprehensive, standardized, and high-throughput analytical methodologies for the efficient screening of 6PPD and 6PPD-Q in food samples, along with expanded dietary exposure assessments, to fully characterize the impacts of 6PPD and 6PPD-Q on human health.

Liu, G., et al. (2026). 6PPD-Q degradation by self-powered bio-electro-Fenton and response of functional microbial communities. *Chemical Engineering Journal, 531*: 174167.

https://doi.org/10.1016/j.cej.2026.174167

N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine quinone (6PPD-Q), an emerging pollutant derived from tire wear particles, exhibits low concentration toxicity and widespread presence, adversely affecting aquatic ecosystems and human health. Its degradation from aquatic environments has become a focal point of research. Existing degradation methods suffer from drawbacks such as requiring external energy input or having excessively long degradation cycles. This study constructed a bio-electro-Fenton (BEF) self-powered device that provides favorable degradation conditions and is environmentally friendly. We investigated the degradation mechanism and potential pathways of 6PPD-Q, as well as the response of microbial communities in the BEF to 6PPD-Q and its degradation products. The BEF achieved a maximum power density of 0.35 W/m2 and a maximum H_2O_2 yield of 1.68 mg/L, degrading 95.64% of 6PPD-Q within 48 h. Six degradation products of 6PPD-Q were identified via UPLC-Orbitrap-MS/MS analysis, proposing three potential degradation pathways (dealkylation, ring oxidation, and quinone epoxidation). Additionally, the addition of 6PPD-Q reduced microbial community diversity in BEF, decreasing the abundance of certain electroactive bacteria such as Pseudomonadota. This study contributes a novel method for degrading the emerging pollutant 6PPD-Q using an autotrophic and environmentally friendly advanced oxidation technology.

Liu, Y., et al. (2025). Targeted and non-targeted analyses reveal trophic biomagnification of rubber-derived chemicals in an estuarine food web near China's largest rubber production region. *Journal of Hazardous Materials, 495*: 138958. https://doi.org/10.1016/j.jhazmat.2025.138958

The emergence of rubber-derived chemicals (RDCs) has garnered significant environmental attention, particularly N-(1,3-dimethylbutyl)-N'-phenyl-p-phenylenediamine quinone (6PPD-Q) linked to acute mortality in salmonids. However, the trophic transfer of RDCs within marine ecosystems is notably sparse. Here, we investigated RDCs in the Nandu River estuary near China's largest rubber production region through an integrated targeted and non-targeted approach. Twelve targeted and 28 non-targeted RDCs were identified in biological samples. For targeted RDCs, total mean concentrations (ng/g, wet weight) showed a descending order of crab (702) > shellfish (531) > snail (491) > shrimp (392) > cuttlefish (342) > fish (190), while those for non-targeted RDCs followed the order of fish (3.91 ×103) > shellfish (3.08 ×103) > cuttlefish (2.96 ×103) > shrimp (2.78 ×103) > snail (1.61 ×103) > crab (1.36 ×103). Thirteen RDCs exhibited biomagnification potential with trophic magnification factors (TMFs) of 1.05–3.24, while nine compounds displayed biodilution trends (TMFs: 0.56–0.93). Organism-water partition coefficients, derived from multi-parameter linear free-energy relationships, more accurately elucidate biomagnification mechanisms of RDCs. Furthermore, 60 % of RDCs exhibited persistence, bioaccumulation and toxicity properties. This research underscores the importance of integrating targeted and non-targeted approaches of RDCs in biomonitoring, offering a more comprehensive view of cumulative chemical risks to humans and wildlife.

Nicomel, N. R. and L. Li (2026). Sewage-sludge-based activated carbon as a sustainable adsorbent for near-complete removal of 6PPD-quinone at environmentally relevant levels. *Ecocycles, 12*(1): 38-49. https://doi.org/10.19040/ecocycles.v12i1.645

