The Abundance and Biochemical Impact of Trifluoroacetic Acid, A Short-Chained PFAS

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Abstract

Per- and polyfluoroalkyl substances (PFAS) are increasingly recognized for their environmental persistence and potential toxicity. Among them, trifluoroacetic acid (TFA) represents an ultra-short-chain PFAS whose global concentrations are rising due to industrial emissions, degradation of other PFAS, and atmospheric transport (Sigmund et al., 2024; Boucher and McGinnis, 2024). While TFA is widely detected in aquatic systems, soils, and precipitation, limited research has examined its chronic biological effects or biochemical mechanisms of toxicity (Dekant and Dekant, 2023). In this study, we conduct a literature review synthesizing geospatial trends in environmental TFA concentrations and experimentally assess its potential to induce oxidative and metabolic stress in Caenorhabditis elegans. L4-stage worms were exposed to varying concentrations of TFA in K medium (0.01–0.5 mM) for 48–60 hours without food, followed by quantitative PCR (qPCR) measurement of stress-related genes (SOD-3, NHR-49, GST-4, CTL-1). Results show concentration-dependent transcriptional modulation, with low to moderate TFA doses (0.01–0.1 mM) generally upregulating oxidative stress–responsive genes and metabolic regulators, and marked downregulation at 0.5 mM coinciding with high lethality. These findings support the hypothesis that TFA exposure induces oxidative stress responses in C. elegans and highlight the need for further investigation into the ecological and human health risks of ultra-short-chain PFAS.

Introduction

Per- and polyfluoroalkyl substances (PFAS) are a chemically diverse class of anthropogenic organofluorines characterized by the presence of at least one fully fluorinated carbon atom (Sigmund et al., 2024). Their strong C–F bonds confer chemical stability, thermal resistance, and hydrophobic/lipophobic properties, which have made PFAS integral to many industrial and consumer applications—including stain-resistant fabrics, non-stick cookware, and firefighting foams (US EPA, 2023). However, these same properties lead to extreme environmental persistence, earning them the nickname “forever chemicals” (US EPA, 2023; ECHA, 2023).

The toxicological risks of long-chain PFAS such as perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS) are well established, with documented links to immune

suppression, endocrine disruption, hepatotoxicity, and developmental toxicity in humans and animals (Li et al., 2025; Sanada et al., 2023). In contrast, research on ultra-short-chain PFAS compounds with two or fewer fully fluorinated carbons remains limited despite evidence of their increasing prevalence in the environment.

Trifluoroacetic acid (TFA) is the simplest perfluorocarboxylic acid, consisting of a $–CF_3$ group attached to a carboxyl carbon. It is both directly emitted and formed as an atmospheric degradation product of hydrofluorocarbons (HFCs) and fluorotelomer-based substances (Garavagno et al., 2024). Due to its high water solubility and low sorption to soils, TFA is highly mobile, frequently detected in precipitation, rivers, lakes, and even drinking water (Sigmund et al., 2024). Environmental surveys have reported TFA concentrations in precipitation ranging from nanograms per liter to over 6 μg/L in certain regions (ECHA, 2023).

Despite its ubiquity, the health and ecological risks of TFA are not well defined. Early in vivo studies in rodents suggested metabolic stress, hepatomegaly, and reproductive impacts at high doses of halothane breakdown to TFA byproducts (Stier et al., 1972; Saillenfait et al., 1997), while more recent work points toward mitochondrial dysfunction and oxidative stress (Dekant and Dekant, 2023; Li et al., 2025). However, longitudinal data and mechanistic insights are lacking, particularly at environmentally relevant concentrations.

The nematode Caenorhabditis elegans offers a powerful model system for investigating environmental toxicants, given its conserved stress-response pathways, short lifespan, and well-characterized genome. Oxidative stress biomarkers such as SOD-3 (superoxide dismutase), CTL-1 (catalase), and GST-4 (glutathione S-transferase) are directly comparable to their human orthologs and provide insight into mitochondrial and cytosolic ROS defense mechanisms (Boucher and McGinnis, 2024). The nuclear hormone receptor NHR-49, orthologous to human HNF4, regulates lipid metabolism, glucose homeostasis, and xenobiotic response.

This study has two primary aims:

- To review the current geospatial literature on TFA abundance in environmental compartments, highlighting trends, data gaps, and regulatory implications.
- To experimentally assess the transcriptional response of oxidative and metabolic stress genes in C. elegans after exposure to increasing concentrations of TFA.

