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When Present in Caenorhabditis Elegans at Quantities Comparable to Those in Contaminated Areas, Perfluorooctanoic Acid (PFOA) Causes Toxicological Effects on Behaviour, Reproduction, and Development

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Introduction

Per- and poly uoroalkyl substances (PFAS) are a class of synthetic chemicals used in a variety of products, including surfactants in re ghting foams, grease-resistant paper for food packaging, non-stick coating for cookware, and stain- and grease-resistant coatings for carpets, fabrics, and upholstery. Heat resistance is provided by the distinctive physicochemical features of PFAS-based polymers, which have been employed in industrial manufacture since 1950 [1]. Because of its longevity in the environment and toxicity to biota, per uorooctanoic acid (PFOA) is one of the PFAS chemicals that has drawn signi cant attention from the general public and researchers. In addition to being employed as a surfactant in many industrial items, PFOA is commonly used in the production of non-stick (Te on® coated) cookware. As a result, PFOA has been discovered in a number of environmental matrices, such as soil, water.

Human unfavourable health consequences from PFOA have been found by epidemiological investigations. Serum PFOA levels have been linked to changes in low-density lipoprotein (LDL), total cholesterol, and high-density lipoprotein (HDL) in human investigations. Exposure to PFOA has been linked to obesity, according to research. Additionally, through the placental bypass of the exposed mother, PFOA can impact foetal development and the risk of obesity in the o spring. PFOA may change liver enzymes, lipids, and size, according to animal studies, which enhance the development of rodent liver peroxisomes. Bartell and co. (2009). Caenorhabditis elegans, an aquatic and terrestrial free-living invertebrate that belongs to the phylum Nematoda, has been proven to be sensitive to a wide range of pollutants. Due to their high brood size (300+) and brief lifespan, C. elegans is simple to cultivate in the laboratory. ey can also generate a big number of animals in a short amount of time. Additionally, the genes of C. elegans are identical to human genes by 60–80%, and the majority of human disease pathways are also present in them. For its capacity to anticipate outcomes in higher eukaryotes (such as humans and rats), C. elegans has been regarded as a superb toxicity model animal [2,3].

Materials and Method

Reagents

e reagents, cadmium nitrate (as a positive control), and per uorooctanoic acid (PFOA) of analytical grade (96% purity) were purchased from Sigma-Aldrich (St. Louis, MO, USA). K-medium (KCl 2.36 g, NaCl 3 g, up to 1 L H2O, cholesterol (5 mg mL1), 1 mL 1 M CaCl2, and 1 mL 1 M MgSO4) was used to generate the PFOA stock solution. PFOA concentrations ranging from 0.25 M (103.5 g/L) to 500 M (207 mg/L) were employed for the acute lethality assay. e following concentrations were employed for various assays: 0.001 M (0.414 g/L), 0.01 M (4.14 g/L), 0.1 M (41.4 g/L), 0.5 M (207 g/L), 1 M (414 g/L), and 2 M (828 g/L). Liquid chromatography-mass spectrometry (LC-MS) was used to assess the PFOA concentrations in the medium; the measured amounts were 99 [4].

PFOA bioaccumulation

Synchronized L4 nematodes were subjected to PFOA at concentrations of 0.001 (0.414 g/L), 0.1 M (41.4 g/L), and 2 M (828 g/L) for 48 hours in order to assess the bioaccumulation. Centrifugation was performed to clean and ush away the bacteria from the worms' intestines using M9 bu er (1 ml 1M MgSO4, 5 g NaCl, 3 g KH2PO4, 6 g Na2HPO4, H20 to 1 L). In Concentrator Plus, samples were dried (Eppendorf). Few modi cations were made to Surowice et alpreviously .'s disclosed procedure for sample extraction (2011). Samples were resuspended in 1 ml of LC-MS grade methanol a er being weighed, and

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Globally, there is growing concern over the possible hazardous efects of PFOA on both human In this work, we examined the acute toxicity of PFOA on Caenorhabditis elegans (C. elegans), a ecologically signifcant animal model. We looked at growth, mortality, lifespan, neurobehavior, 4.42 M (1.83 mg/L) was discovered to be the acute toxicity (LC50) of PFOA. The locomotor beha altered by PFOA exposure. Additionally, after being exposed to 0.1 M (41.4 g/L) PFOA, C. elega was diminished. Likewise, the modifcation in chemotaxis plasticity and lifespan reduction abov were both evidence of developmental toxicity.

they were then homogenised for 5 min using a Branson digital soni er (Model: 102 C - CE) at 15% amplitude. As previously mentioned, the