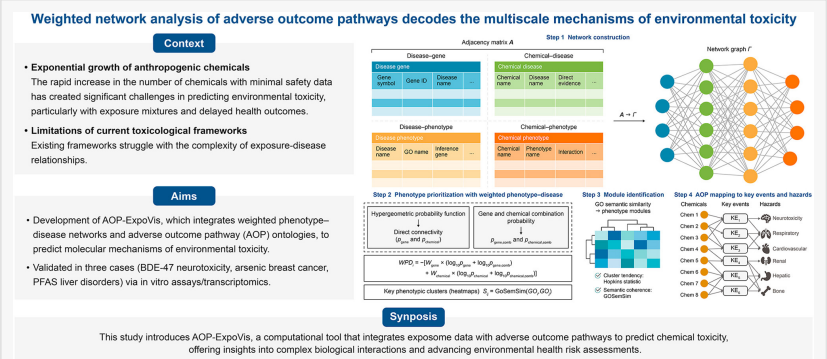
Decoding environmental toxicity: a network-based view of chemical harm

GA, UNITED STATES, February 7, 2026 /EINPresswire.com/ -- Environmental exposure to thousands of synthetic chemicals poses a growing challenge for public health, largely because their biological effects are complex, multiscale, and poorly characterized. This study presents a network-based framework that systematically connects chemical exposures to adverse health outcomes by integrating molecular, phenotypic, and disease-level data. Using a weighted network strategy, the research identifies key biological phenotypes that act as mechanistic bridges between chemicals and disease endpoints, and maps them onto established adverse outcome pathways (AOPs). The approach enables the prioritization of critical toxicological mechanisms across different disease types, offering a scalable way to interpret how diverse environmental chemicals disrupt biological systems and lead to adverse health effects.



Overview of the AOP-ExpoVis framework for decoding environmental toxicity mechanisms.

Modern societies rely on an ever-expanding number of synthetic chemicals, many of which enter the environment with limited toxicological characterization. Traditional toxicity testing, which often focuses on single chemicals and isolated endpoints, cannot keep pace with the scale and complexity of real-world exposures. Although the adverse outcome pathway (AOP) framework provides a structured way to link molecular events to disease outcomes, existing AOPs are fragmented and biased toward well-studied mechanisms. At the same time, exposome research captures broad exposure–disease associations but often lacks mechanistic resolution. Based on these challenges, there is a clear need to develop integrative approaches that can systematically decode the biological mechanisms underlying environmental toxicity.

In a study published in Environmental Science and Ecotechnology on January 31, 2026, researchers from China Medical University and collaborating institutions introduced AOP-ExpoVis, a computational platform that integrates exposome data with AOP knowledge to predict chemical toxicity mechanisms. By combining chemical–gene–phenotype–disease associations into a weighted network, the framework enables the identification of key biological

events linking environmental exposures to adverse health outcomes. The approach was validated across multiple chemical case studies, demonstrating its ability to uncover both shared and compound-specific toxicological pathways.

At the core of the study is a weighted phenotype–disease network that quantifies how strongly specific biological phenotypes connect chemical exposures to disease outcomes. The platform assigns each phenotype a composite score based on both statistical enrichment and network centrality, allowing biologically meaningful mechanisms to emerge while reducing bias from well-studied "hub" genes or chemicals. These prioritized phenotypes are then systematically mapped onto curated AOPs to generate testable mechanistic hypotheses.

The framework was applied to three representative environmental contaminants. For the flame retardant BDE-47, the analysis highlighted neurotoxicity pathways centered on aryl hydrocarbon receptor (AhR) activation, which were subsequently validated using neuronal cell experiments and transcriptomic profiling. In the case of arsenic exposure, the network revealed convergent pathways linking the chemical to breast cancer, including established AhR-mediated mechanisms as well as less explored nuclear receptor signaling routes. For polyfluoroalkyl substances (PFAS), the model distinguished compound-specific liver toxicity patterns, separating inflammation-driven effects from disruptions in lipid metabolism.

Across all cases, computational predictions showed strong concordance with experimental or external transcriptomic evidence, demonstrating that network-based integration can reliably capture multiscale mechanisms of environmental toxicity.

"Environmental toxicity rarely follows a single linear pathway," said the study's corresponding author. "What we see instead is a network of interconnected biological events that differ across chemicals and disease contexts." The researcher explained that AOP-ExpoVis was designed to reflect this complexity by integrating diverse data sources into a unified analytical framework. "By prioritizing biologically central phenotypes rather than isolated molecular signals, the platform helps translate large toxicological datasets into mechanistic insights that are directly relevant for risk assessment and regulatory decision-making."

The AOP-ExpoVis framework offers practical implications for chemical safety evaluation and environmental health research. By rapidly prioritizing hazardous mechanisms from existing data, it can help regulators identify chemicals of concern and guide targeted experimental testing. The approach is particularly valuable for data-poor chemicals, complex exposure scenarios, and emerging contaminants where traditional toxicological evidence is limited. Beyond regulatory use, the platform provides researchers with a systematic tool to explore disease-specific toxicity pathways, supporting hypothesis generation and experimental design. As environmental exposures continue to diversify, network-based strategies like this may play an increasingly important role in protecting public health and advancing predictive toxicology.

References

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