By integrating environmental chemistry with molecular biology, this work addresses a critical gap in PFAS toxicology and contributes to the ongoing debate over whether ultra-short-chain PFAS like TFA should be subject to the same regulatory scrutiny.

Methods and Procedure

2.1 Literature Review of Environmental TFA Concentrations

We conducted a targeted literature review to compile and synthesize available data on environmental concentrations of trifluoroacetic acid (TFA) across global compartments, including precipitation, surface water, groundwater, and drinking water. Search terms included "TFA environmental monitoring," "TFA precipitation," "TFA geospatial distribution," and "TFA PFAS degradation." Databases searched were Web of Science, Scopus, and Google Scholar, supplemented by recent government and regulatory reports (ECHA, 2023; US EPA, 2023).

Data inclusion criteria were as follows:

- Quantitative concentration measurements with units convertible to ng/L or µg/L.
- Sampling location and environmental matrix reported.
- Method of detection (LC-MS/MS or equivalent) provided.

Where available, multi-year datasets were extracted to examine temporal trends. Concentration data from diverse geographic regions were normalized and tabulated in Microsoft Excel, enabling comparisons across continents and environmental compartments. The dataset was organized by sphere (hydrosphere, atmosphere, lithosphere, etc.)

Sphere	Medium	Region (Year)	TFA Concentration	Concentration_ug/L	Concentration_umol/L	Source URL
Atmospher	Rainwater (rural & urban)	Canada	<0.5–350 ng/L	0.0005–0.35 µg/L	0.000004–0.003070	Garavagno et al. 2024
Atmospher	Rain, Rivers, Lakes, Drinking Water	China	Rain: 25–280 ng/L; Rivers: 932–1,014 ng/L; Lakes: up to 2,688 ng/L; Drinking: 319–384 ng/L	0.025–0.28 µg/L	0.000219–0.002456	Garavagno et al. 2024
Atmospher	Rainwater	Fuxin, China	0.70 µg/L	0.7 µg/L	0.006139	Arp et al 2024
Atmospher	Rainwater	Germany	0.21 µg/L	0.21 µg/L	0.001842	Arp et al 2024
Atmospher	Rain, Drinking Water, Surface Water	Germany	Rain: 10–410 ng/L; Drinking: 900–12,400 ng/L; Surface: 480–1,200 ng/L	0.01–0.41 µg/L	0.000088–0.003596	Cahill 2022
Atmospher	Rainwater	Guangzhou	0.152 µg/L	0.152 µg/L	0.001333	Wang et al 2013
Atmospher	Rainwater	Guangzhou (China)	45–974 ng/L	0.045–0.974 µg/L	0.000395–0.008542	Freeling et al 2020
Atmospher	Soil, Rain	Malawi	<0.1–7.5 ng/g soil	0.0001–0.0075 µg/L	0.000001–0.000066	Solomon et al 2016
Atmospher	Rainwater	USA	0.29 µg/L	0.29 µg/L	0.002543	Arp et al 2024
Atmospher	Rain & Fog	USA (CA/NV, Midwest)	20–3,800 ng/L	0.02–3.8 µg/L	0.000175–0.033327	Garavagno et al. 2024
Atmospher	Rain, Drinking Water, Surface Water	United States	Rain: 21–760 ng/L; Drinking: ~79 ng/L; Surface: up to 2,790 ng/L	0.021–0.76 µg/L	0.000184–0.006665	Garavagno et al. 2024
Atmospher	Fog	Europe*	2154 ng/L	2.154 ug/L	0.018891	ECHA Chem
Biosphere	Serum	China	8.46 µg/L	8.46 µg/L	0.074198	Arp et al 2024
Biosphere	Serum	USA	6 µg/L	6.0 µg/L	0.052622	Arp et al 2024
Hydrospher	Surface Water	Canada	Surface: ~21–63 ng/L	0.021- 0.063	0.000184–0.000553	ECHA Chem
Hydrospher	Groundwater	Austria	Avg 0.71 µg/L, max 7.0	0.71 µg/L	0.006227	Joerss et al 2024
Hydrospher	Urban Water	Beijing (2002→2012)	155 ng/L	0.155 µg/L	0.001359	Zhai et al 2015
Hydrospher	Surface & Tap Water	Beijing (urban waters)	Waters: up to 828 ng/L; Tap: 155 ng/L	0.828 µg/L	0.007262	Garavagno et al. 2024
Hydrospher	Surface Water	Denmark	2–10 ng/L	0.002–0.01 µg/L	0.000018–0.000088	Garavagno et al. 2024
Hydrospher	Surface/Ground Water	Europe	>0.5 µg/L in 79% of samples	0.5 µg/L	0.004385	Arp et al 2024
Hydrospher	Drinking Water	Europe (19 countries)	0.23 µg/L	0.23 µg/L	0.002017	Arp et al 2024
Hydrospher	Drinking Water	Germany (2020)	12.4 µg/L	12.4 µg/L	0.108753	Garavagno et al. 2024
Hydrospher	Drinking Water	Indiana, USA (2020)	0.079 µg/L	0.079 µg/L	0.000693	Garavagno et al. 2024
Hydrospher	Surface Water	Sweden	30–820 ng/L	0.03–0.82 µg/L	0.000263–0.007192	Garavagno et al. 2024
Hydrospher	Groundwater	Switzerland	~1–5 µg/L; up to 23 µg/L	1.0–5.0 µg/L; up to 23.0 µ	0.008770–0.043852	Swiss FOEN
Hydrospher	Drinking Water	Switzerland (2023)	>0.5 µg/L	0.5 µg/L	0.201719	Arp et al 2024
Hydrospher	Seawater	Various Global Oceans	0.5–250 ng/L	0.0005–0.25 µg/L	0.004385	ECHA Chem
Hydrospher	Rainwater	Europe*	<0.2-850 ng/L)	0.0002-0.850µg/L	0.000439–0.004385	ECHA Chem
Hydrospher	surface waters	Europe*	<0.2-280 ng/L	(2154 ng/L)	0.000002–0.007455	ECHA Chem
Other	Ice Cores	Arctic (pre-1989 → 2017)	0.13 µg/L	0.13 µg/L	0.000001–0.000082	Arp et al 2024
Other	Plants near site	China	Up to 3,800 mg/kg	3,800,000.0 µg/L	0.00114	Chen et al 2018
Other	Beverages	Global	6.1 µg/L	6.1 µg/L	33327.48641	Scheurer and Nödler 2021
Other	Crop Surveys	Global	50–500 µg/kg	0.05–0.5 µg/L	0.053499	Garavagno et al. 2024