The tyre-derived transformation product 6PPD-quinone (6PPD-q) poses a growing threat to aquatic ecosystems, particularly sensitive salmonid species. However, practical and affordable treatment options remain limited. This study investigated the potential of sewage-sludge-based activated carbon (SBAC) produced via $ZnCl_2$ activation and pyrolysis as a sustainable adsorbent for removing 6PPD-q from contaminated water and advancing circular-economy approaches for sludge valorisation. Fourier-transform infrared spectroscopy revealed the presence of oxygen-containing surface groups on SBAC that can facilitate hydrogen bonding and *p–p* interactions with 6PPD-q. Batch adsorption experiments were performed to evaluate equilibrium behaviour and thermodynamic properties under controlled conditions. Results showed rapid uptake, achieving >99% removal from an initial concentration of 200 microg/L within 0.5 h at pH 3.5. The Langmuir model best fit the equilibrium data, with R^2 value of 0.95, yielding a maximum adsorption capacity of 583.3 microg/g. Thermodynamic analysis indicated a spontaneous and endothermic process, suggesting chemisorption as the dominant mechanism. The adsorption efficiency remained stable within the temperature range of 7°C–35°C, and sequential treatments maintained high removal performance. The experimental results demonstrate that SBAC is an efficient and low-cost adsorbent for mitigating 6PPD-q contamination in stormwater, offering a sustainable solution for valorising sewage sludge within circular economy frameworks.

Oosthoek, S. (2024 March). Permeable pavements could protect fish by trapping car tire chemicals. *CIC News*. https://www.cheminst.ca/magazine/article/permeable-pavements-could-protect-fish-by-trapping-car-tire-chemicals/.

Permeable pavements have been found to effectively capture car tire particles and associated chemicals, potentially protecting coho salmon from harm. A study conducted by Washington State University researchers tested four types of permeable pavements and found that they captured an average of 96% of applied tire particle mass. These pavements also retained 68% of the toxic chemical 6PPD-quinone, which has been linked to the death of salmon in waterways. The study suggests that permeable pavements, reinforced with carbon fibers, could be a solution to mitigate the contamination of urban waterways.

Platt, K. L., et al. (2025). Aquatic Thermal and Photochemical Reactivity of N-(1,3-Dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD), N-Isopropyl-N′-phenyl-p-phenylenediamine (IPPD), and 6PPD-quinone. *Environmental Science & Technology, 59*(25): 12900-12909. https://pubs.acs.org/doi/10.1021/acs.est.4c12896

A ubiquitously used tire rubber antidegradant, 6PPD (N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine), and its toxic ozonation product, 6PPD-quinone (N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine quinone), have become recognized as important environmental pollutants since 6PPD-quinone (6PPD-Q) was identified as the likely cause of decades of mass Coho salmon kills. The reactivity of 6PPD, 6PPD-Q, and similar phenylenediamines requires study to better understand their environmental fate. This study explores the aquatic reactivity of 6PPD, N-isopropyl-N′-phenyl-1,4-phenylenediamine (IPPD), and 6PPD-Q through thermal and photochemical pathways using both steady-state photochemistry and time-resolved laser spectroscopy techniques. 6PPD was found to rapidly degrade in the dark, with its degradation

rate being highly dependent on the pH, temperature, and oxygen concentrations. IPPD behaves similarly to 6PPD. In contrast, 6PPD-Q is much more stable in the dark. All three chemicals are degraded via direct photochemistry. Regarding indirect photochemistry, 3CDOM* plays a role in the degradation of 6PPD and IPPD but not 6PPD-Q, while 1O2 does not play a significant role for any of the compounds. Reaction rate constants are reported as well as 6PPD-Q molar yields from 6PPD, which were minimal for all aqueous pathways examined. 6PPD-Q may have a longer environmental lifetime as there are fewer degradation pathways. This research will help us to better understand and control these chemicals in the environment.

Environmental Assessment and Water Quality Programs. (2022 October). 6PPD in Road Runoff Assessment and Mitigation Strategies (Publication 22-03-020). Department of Ecology, State of Washington. https://apps.ecology.wa.gov/publications/documents/2203020.pdf

The Washington State Department of Ecology (Ecology) was directed by the state Legislature to seek interim strategies to address the [6PPD-q contamination] issue until longer-term solutions could be identified. The interim strategies describe best management practices (BMPs). Priority areas were defined by their vulnerability to the co-occurrence of the contaminant and salmon. This report is an initial assessment to help with reconnaissance (monitoring and science) of a new contaminant of emerging concern. A prioritization process was cooperatively developed amongst the committee members and future research opportunities were identified.

