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Occupational Whole-Body Vibration and Subclinical Mild Traumatic Brain Injury: A Hypothesis on an Oxidative Stress–Mediated Pathway to Neurodegeneration

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Abstract: *Long-term occupational exposure to low-frequency whole-body vibration (WBV) is a recognised risk factor for musculoskeletal disorders. Given that mechanical forces are transmitted systemically, similar effects may plausibly extend to the central nervous system (CNS), although this remains insufficiently investigated. Emerging evidence suggests that WBV exposure may act as a source of repetitive subclinical brain microtrauma, described as minimal (mi-TBI). These low-intensity insults do not produce immediate clinical symptoms or detectable abnormalities on conventional neuroimaging (CT or MRI), but may accumulate and contribute to delayed neuronal dysfunction and long-term neurological consequences. Oxidative stress appears to be central to this process. Increased reactive oxygen species production, combined with impaired antioxidant defenses, may induce lipid peroxidation, mitochondrial dysfunction, neuroinflammation, and blood–brain barrier disruption. Similar mechanisms are established in vibration-exposed peripheral tissues, supporting the plausibility of analogous CNS effects. This review examines the hypothesis that chronic WBV exposure contributes to CNS oxidative imbalance and early neurodegenerative processes. Particular emphasis is placed on oxidative stress biomarkers—including malondialdehyde (MDA), superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase (CAT)—as potential tools for detecting subclinical brain alterations. Additionally, neurofunctional evaluation and emerging technologies such as virtual reality–based tools, are highlighted for detecting subtle cognitive changes. By integrating mechanistic, molecular, and occupational perspectives, we propose a conceptual framework in which WBV is considered a potential source of subclinical mild traumatic brain injury (m-TBI). However, this hypothesis remains supported primarily by indirect evidence and requires validation in human occupational studies.*

Keywords: *whole-body vibration; occupational exposure; subclinical mild traumatic brain injury; minimal Traumatic brain injury; oxidative stress; neuroinflammation; redox biomarkers; neurodegeneration.*

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1. Introduction

Long-term occupational exposure to WBV is widely recognised as a significant risk factor for musculoskeletal disorders, particularly those affecting the spine and peripheral tissues. Workers in transportation, construction, and heavy machinery operation are routinely exposed to low-frequency mechanical vibrations transmitted through seats, platforms, and equipment. Consequently, most research in this field has primarily focused on peripheral tissue damage and biomechanical strain. However, the human body functions as an integrated system, and mechanical forces generated by WBV are not confined to peripheral structures. These vibrations can propagate throughout the body and reach the cranial compartment, where they may exert repetitive mechanical stress on brain tissue. Despite this systemic transmission, the potential effects of WBV on the central nervous system (CNS) remain insufficiently investigated (Yan et al., 2015; Griffin, 1998).

Traumatic brain injury, even in its mildest form, may be a major cause of long-term neurological impairment and involves a complex cascade of biological processes, including oxidative stress, neuroinflammation, mitochondrial dysfunction, and disruption of the blood–brain barrier. While moderate and severe TBI are well characterised, increasing attention has been directed toward mild, repetitive, and subclinical forms of injury. These forms often lack immediate clinical manifestations but may result in cumulative neuronal damage over time (Maas et al., 2017; Ng & Lee, 2019; Haarbauer-Krupa et al., 2021).

In this context, repeated exposure to low-intensity mechanical forces, such as those generated by WBV, may act as a source of repetitive subclinical brain microtrauma, analogous to minimal or subclinical m-TBI. These micro-injuries are typically not associated with overt clinical symptoms or detectable abnormalities on conventional neuroimaging techniques such as computed tomography (CT) or magnetic resonance imaging (MRI). Nevertheless, their cumulative effects may lead to delayed neurological dysfunction and progressive neurodegenerative changes.

Oxidative stress plays a central role in the pathophysiology of brain injury. It is characterised by an imbalance between the production of reactive oxygen species and antioxidant defense systems. Increased oxidative burden, combined with overwhelmed antioxidant defenses, can result in lipid peroxidation, protein oxidation, DNA damage, and mitochondrial dysfunction, ultimately contributing to neuronal injury and degeneration. Notably, similar oxidative mechanisms have been well documented in peripheral tissues exposed to vibration, supporting the hypothesis that comparable processes may also occur within the CNS (Yan et al., 2015; Halliwell, 2006; Aborode et al., 2022; Ismail et al., 2020).