2.2 C. elegans Strain and Culture Conditions

The nematode strain Caenorhabditis elegans (wild-type N2 Bristol) was used as the experimental model organism. Worms were maintained on nematode growth medium (NGM) agar plates seeded with Escherichia coli OP50 at 20 °C following standard protocols (Brenner, 1974). Synchronized L4-stage worms were obtained via timed egg laying and developmental staging.

2.3 TFA Exposure Conditions

Stage L4 worms were concentrated via centrifugation, and 10–20 µL of the condensed worm pellet was transferred to wells containing 1 mL of K medium (pH adjusted according to treatment concentration). Experimental groups were exposed to the following TFA concentrations: 0.01 mM, 0.025 mM, 0.05 mM, 0.1 mM, and 0.5 mM, corresponding to environmental relevance ranges and lethality thresholds determined in preliminary tests (Boucher and McGinnis, 2024).

TFA exposure was conducted in the absence of E. coli to prevent confounding from bacterial metabolism or nutrient availability, following protocols used in prior C. elegans PFAS studies (Boucher and McGinnis, 2024). Exposure duration was 48-60 h at 20 °C with gentle agitation to prevent hypoxic stress.

2.4 RNA Extraction and cDNA Synthesis

Following exposure, worms were collected by centrifugation, washed three times in sterile M9 buffer to remove residual TFA, and mechanically lysed using a bead beater. Total RNA was extracted using the Qiagen RNeasy Mini Kit, following manufacturer's instructions. RNA concentration and purity were determined via NanoDrop spectrophotometry (A260/A280 ratio).

First-strand complementary DNA (cDNA) was synthesized from 1 µg of total RNA using the Thermo Fisher RevertAid First Strand cDNA Synthesis Kit.

2.5 Quantitative PCR (qPCR)

qPCR was performed on an Applied Biosystems QuantStudio system using SYBR Green chemistry. Primers were designed for the following C. elegans genes:

- NHR-49 (nuclear hormone receptor; metabolic regulation; HNF4 ortholog)
- SOD-3 (mitochondrial superoxide dismutase; oxidative stress defense)
- GST-4 (glutathione S-transferase; detoxification enzyme)
- CTL-1 (cytosolic catalase; hydrogen peroxide detoxification)

The housekeeping gene act-1 (actin) was used for normalization. Amplification efficiency was verified for each primer set, and melt curve analysis confirmed the presence of single amplicon products. Relative expression changes were calculated using the ΔΔCt method (Livak and Schmittgen, 2001). Technical triplicates were performed for each biological replicate (n = 3 per treatment).