Ren, S., et al. (2024). Development and application of diffusive gradients in thin-films for in-situ monitoring of 6PPD-Quinone in urban waters. *Water Research, 266*. https://doi.org/10.1016/j.watres.2024.122408

The occurrence and risk of N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-Q), derived from the oxidation of the tire antidegradant 6PPD, has raised significant concern since it was found to cause acute mortality in coho salmon when exposed to urban runoff. Given the short half-life period and low solubility of 6PPD-Q, reliable in situ measurement techniques are required to accurately understand its occurrence and behaviour in aquatic environments. Here, using the diffusive gradients in thin-films (DGT) method with HLB as a binding agent, we developed a new methodology to measure 6PPD-Q in urban waters. 6PPD-Q was rapidly and strongly adsorbed on the HLB-binding gel and was efficiently extracted using organic solvents. The HLB-DGT accumulated 6PPD-Q linearly for >7 d and its performance was not significantly affected by pH (6.5–8.5), ionic strength (0.0001–0.5 M) or dissolved organic matter (0–20 mg L−1). Field evaluation of the DGT method demonstrated its effectiveness in urban runoff, detecting 6PPD-Q levels of 15.8–39.5 ng L−1 in rivers. In snowmelt, DGT detected 6PPD-Q levels of 210 ng L−1 which is two times higher than the value obtained by grab sampling. 6PPD-Q levels were much higher in snowmelt than those in rivers. This indicates that snowfall constitutes an important transport pathway for 6PPD-Q and that DGT effectively captured the fraction continuously released from dust particles in the snow samples. 6PPD-Q posed a substantial risk to migratory fish in urban waters, and its release from tire wear particles requires further investigation. This study is the first to develop a DGT-based method for 6PPD-Q determination in urban waters, and the method can ensure an accurate measurement of the release of 6PPD-Q to

the environment, particularly in rainfall or snowmelt, important pathways for its entry into the aquatic environment.

Rødland, E. S., et al. (2026). A multi-analytical approach to investigate the retention of tyre-road wear particles, tyre-derived chemicals and metals in tunnel wash water. *Environmental Chemistry, 23*(2). https://doi.org/10.1071/EN25075

Tunnel wash water contains high concentrations of tyre-road wear particles, tyre-derived chemicals, and metals, which can threaten aquatic ecosystems if untreated. This study applies a multi-analytical approach to assess pollutant retention and transformation during sedimentation, revealing treatment limitations and emphasising the need for advanced strategies to reduce pollution from enclosed road infrastructures. Tunnel wash water (TWW) accumulates tyre-road wear particles (TRWPs), tyre-derived chemicals (TDCs) and metals at high concentrations, posing risks to aquatic ecosystems if untreated. Understanding pollutant retention and transformation during treatment is essential for effective mitigation. We investigated TWW from the Vålereng tunnel (Oslo, Norway) using a multi-analytical approach. TRWPs were quantified by pyrolysis–gas chromatography–mass spectrometry (PYR-GC/MS) with a marker mix (M4), a single marker (4-vinylcyclohexene, 4-VCH), and automated scanning electron microscopy–energy dispersive X-ray spectroscopy (SEM-EDX) single particle analysis with machine-learning classification (automated single particle analysis, ASPA, MC2). Metals were analysed by inductively coupled plasma–mass spectrometry (ICP-MS) and TDCs by ultraperformance liquid chromatography–tandem mass spectrometry (UPLC-MS/MS). Samples were collected before, during and after sedimentation treatment over 21 days. Zinc (Zn) was the most abundant metal (274–2300 µg L–1), reduced by up to 93% post-treatment. TRWP concentrations ranged from 15–160 mg L–1 (M4), 13–122 mg L–1 (4-VCH) and 13–240 mg L–1 (ASPA MC2), with treatment efficiencies of 95–99.7%. Despite reductions, fine particles (<20 µm) dominated both untreated and treated water (up to 85%). For TDCs, N-(1,3-dimethylbutyl)-N-phenyl-p-phenylenediamine (6PPD) showed 85% reduction, whereas 6PPD-quinone (6PPD-Q) (−26%) and N,N-diphenylguanidine (DPG) (−3.7%) exhibited negative retention, likely due to leaching and transformation. 1-hydroxybenzotriazole hydrate (OHBT) increased by up to 167% during sedimentation. Sedimentation effectively removed particles and metals but was insufficient for several dissolved TDCs, releasing them at ecotoxicologically relevant concentrations (e.g. 6PPD-Q exceeded Environment Protection Authority freshwater limits). This first cross-validation of three TRWP quantification methods highlights the need for advanced treatment technologies and regulatory thresholds for tyre-derived pollutants.