In addition to oxidative stress, other interconnected mechanisms, including neuroinflammation and blood–brain barrier dysfunction, may contribute to progressive neuronal injury. Despite these well-established pathways, the potential contribution of occupational WBV exposure to central nervous system injury has received limited attention (Griffin, 1998; Simon et al., 2017; Shlosberg et al., 2010).

Therefore, this review aims to examine the hypothesis that long-term occupational exposure to WBV may contribute to repetitive subclinical brain injury through oxidative stress–mediated mechanisms. Particular emphasis is placed on oxidative stress biomarkers as potential tools for detecting early, subclinical CNS alterations at the molecular level. Furthermore, this work proposes a conceptual framework linking WBV exposure to delayed neurological outcomes and highlights the need for updated occupational safety strategies, including the integration of advanced assessment approaches for early detection of subtle functional impairment. The proposed conceptual framework is illustrated in Figure 1.

Research Question

Does long-term occupational exposure to WBV induce subclinical m-TBI and oxidative stress in the brain, beyond its known peripheral effects?

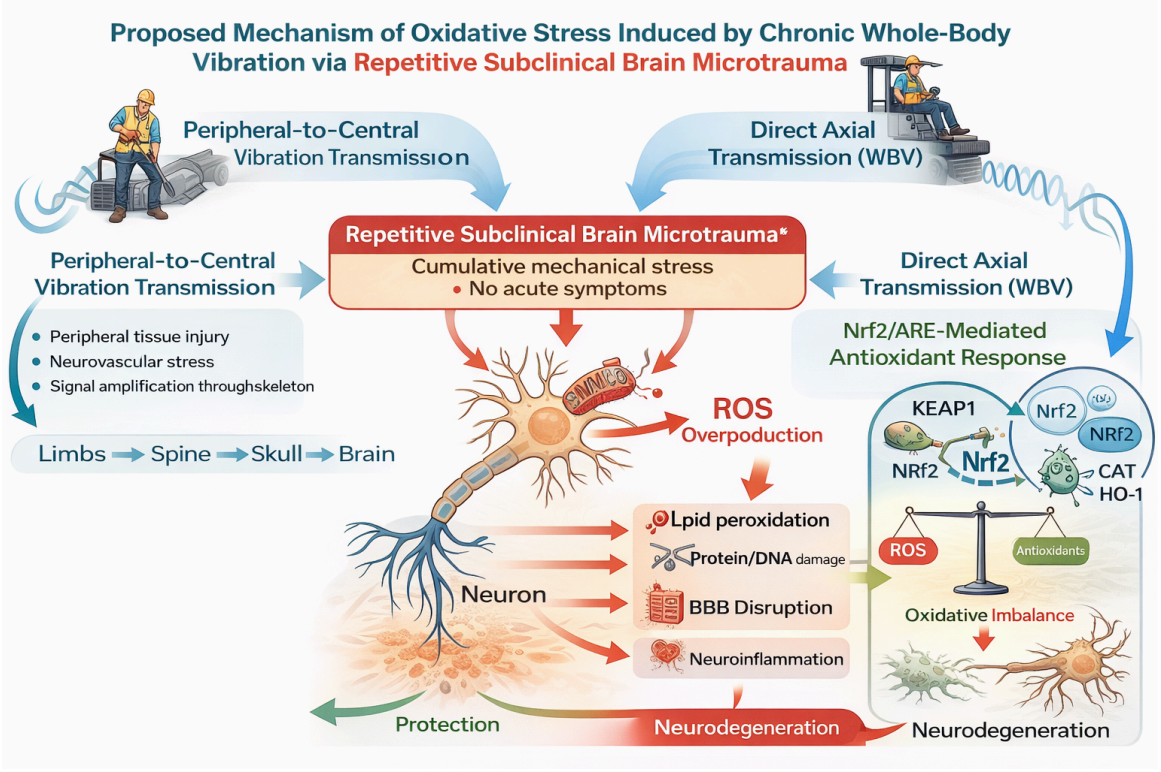

Figure 1. Conceptual framework linking chronic whole-body vibration exposure to oxidative stress and neurodegeneration via repetitive subclinical brain microtrauma.

2. Methodology

This study was conducted as a conceptual narrative review to explore the relationship between long-term WBV, subclinical traumatic brain injury, oxidative stress, and neurodegenerative processes. A literature search was performed in PubMed, Scopus, and Web of Science for studies published between 2000 and March 2026. The search included combinations of keywords such as *whole-body vibration, mild traumatic brain injury, subclinical brain injury, oxidative stress, neuroinflammation,* and *blood–brain barrier.*

A total of 120 records were identified. After removing duplicates and screening titles and abstracts, 40 articles were selected for full-text review. Of these, 33 studies met the inclusion criteria and were included in the final analysis. Relevant studies were selected based on being peer-reviewed, published in English, and examined WBV in relation to central nervous system effects, including oxidative stress or brain injury mechanisms. Both human and experimental studies were included, while studies focusing exclusively on musculoskeletal outcomes without neurological or biochemical findings were excluded.