2.6 Data Analysis

Environmental concentration data were summarized as medians, ranges, and maximum reported values per compartment. For gene expression data, fold changes relative to controls were calculated, and concentration–response trends were visualized using GraphPad Prism. Due to small sample sizes, statistical analyses were limited to descriptive trends, with emphasis on identifying patterns in upregulation or downregulation at specific concentrations.

Results

3.1 Environmental Distribution of TFA

The literature review revealed consistent detection of TFA across environmental compartments in multiple continents, supporting its classification as a global contaminant. Concentrations vary widely by region and environmental matrix:

- Atmosphere and Precipitation
- Surface Waters
- Drinking Water

Spatial analysis showed that concentrations tend to be higher near:

- Industrial regions with known PFAS manufacturing or fluoropolymer use.
- Agricultural zones where fluorinated pesticides or agrochemicals are applied.
- Regions with heavy reliance on HFCs and fluorotelomers that degrade into TFA.
- The consistent detection of TFA in both highly impacted and remote areas underscores its persistence and mobility, qualities that complicate remediation and regulatory oversight.

3.2 Gene Expression Patterns in C. elegans

Quantitative PCR analysis revealed concentration-dependent effects on the transcription of oxidative stress–related (SOD-3, CTL-1, GST-4) and metabolic (NHR-49) genes following TFA exposure.

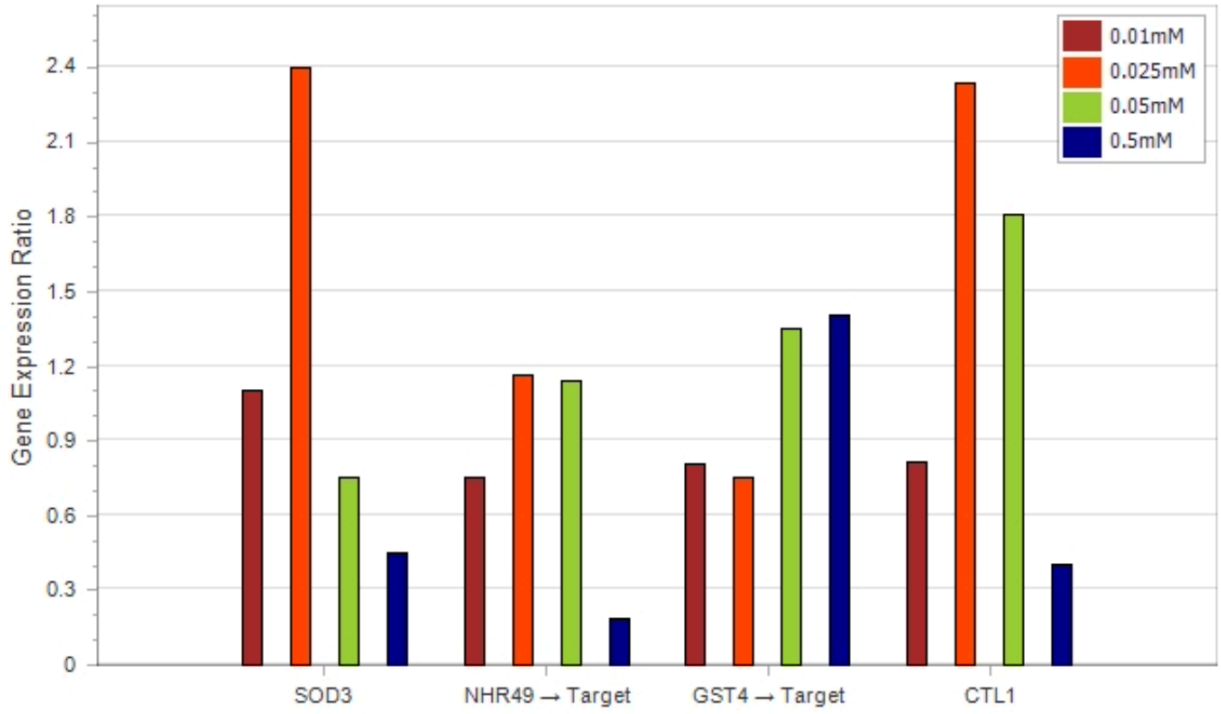

Figure 1. The effects of various TFA concentrations on SOD3, NHR49, GST4, and CTL1 gene expressions

