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BIOLOGY

A SHORT EDITORIAL PERSPECTIVE ON MOLECULAR BIOLOGY METHODS FOR ASSESSING OXIDATIVE STRESS BIOMARKERS IN OCCUPATIONAL WHOLE-BODY VIBRATION–INDUCED NEURODEGENERATION: EVIDENCE FROM ZEBRAFISH MODELS

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Occupational exposure to whole-body vibration (WBV) is an underrecognized risk factor for neurodegenerative changes linked to repeated micro-traumatic brain injuries. These subtle injuries trigger oxidative stress, a key mechanism contributing to neuronal dysfunction and chronic degeneration.This short editorial perspective summarizes current molecular biology methods used to assess oxidative stress biomarkers–superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and malondialdehyde (MDA)–in zebrafish (*Danio rerio*) models of vibration-induced neurotoxicity. Emphasis is placed on quantitative RT-PCR, Western blotting, enzymatic activity assays, and Thio-barbituric acid reactive substances (TBARS) analysis as complementary techniques for evaluating redox imbalance. Findings from reviewed studies demonstrate consistent oxidative patterns between zebrafish and vibration-exposed workers, supporting the translational relevance of this model for occupational neurodegeneration research.

This Short editorial highlights the importance of methodological standardization and cross-species biomarker validation to strengthen oxidative stress assessment in vibration-related disorders. Zebrafish models offer a practical and ethical platform for advancing early detection and prevention strategies in occupational neuroprotection.

Keywords: whole-body vibration (WBV); oxidative stress; zebrafish (*Danio rerio*); neurodegeneration; molecular biology methods; oxidative biomarkers; qRT-PCR; TBARS assay.

1. INTRODUCTION

Occupational exposure to whole-body vibrations is an important yet often underestimated ergonomic and biological hazard for industrial operators, professional drivers, and heavy machinery workers. Chronic WBV exposure produces repeated low-magnitude impacts that can lead to micro-traumatic brain injuries (micro-TBIs) and progressive neurodegenerative changes [1, 2]. A central mechanism linking WBV to neuronal dysfunction is **oxidative stress**, defined as an imbalance between the generation of reactive oxygen species (ROS) and antioxidant defense capacity [3]. Excessive ROS can damage lipids, proteins, and DNA, impairing neuronal communication and cellular integrity.

Within the antioxidant defense system, three enzymes–superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx)–play central roles in detoxifying ROS, while malondialdehyde (MDA) represents a stable end-product of lipid peroxidation and membrane injury [4, 9]. Alterations in these biomarkers reflect redox imbalance and are widely used to quantify oxidative stress in both experimental and occupational contexts. Recent years have seen the emergence of **zebrafish (Danio rerio)** as an efficient vertebrate model for investigating oxidative and neurodegenerative mechanisms. Approximately 70% of human genes, including *SOD1*, CAT, and GPx*1*, have orthologs in zebrafish [4, 8], making this species ideal for mechanistic research on WBV-induced stress.

The present article provides a **short editorial perspective**, grounded in a focused **literature review**, on the molecular biology methods employed to quantify oxidative stress biomarkers (SOD, CAT, GPx, MDA) in zebrafish models of vibration or mechanical neurotrauma [5–7]. By integrating published evidence with interpretative commentary, this paper aims to (i) summarize the principal molecular approaches used to assess oxidative imbalance, (ii) evaluate their experimental applicability and translational potential to occupational health, and (iii) propose directions for future biomarker standardization in WBV-related neurodegeneration research.

OVERVIEW OF THE REVIEWED LITERATURE AND ANALYTICAL APPROACH

A focused review was conducted on studies (2005–2025) indexed in PubMed, Scopus, and ScienceDirect using the terms *whole-body vibration*, *oxidative stress*, *zebrafish*, *SOD*, *CAT*, *GPx*, *MDA*, and *molecular biology methods*. Only peer-reviewed English-language papers were included.

Eligible studies employed zebrafish (*Danio rerio*) or other vertebrate models to examine oxidative stress from mechanical or neurotraumatic exposure, quantified at least one biomarker (SOD, CAT, GPx, or MDA), and applied molecular techniques such as qRT-PCR, Western blotting, enzymatic assays, or TBARS analysis. Non-peer-reviewed and non-quantitative studies were excluded.

The selected literature was analyzed for methodological quality, analytical reliability, and translational relevance. Emphasis was placed on molecular

approaches most consistently used across studies, highlighting their role in elucidating WBV-induced oxidative stress and neurodegenerative mechanisms.