Srivastava, P., et al. (2026). A novel method for assessing chemical leaching from surface water–pavement interactions applied to recycled-tyre reuse products. *Environmental Science: Processes & Impacts, 28*(1): 149-159. https://doi.org/10.1039/D5EM00516G

Globally, recycling of otherwise waste materials into new products is desired. End-of-life tyres are increasingly incorporated into new pavement materials but leaching of entrained chemicals from such products is not well quantified. Chemical concentrations in runoff from pavements may pose environmental and human health risks. High liquid–solid ratio, batch-agitated leaching is standard

practice for assessing leachability and hazards of chemicals-of-potential-concern in contaminated soil and wastes but is not reflective of important exposure scenarios and may mislead. A new static surface leaching procedure (SSLP) is introduced that is more representative of chemical leaching from pavement reuse materials whilst in contact with rainfall/runoff water. SSLP was evaluated over 2–14 d intervals against batch-agitated leaching for two rubberised pavement products containing 10-fold different proportions of crumbed end-of-life tyres. Although, batch leaching showed high mass removal of 1,3-diphenylguanidine (1,3-DPG, 34%) and hexamethoxymethylmelamine (HMMM, 30%), both batch- and SSLP-leached concentrations of 1,3-DPG, HMMM and N1-(4-methylpentan-2-yl)-N4-phenylbenzene-1,4-diamine quinone (6PPD-Q) were below ECOSAR-predicted toxicity thresholds for fish and daphnids. SSLP highlighted differences in chemical leachability based on rubber content of pavement products and offers a method applicable to other scenarios, such as PFAS leaching from concrete/asphalt pavements. The SSLP was shown to approximate one-dimensional leaching from the surface of the pavement and to be dominated by diffusive processes, thus yielding a simple repeatable approach.

Wang, B., et al. (2025). Occurrence, analytical methods, and ecotoxicological effects of 6PPD-Quinone in aquatic environments: A review. *Trends in Analytical Chemistry, 193*. https://doi-org.slo.idm.oclc.org/10.1016/j.trac.2025.118449

The release of 6PPD-quinone (6PPD-Q), a transformation product of the tire antioxidant N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD), has attracted considerable research attention. However, knowledge of 6PPD-Q fate and effects in the aquatic environment remain limited. Here, we systematically review 6PPD-Q detection techniques in aquatic environments, current environmental pollution status, and 6PPD-Q's toxic effects toward aquatic organisms. Currently, 6PPD-Q is mainly detected using high-performance liquid chromatography-tandem mass spectrometry and ultra-high-performance liquid chromatography-tandem high-resolution mass spectrometry. 6PPD-Q is widely detected in road runoff, snowmelt, and river waters with concentrations ranging from 4 × 10−5 to 19 μg/L, which exceed the acute toxicity thresholds for sensitive species, such as coho salmon. In addition, the potential adverse biological effects included developmental toxicity, neurotoxicity, and oxidative stress. Ultimately, these effects may contribute to ecological risk in contaminated aquatic systems. Finally, future directions were highlighted for the detection methods, environmental behaviors, toxicity mechanisms, and long-term ecological effects of 6PPD-Q.

Yu, H., et al. (2025). Efficient catalytic degradation and detoxification of 6PPD-quinone by the multifunctional enzyme system of phanerochaete chrysosporium. *Journal of Hazardous Materials, 494*: 138634. https://doi.org/10.1016/j.jhazmat.2025.138634

The widespread environmental presence and toxicity of N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-quinone, 6PPD-q), a rubber-derived pollutant, necessitates effective degradation strategies. This study demonstrates for the first time that Phanerochaete chrysosporium (P. chrysosporium) achieves a 99.06 % removal rate of 6PPD-q within 7 days through adsorption combined with enzyme catalysis. The breakdown of the quinone structure, primarily driven by lignin peroxidase isoenzymes, is accompanied by carbon chain shortening and

structural simplification, which enhance the bioavailability of degradation products. These metabolites are assimilated and further mineralized by the P. chrysosporium metabolic system. Comprehensive toxicity assessments using zebrafish and Escherichia coli confirmed the biosafety of all degradation products. This study provides mechanistic insights into the fungal degradation of 6PPD-q and presents a sustainable approach for mitigating the environmental risks posed by other pollutants. Furthermore, a new generation of innovative bioremediation technologies can be developed by engineering fungi to regulate extracellular electric potential and enhance catalytic enzyme activity.