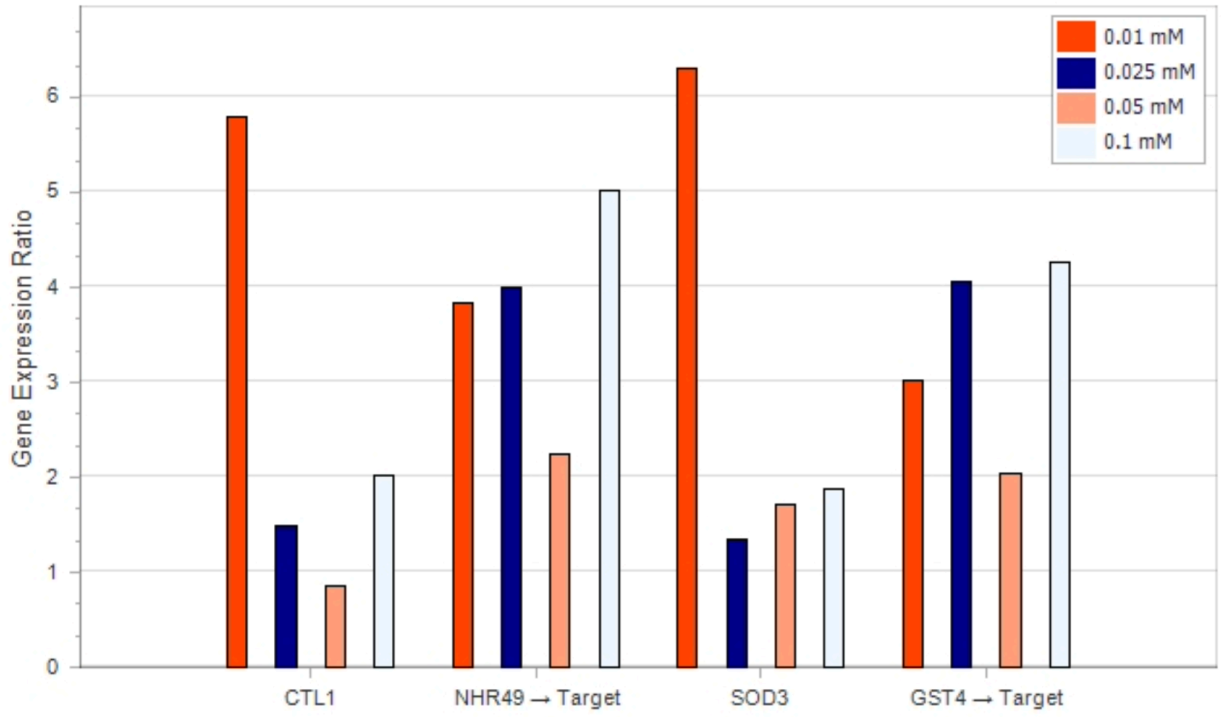

Figure 2. The effects of various TFA concentrations on SOD3, NHR49, GST4, and CTL1 gene expressions

Low concentrations (0.01 mM):

- SOD-3 expression increased relative to controls, indicating early activation of mitochondrial antioxidant defenses.
- CTL-1 was moderately upregulated, consistent with hydrogen peroxide detoxification.
- NHR-49 also increased, suggesting a metabolic adjustment, potentially in lipid metabolism or energy balance.
- GST-4 showed minimal induction, possibly reflecting a threshold for phase II detoxification enzyme activation.

Moderate concentrations (0.025-0.1 mM):

- Upregulation of SOD-3 and CTL-1 continued, although the magnitude plateaued between 0.05-0.1 mM.
- NHR-49 displayed a biphasic pattern of downregulation at 0.025 mM, partial recovery at 0.05 mM, and re-upregulation at 0.1 mM. This fluctuation may indicate shifts between protective metabolic adaptation and early signs of metabolic stress.
- GST-4 expression became more consistently elevated at these concentrations, aligning with an enhanced detoxification response.

High concentration (0.5 mM):

- All four genes were strongly downregulated relative to controls.

- Lethality exceeded 50%, suggesting that transcriptional suppression was associated with acute toxicity and potentially organismal collapse.
- The acidic pH at this concentration (pH ~3.3) may have compounded toxicity, overwhelmed buffering capacity, and contributed to mortality.