Relevant data were extracted, including study design, model or population, exposure characteristics, and key neurological and biochemical findings. Due to differences between studies, a narrative synthesis was performed, focusing on main mechanisms such as oxidative stress, neuroinflammation, mitochondrial dysfunction, and blood–brain barrier disruption.

A summary of representative studies is provided in Table 1.

Table 1. Summary of representative studies included in the review

Author (Year)	Study Type	Model/ Population	Exposure	Main Findings
Yan et al. (2015)	Experimental	Animal	Whole-body vibration (WBV)	Repetitive WBV induced brain injury and oxidative stress attenuated by the effect of ApoA-I mimetic peptide
Chen et al. (2022)	Experimental	Animal (TBI model)	Controlled WBV	WBV reduced brain damage and neuroinflammation under specific conditions
Tang et al. (2026)	Experimental	Human/ occupational	Hand–arm vibration	Increased oxidative stress biomarkers linked to vibration exposure
Noël et al. (2022)	Modelling study	Vascular model	Vibration exposure	Reduced wall shear stress and predicted long-term vascular damage
Ionescu et al. (2025)	Clinical/ biomarker	TBI patients	Not WBV-specific	Strong correlation between oxidative stress biomarkers and brain injury
Nguyen et al. (2023)	Review	Experimental models	Brain injury	Mitochondrial oxidative stress is central to brain injury mechanisms
Ismail et al. (2020)	Review	Human/ experimental	TBI	Oxidative stress plays a key role in neuronal damage
Bogdanova et al. (2024)	Systematic review	Human	Cognitive rehab (VR)	VR tools effective in detecting and rehabilitating brain injury
Reneker et al. (2025)	Experimental/ clinical	Human	VR-based detection	VR systems can detect subtle concussion-related deficits
Salawu et al. (2026)	Meta-analysis	Human	VR rehabilitation	VR improves cognitive recovery in brain injury patients

The included studies encompass mechanistic, experimental, and human evidence, which are distinguished in Figure 2 according to their relative level of support for the proposed hypothesis.