PATHOPHYSIOLOGICAL BACKGROUND

Chronic occupational exposure to WBVs produces repetitive low-level mechanical stress that disrupts neuronal metabolism and may induces micro-traumatic brain injuries. These mechanical insults activate oxidative and inflammatory cascades, leading to the overproduction of ROS and subsequent impairment of antioxidant defense systems. Elevated ROS levels contribute to lipid peroxidation, mitochondrial dysfunction, and protein oxidation—key processes underlying progressive neurodegeneration [11–15,17].

WBV-INDUCED OXIDATIVE STRESS AND THE ZEBRAFISH MODEL

Evidence from recent studies shows that prolonged occupational exposure to whole-body vibration (WBV) contributes to musculoskeletal, vascular, peripheral and central nervous system impairments [1,2,17]. Repeated low-intensity mechanical loading activates oxidative and inflammatory cascades that disrupt neuronal integrity and function [3,17]. These effects arise mainly from excessive production of reactive oxygen species (ROS) and the consequent weakening of endogenous antioxidant defenses. Oxidative biomarkers such as SOD, CAT, GPx, and MDA provide measurable indicators of redox imbalance and cellular damage. Monitoring these markers helps characterize the molecular mechanisms of WBV-induced neurotoxicity and identify potential targets for intervention [16,17].

The zebrafish (*Danio rerio*) has proven to be a valuable vertebrate model for investigating these oxidative mechanisms. With approximately 70% of human genes–including *sod1*, *cat*, and *gpx1*–having functional orthologs [4,8,18], zebrafish enable precise molecular and histological analyses under controlled vibration or micro-impact conditions. Their rapid development, optical transparency, and genetic tractability further enhance their suitability for neurotoxicological studies [18]. Across the reviewed literature, measurements of SOD, CAT, GPx, and MDA in zebrafish yielded oxidative stress patterns closely resembling those found in vibration-exposed workers. These parallels underline the translational significance of zebrafish as a model for identifying early oxidative biomarkers and evaluating preventive or therapeutic strategies in vibration-related neurodegeneration [2,3,18].

MOLECULAR BIOLOGY METHODS FOR ASSESSING OXIDATIVE STRESS BIOMARKERS

The reviewed studies employed a range of molecular biology methods to evaluate oxidative stress in zebrafish models of WBV and related neurotoxic exposures. These techniques differ in sensitivity, specificity, and feasibility, but

together they provide complementary information on enzymatic and molecular responses to oxidative imbalance.

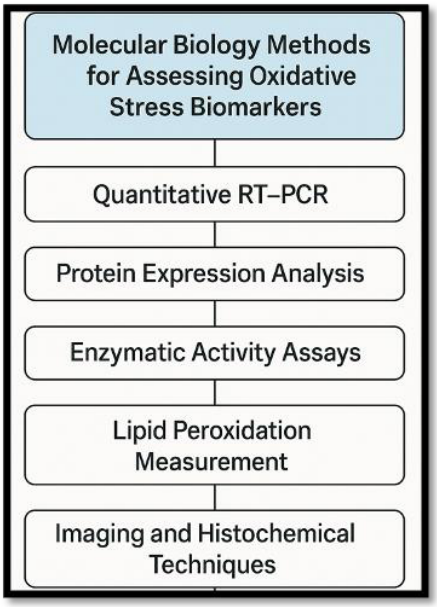

Figure 1. Molecular biology methods for assessing SOD, CAT, GPx, and MDA.
Created by the author using an AI tool.

QUANTITATIVE REVERSE TRANSCRIPTION POLYMERASE CHAIN REACTION (QRT-PCR)

This method measures mRNA expression levels of oxidative stress–related genes such as *sod1*, *sod2*, *cat*, and *gpx1a* [6]. Total RNA is extracted, reverse-transcribed into cDNA, and amplified using specific primers. Expression levels are normalized to housekeeping genes (*e.g.*, *β-actin*, *ef1α*) and calculated using the ΔΔCt method. qRT-PCR is widely used due to its high sensitivity, low sample requirements, and suitability for detecting early transcriptional responses to mechanical stress.

PROTEIN QUANTIFICATION (WESTERN BLOT AND ELISA)

Western blotting remains the standard for confirming oxidative changes at the protein level, allowing semi-quantitative analysis of SOD, CAT, and GPx expression [7]. ELISA assays provide a simpler and faster alternative, enabling

high-throughput measurement of enzyme and MDA concentrations and are readily applicable to zebrafish homogenates [5].

ENZYMATIC ACTIVITY ASSAYS

Spectrophotometric and colorimetric assays remain cost-effective tools for determining enzyme activity. SOD activity is quantified by inhibition of nitroblue tetrazolium reduction, CAT by the decomposition of hydrogen peroxide at 240 nm, and GPx by NADPH oxidation in a coupled reaction with glutathione reductase [7,9].