3.3 pH and Toxicity Considerations

Measured pH values decreased sharply with increasing TFA concentration: 5.00 at 0.01 mM, 4.60 at 0.025 mM, 4.30 at 0.05 mM, 4.00 at 0.1 mM, and 3.30 at 0.5 mM. The concurrent rise in mortality and gene suppression at high concentrations suggests that pH stress was a major factor at the upper range, although gene-specific responses at lower concentrations indicate that TFA itself, not just acidity, contributed to the observed oxidative and metabolic stress patterns.

Discussion

4.1 Linking Environmental Data to Biological Responses

The environmental monitoring data and C. elegans assays together point to a plausible risk scenario: TFA is both widespread and biologically active at sublethal concentrations. The

transcriptional activation of SOD-3, CTL-1, and GST-4 at environmentally relevant concentrations suggests that organisms in nature could experience oxidative stress even at low µM exposures, particularly in habitats where concentrations approach or exceed 0.1 mM equivalents.

Given TFA's high water solubility and inability to sorb significantly to sediments, aquatic and soil invertebrates are likely among the first ecological receptors. This is especially concerning for benthic organisms in lakes or rivers with high atmospheric deposition inputs, as continuous exposure could translate into chronic oxidative burden and altered energy metabolism.

4.2 Mechanistic Insights

The observed gene expression trends suggest that TFA triggers oxidative stress responses in C. elegans through increased reactive oxygen species (ROS) production.

SOD-3 upregulation implies mitochondrial ROS scavenging, a primary line of defense against superoxide radicals. CTL-1 induction reflects cytosolic hydrogen peroxide detoxification, a secondary step in ROS neutralization. GST-4 activation indicates phase II detoxification pathways, often regulated by SKN-1/Nrf2 in response to oxidative and xenobiotic stress. NHR-49 changes may reflect lipid metabolism adjustments in response to stress, consistent with HNF4 regulation in mammals.

At higher concentrations, the collapse of gene expression and organismal lethality suggests a tipping point beyond which cellular defenses are ineffective, likely due to combined TFA toxicity and acidic stress.

4.3 Regulatory and Policy Implications

One of the most significant implications of this work is regulatory. Under the current U.S. EPA PFAS definition, TFA is often excluded due to its ultra-short-chain structure, despite fitting the broader ECHA definition. Our findings support calls from Sigmund et al. (2024) and others for adopting structure–activity–persistence–toxicity criteria that capture such ultra-short-chain compounds.

The persistence of TFA, combined with its resistance to conventional drinking water treatment and potential to cause oxidative stress at environmentally relevant levels, challenges the assumption that only long-chain PFAS are hazardous. Our environmental synthesis indicates that TFA has already reached a global scale of distribution, making proactive regulation critical.

4.4 Study Limitations and Future Research

Limitations of this study include:

- Acute exposure design; only 48–60 h exposure was tested; chronic effects remain unknown.
- pH confounding; buffering systems should be strengthened in future work to isolate TFA-specific effects.
- Single-species focus; C. elegans is a robust model, but effects on vertebrates and other invertebrates may differ.

Future studies should:

- Conduct similar experiments with other strong acids to see if the effect observed is just in response to pH changes
- Conduct multi-generational C. elegans assays to detect potential transgenerational epigenetic effects.
- Examine behavioral endpoints such as chemotaxis and locomotion, which are sensitive to neurotoxicants.
- Expand cross-species comparisons, particularly with aquatic insects and fish larvae.
- Incorporate environmentally realistic mixtures of PFAS to determine additive or synergistic effects.

Conclusion

This integrated environmental and toxicological assessment reveals that TFA is globally distributed, detected in rain, rivers, lakes, and drinking water, with concentrations in some areas approaching levels that induce oxidative stress in laboratory settings.

In C. elegans, sublethal TFA exposure activates genes central to oxidative defense and metabolism, while higher exposures suppress transcription and cause significant mortality. These findings reinforce concerns about TFA's biological activity and challenge regulatory frameworks that exclude ultra-short-chain PFAS from oversight. Given its persistence, mobility, and demonstrated ability to elicit oxidative and metabolic responses in a model organism, TFA should be considered a PFAS of emerging concern. Though results show a promising relationship, more trials need to be conducted with expanded experimental design to establish significance. Monitoring efforts, refining regulatory definitions, and conducting further mechanistic studies are critical next steps for protecting both ecosystem and human health.

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