Yu, W., et al. (2024). Degradation and detoxification of 6PPD-quinone in water by ultraviolet-activated peroxymonosulfate: Mechanisms, byproducts, and impact on sediment microbial community. *Water Research, 263*: 122210. https://doi.org/10.1016/j.watres.2024.122210

N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine quinone (6PPD-Q) has been identified to induce acute toxicity to multifarious aquatic organisms at exceptionally low concentrations. The ubiquity and harmful effects of 6PPD-Q emphasize the critical need for its degradation from water ecosystems. Herein, we explored the transformation of 6PPD-Q by an ultraviolet-activated peroxymonosulfate (UV/PMS) system, focusing on mechanism, products and toxicity variation. Results showed that complete degradation of 6PPD-Q was achieved when the initial ratio of PMS and 6PPD-Q was 60:1. The quenching experiments and EPR tests indicated that SO4•− and •OH radicals were primarily responsible for 6PPD-Q removal. Twenty-one degradation products were determined through high-resolution orbitrap mass spectrometry, and it was postulated that hydroxylation, oxidative cleavage, quinone decomposition, ring oxidation, as well as rearrangement and deamination were the major transformation pathways of 6PPD-Q. Toxicity prediction revealed that all identified products exhibited lower acute and chronic toxicities to fish, daphnid and green algae compared to 6PPD-Q. Exposure experiments also uncovered that 6PPD-Q considerably reduced the community diversity and altered the community assembly and functional traits of the sediment microbiome. However, we discovered that the toxicity of 6PPD-Q degradation solutions was effectively decreased, suggesting the superior detoxifying capability of the UV/PMS system for 6PPD-Q. These findings highlight the underlying detrimental impacts of 6PPD-Q on aquatic ecosystems and enrich our understanding of the photochemical oxidation behavior of 6PPD-Q.

Zhang, H., et al. (2025). Developing water quality criteria and assessing ecological risks for 6PPD and 6PPD-Q in freshwater ecosystems. *Environmental Pollution, 387*: 127303. https://doi.org/10.1016/j.envpol.2025.127303

N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD) and its derivative N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine-quinone (6PPD-Q), originating from tire wear particles (TWPs), are emerging contaminants (ECs) of growing concern. This study focused on the development of water quality criteria (WQC) for 6PPD and 6PPD-Q in freshwater ecosystems, alongside an ecological risk assessment (ERA). We first derived the hazardous concentration for 5% of species (HC5) for both compounds by constructing species sensitivity distribution (SSD) models based on experimental toxicity data, interspecies correlation estimation (ICE) models, and

quantitative structure-activity relationships (QSAR) models. The short-term and long-term WQC for 6PPD were 26.02 μg/L and 4.30 μg/L, respectively, while those for 6PPD-Q were 0.20 μg/L and 0.15 μg/L, respectively. Subsequently, we used reported surface water concentration data of 6PPD and 6PPD-Q to assess their risk to freshwater ecosystems by calculating risk quotients (RQs) and determining risk probabilities via Monte Carlo simulation and joint probability curves. In contrast to 6PPD, which posed a negligible aquatic ecological risk, 6PPD-Q presented ecological risks in some urban-influenced surface water bodies, particularly after rainfall events. This study provided a theoretical foundation for establishing water quality standards and managing the risks of 6PPD and 6PPD-Q in aquatic ecosystems, while also serving as a valuable methodological reference for the future development of WQC and ERA of other ECs.