LIPID PEROXIDATION AND MDA DETERMINATION

MDA levels are most commonly assessed using the TBARS assay, which detects the MDA–TBA adduct at 532 nm [7]. Although less specific than chromatographic methods, TBARS remains the most practical and affordable approach for evaluating lipid peroxidation in both experimental and occupational studies.

IMAGING AND HISTOCHEMICAL TECHNIQUES.

Fluorescent probes such as DCFH-DA or MitoSOX Red visualize intracellular ROS in live zebrafish embryos, while immunohistochemistry for SOD, CAT, and GPx provides spatial localization of antioxidant activity [5].

Collectively, the reviewed evidence highlights that qRT-PCR, enzyme assays, ELISA, and TBARS represent the most accessible and reproducible combination of techniques for assessing oxidative stress in zebrafish models of WBV exposure [5–7].

TRANSLATIONAL RELEVANCE TO HUMAN OCCUPATIONAL EXPOSURE

The reviewed studies demonstrate strong parallels between oxidative responses observed in zebrafish and those reported in human populations exposed to whole-body vibration (WBV). In both models, reductions in antioxidant enzyme activities–SOD, CAT, and GPx–are accompanied by elevated malondialdehyde (MDA) levels, reflecting lipid peroxidation and oxidative damage [2,3]. These biochemical patterns mirror findings in professional drivers, machinery operators, and other vibration-exposed workers, confirming the translational validity of the zebrafish model.

Experimental designs using zebrafish allow precise control of vibration frequency, amplitude, and exposure duration—variables that are difficult to standardize in human studies. Consequently, zebrafish models provide valuable baseline data for identifying early oxidative stress thresholds and dose–response relationships relevant to occupational exposure [4,5,18].

Integrating molecular findings from zebrafish with human biomonitoring data can strengthen risk assessment frameworks and improve early detection of vibration-induced neurodegenerative changes. The reviewed evidence therefore supports zebrafish as a practical and ethically sound model for advancing molecular occupational health research and developing preventive strategies in workers exposed to chronic vibration.

DISCUSSION AND RECOMMENDATIONS

The reviewed literature consistently identifies oxidative stress as a key mechanism linking whole-body vibration (WBV) exposure with progressive neurodegeneration [2,3,9]. Zebrafish provide a cost-efficient and ethically acceptable model for exploring these molecular pathways due to their genetic and physiological similarities to mammals [4,8,18].

Among the molecular approaches analyzed, enzyme activity assays, ELISA, and TBARS emerged as the most accessible and economical for routine laboratory use, while qRT-PCR offers higher sensitivity for detecting early transcriptional changes [6,7]. Combined application of these techniques yields complementary data at both the gene and protein levels, enabling a comprehensive assessment of oxidative status following vibration or mechanical stress.

The comparative synthesis of studies also highlights the need for **methodological standardization** across laboratories. Variations in assay protocols, exposure parameters, and normalization strategies often limit data reproducibility and inter-study comparability. Establishing unified analytical guidelines–such as consistent biomarker units, reference genes, and enzyme activity normalization per protein content–would significantly improve the translational relevance of findings between zebrafish and human studies.

Future research should aim to integrate zebrafish molecular data with human occupational biomonitoring to validate oxidative biomarkers as early indicators of WBV-induced neurodegeneration. Moreover, applying these biomarkers in ergonomic and antioxidant intervention trials could guide evidence-based preventive strategies in vibration-exposed populations.

CONCLUSIONS

Evidence from the reviewed literature confirms that chronic occupational exposure to whole-body vibration (WBV) induces oxidative stress, contributing to neuronal injury and long-term neurodegenerative processes [2,3,9]. Zebrafish (*Danio rerio*) represent a valuable translational model for studying these mechanisms due to their conserved antioxidant pathways and experimental flexibility [4,8].

Molecular biology techniques–particularly qRT-PCR, ELISA, enzyme activity assays, and TBARS–provide reliable and cost-effective means for quantifying the key oxidative biomarkers SOD, CAT, GPx, and MDA in zebrafish models [5–7]. The consistent oxidative patterns observed across animal and human studies support the validity of this model for early detection, mechanistic exploration, and occupational risk assessment.

By integrating molecular findings from zebrafish with human biomonitoring, researchers can establish standardized oxidative stress indicators and develop preventive strategies aimed at reducing WBV-related neurodegeneration. This focused editorial perspective underscores the importance of harmonizing molecular protocols and applying cross-species evidence to advance occupational neuroprotection [18].

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