

Applications of *Caenorhabditis elegans* as a Model Organism in Toxicological Research and Forensic Science: An Integrative Overview

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ABOVE THE STUDY

Caenorhabditis elegans (*C. elegans*) has emerged as a powerful model organism in toxicological research and is increasingly being explored for applications in forensic science due to its short life cycle, well-characterized genetics, and strong evolutionary conservation with higher organisms. This nematode completes its life cycle in approximately four days under laboratory conditions, enabling rapid experimental turnover and cost-effective toxicological screening [1]. Its fully sequenced genome and significant genetic homology with humans (approximately 60%-80%) make it highly valuable for translational toxicology, as many biological pathways are conserved across species [2]. In addition, *C. elegans* possesses a completely mapped nervous system with conserved neurotransmitter signaling pathways, making it particularly suitable for studying neurotoxicity and neurodegenerative processes [3]. Its simple, quantifiable, and reproducible behavioral responses, including locomotion, feeding, and reproductive output, allow precise evaluation of toxicological effects [4]. Furthermore, the availability of transgenic strains expressing Green Fluorescent Protein (GFP), such as CL2166 (*gst-4::GFP*) and SJ4005 (*hsp-4::GFP*), has significantly expanded its use in toxicology by enabling real-time monitoring of oxidative stress and endoplasmic reticulum stress responses [5]. These strains are widely distributed through the *Caenorhabditis* Genetics Center (CGC), ensuring standardized experimental use across laboratories. Toxicological studies using *C. elegans* typically involve exposure to toxicants via liquid, solid, or gaseous routes to simulate environmental exposure conditions, and toxicity is assessed through endpoints such as survival rate, growth inhibition, reproductive capacity, developmental delay, and behavioral alterations [6]. Recent advancements have integrated high-throughput screening systems, fluorescence imaging, metabolomics, microfluidic chip technology, and CRISPR/Cas9 gene editing, which together enable detailed mechanistic investigation of toxic effects at molecular, cellular, and organismal levels [7]. In environmental forensic medicine,

C. elegans has been widely applied to evaluate the toxicity of pesticides, heavy metals, nanoparticles, and organic pollutants, providing important insights into environmental contamination and its potential impact on human health [8]. These studies support environmental risk assessment and help establish correlations between pollutant exposure and biological damage, thereby contributing to forensic environmental investigations [9]. For example, exposure to trifluralin has been shown to induce oxidative stress and mitochondrial dysfunction in *C. elegans*, which can be evaluated through measurable physiological and behavioral endpoints such as reduced body length, altered pharyngeal pumping rate, and impaired locomotion [8]. In forensic toxicology, *C. elegans* is used to investigate mechanisms of addiction, neurotoxicity, and developmental toxicity, with ethanol studies identifying multiple genes associated with alcohol response, many of which have human homologs linked to addiction and metabolic disorders [9]. Additionally, it is used in genetic toxicology to assess transgenerational and multigenerational toxic effects of heavy metals and organic pollutants, where oxidative stress and epigenetic regulation are considered key mechanisms underlying inherited toxicity [10]. Observed outcomes include reproductive impairment, neuronal damage, growth inhibition, and cumulative physiological dysfunction across generations. Despite these strengths, challenges remain in standardizing methodologies and integrating *C. elegans*-derived data with human forensic toxicology systems. Future progress should focus on developing comprehensive databases linking metabolic and toxicological pathways from *C. elegans* studies with human forensic data to improve interpretability and legal applicability. Strengthening validation frameworks and improving the evidentiary value of model-derived toxicological findings will be essential for its broader adoption in forensic science. Overall, *C. elegans* represents a highly promising model for advancing toxicological and forensic research, offering a bridge between experimental biology and real-world toxicological applications while enhancing understanding of environmental and human toxic exposures.

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REFERENCES

1. Wu CW, Wimberly K, Pietras A, Dodd W, Atlas MB, Choe KP, et al. RNA processing errors triggered by cadmium and integrator complex disruption are signals for environmental stress. *BMC Biology*. 2019;17:56.
2. Wang Y, Zhang H, Wu X, Xue C, Hu Y, Khan A, et al. Ecotoxicity assessment of sodium dimethyldithiocarbamate and its micro-sized metal chelates in *Caenorhabditis elegans*. *Sci Total Environ*. 2020;720:137666.
3. Wang S, Chu Z, Zhang K, Miao G. Cadmium-induced serotonergic neuron and reproduction damages conferred lethality in the nematode *Caenorhabditis elegans*. *Chemosphere*. 2018;213:11-18.
4. Ngo LT, Huang WT, Chan MH, Su TY, Li CH, Hsiao M, et al. Comprehensive neurotoxicity of lead halide perovskite nanocrystals in nematode *Caenorhabditis elegans*. *Small*. 2023;20:2306020.
5. Currie SD, Doherty JP, Xue KS, Wang JS, Tang L. The stage-specific toxicity of per- and Polyfluoroalkyl Substances (PFAS) in nematode *Caenorhabditis elegans*. *Environ Pollut*. 2023;336:122429.
6. Abbass M, Chen Y, Arlt VM, Stürzenbaum SR. Benzo[a]pyrene and *Caenorhabditis elegans*: Defining the genotoxic potential in an organism lacking the classical CYP1A1 pathway. *Arch Toxicol*. 2021;95:1055-1069.
7. Bargmann CI, Hartweg E, Horvitz HR. Odorant-selective genes and neurons mediate olfaction in *C. elegans*. *Cell*. 1993;74:515-527.
8. Hiroki S, Yoshitane H, Mitsui H, Sato H, Umatani C, Kanda S, et al. Molecular encoding and synaptic decoding of context during salt chemotaxis in *C. elegans*. *Nat Commun*. 2022;13:2928.
9. Yang W, Wu T, Tu S, Qin Y, Shen C, Li J, et al. Redundant neural circuits regulate olfactory integration. *PLOS Genetics*. 2022;18:e1010029.
10. Sun Q, Liu C, Jiang K, Fang Y, Kong C, Fu J, et al. A preliminary study on the neurotoxic mechanism of harmine in *Caenorhabditis elegans*. *Comp Biochem Physiol C Toxicol Pharmacol*. 2021;245:109038.