Zhang, Q., et al. (2024). Accurate and stable detection of p-phenylenediamine antioxidants and their transformation products in aquatic products using antioxidant protection – Analysis of actual aquatic products. *Journal of Hazardous Materials, 480*: 136099. https://doi.org/10.1016/j.jhazmat.2024.136099

Given the high toxicity of N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine (6PPD) derivatives, such as 6PPD quinone (6PPDQ) to salmon, as well as their ubiquitous presence in the environment, the contaminant of aquatic food products has drawn significant attention. However, analytical methods for p-Phenylenediamines (PPDs) and their transformation products (TPs) in aquatic products remain underdeveloped. In particular, the degradation of some compounds and strong matrix effects complicate detection. In this study, we present a stable, rapid, and sensitive method combining salt-out assisted extraction, antioxidant protection, and multi-plug filtration clean-up (m-PFC) to detect two PPDs and five TPs in aquatic products. Crucially, the appropriate selection of antioxidants prevented the degradation of the easily oxidized target compounds. Further, the m-PFC method significantly enhanced the purification efficiency, achieving satisfactory recoveries (62.1–115 %), and method detection limits (MDLs) ranging from 0.00300 to 0.400 μg/kg. Subsequently, the method was applied to monitor PPDs and their TPs in aquatic products systematically, revealing the presence of 6PPD and N-isopropyl-N′-phenyl-1,4-phenylenediamine (IPPD) in white shrimp from aquafarms, whereas none of the seven target analytes were detected in fish and crab samples. These findings contribute to the detection of PPDs, their TPs and other unstable chemicals in aquatic products, thereby providing insights into their concentrations in these products.

Zhou, L.-J., et al. (2024). Nationwide occurrence and prioritization of tire additives and their transformation products in lake sediments of China. *Environment International, 193*. https://doi-org.slo.idm.oclc.org/10.1016/j.envint.2024.109139

As a group of emerging contaminants of global concern, tire additives and their transformation products (TATPs) are causing a severe threat to aquatic ecosystems, particularly the highly lethal effects of N-(1,3-dimethylbutyl)-N′-phenyl-p-phenylenediamine quinone (6PPD-Q) on certain fish species. Yet, the contamination status of TATPs in the lake ecosystems remains largely uncharacterized. This study conducted the first nationwide monitoring of the distribution characteristics of TATPs in 208 lake sediments collected from five lake regions across China. All the 13 TATPs were identified in lake sediments, with the total levels varying between 1.4 and

1355 ng/g, and 4-hydroxydiphenylamine (4-OH-PPD) as the most dominant. The total levels of TATPs decreased in the following order: Yunnan-Guizhou Plateau > Inner Mongolia-Xinjiang Region, Eastern Plain > Qinghai-Tibet Plateau, and Northeast Plain ($p < 0.05$). The geographical distribution of TATPs in lake sediments was significantly driven by total organic carbon content, temperature, and population density. N,N′-di-2-naphthyl-p-phenylenediamine, 6PPD-Q, N,N′-diphenyl-p-phenylenediamine, and 4-OH-PPD belonged to high-priority contaminants. Our study emphasizes that emerging pollutant TATPs place significant pressure on lake ecosystems and deserve urgent attention.

Zhu, R., et al. (2026). Development of HECAM passive samplers for discovering the occurrence, sources, and transport of tire additives and their transformation products in surface waters. *Environmental Pollution, 395*: 127789. https://doi.org/10.1016/j.envpol.2026.127789

Tire additives and their transformation products (TATPs) such as p-phenylenediamines (PPDs), PPDs-derived quinones (PPDQs), and other industrial additives have been highly concerned emerging contaminants in recent years. The hydrophilic-lipophilic balance sorbent-embedded cellulose acetate membrane (HECAM) is a highly sensitive and efficient passive sampler designed to simultaneously monitor hydrophilic and hydrophobic contaminants in waters. This work evaluated passive sampling performance of twelve TATPs in the HECAM, and a field application was conducted for discovering occurrence, sources, and transport of TATPs in northeast China (Hinggan League, Inner Mongolia). In the laboratorial experiments, the uptake of twelve TATPs in HECAM well followed a first-order kinetic model. Chemical structures of TATPs affect their accumulation and TATPs with moderate polarities have relatively higher equilibrium partition coefficients. In the field sampling, nine TATPs were detected with concentrations ranging from 0.06 ng L−1 to 423 ng L−1. Azoles have much higher levels than PPDs and PPDQs, and all TATPs would transport with runoffs and rivers. As reported data in water of remote northeast China, the PPDs and PPDQs concentrations were much lower than those of south China, which could be related to the low population density (traffic activities) and intense photodegradation in Inner Mongolia. In this area, both human settlements and fungicides use are important sources of the azoles, and they would transport to nearby rivers by the farmland runoffs. This work indicated that HECAM shows potential as a highly efficient sampler for comprehensive monitoring TATPs in surface waters.

Questions?

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