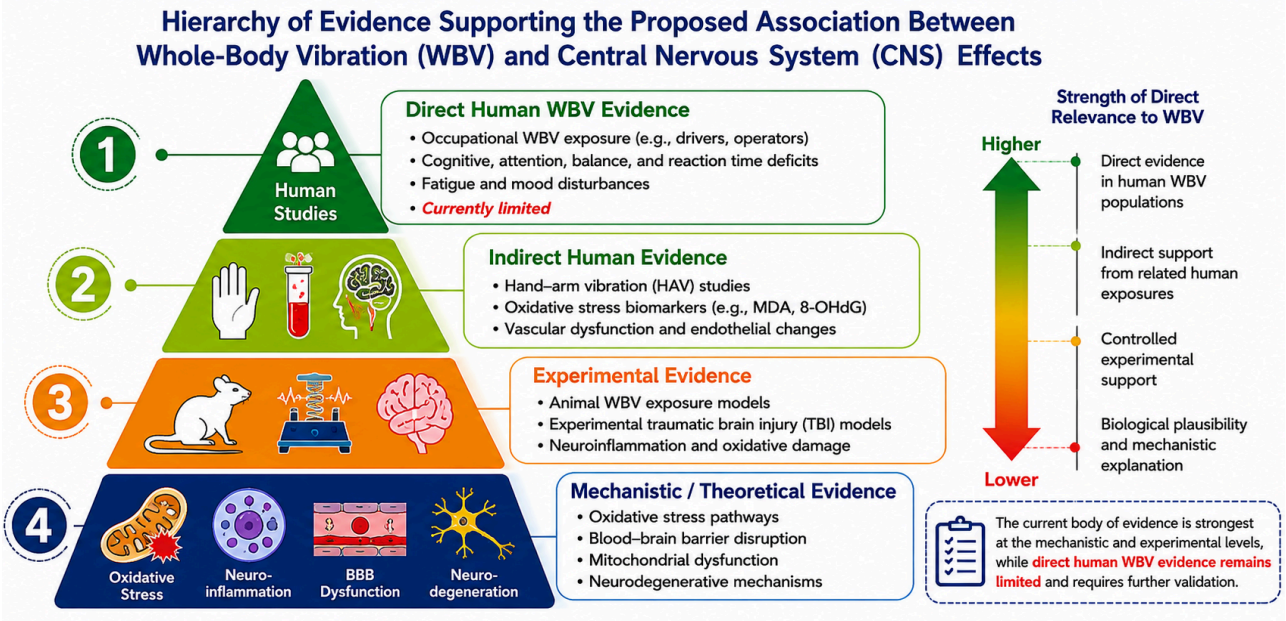

Figure 2. Evidence hierarchy supporting the proposed link between occupational whole-body vibration and subclinical CNS effects, highlighting the predominance of mechanistic and experimental evidence and the limited direct human data.

3. Scientific Background

Traumatic brain injury is a major cause of long-term neurological impairment, characterised by complex and progressive alterations in brain structure and function. Beyond acute injury, increasing evidence highlights the role of repetitive and low-intensity mechanical stress in inducing subtle but cumulative neuronal damage, even in the absence of immediate clinical symptoms (Holm et al., 2005; Maas et al., 2017; Hay et al., 2015).

The pathophysiology of TBI involves multiple interconnected mechanisms, including oxidative stress, neuroinflammation, mitochondrial dysfunction, vascular alterations, and disruption of the blood–brain barrier (BBB). These processes interact and amplify each other, contributing to progressive neuronal injury (Ng & Lee, 2019; Prins et al., 2013).

Oxidative stress represents a central mechanism in this cascade. It occurs when excessive production of reactive oxygen species (ROS) overwhelms the antioxidant defense system, leading to redox imbalance. This imbalance results in lipid peroxidation, protein oxidation, and DNA damage, ultimately impairing cellular integrity and function (Aborode et al., 2022; Algattas & Huang, 2014). In addition, oxidative stress disrupts neuronal ion channels and neurotransmitter systems, contributing to excitotoxicity and neuronal dysfunction, which may culminate in cell death.

Neuroinflammation further amplifies this process. Activation of microglial cells and the release of pro-inflammatory cytokines create a sustained inflammatory environment that exacerbates oxidative damage and neuronal injury. At the same time, vascular dysfunction plays a critical role, as mechanical and biochemical stress can impair cerebral microcirculation, reduce oxygen delivery, and promote endothelial damage (Simon et al., 2017; Muneer et al., 2015; Shlosberg et al., 2010; Hay et al., 2015).

Disruption of the BBB is another key mechanism. Increased permeability allows the entry of circulating toxins, inflammatory mediators, and immune cells into the brain parenchyma, further aggravating neuronal damage and contributing to chronic neurodegenerative processes (Shlosberg et al., 2010; Hay et al., 2015).

A key regulator of the cellular antioxidant response is the Nrf2–Keap1 system. Under normal conditions, Nrf2 is bound to Keap1 in the cytoplasm and undergoes continuous degradation. During oxidative stress, reactive oxygen species modify Keap1, allowing Nrf2 to dissociate, accumulate, and translocate to the nucleus, where it activates antioxidant and cytoprotective genes. Impairment of this system reduces the ability of cells to counteract oxidative stress, increasing susceptibility to neuronal damage and neurodegeneration (Johnson et al., 2008; Casili et al., 2018; Yan et al., 2008; Cheng et al., 2013).

Importantly, these biological processes are not restricted to acute trauma. Similar mechanisms may be triggered by chronic exposure to low-intensity mechanical stress, supporting the concept that persistent external factors—such as whole-body vibration—can induce TBI-like pathological changes over time (Yan et al., 2015).

4. Occupational Exposure to Whole Body and to Hand Arm Vibration (HAV)

Whole-body vibration (WBV) is a common occupational exposure in industries such as transportation, construction, agriculture, and heavy machinery operation (Griffin, 1998). Workers, particularly drivers and machine operators, are routinely exposed to low-frequency mechanical vibrations transmitted through seats, platforms, and equipment. In parallel, hand–arm vibration (HAV) is a major occupational exposure in workers using handheld vibrating tools, and both forms of vibration are typically chronic and repetitive.

Occupational vibration exposure is commonly quantified as a daily exposure standardised to an 8-hour reference period, based on frequency-weighted root-mean-square acceleration according to ISO 5349-1/2 (Griffin, 1998; Noël et al., 2022). For HAV, mechanistic evidence suggests that current weighting approaches may underestimate the pathological importance of some frequency ranges, particularly frequencies above 50 Hz relevant to vascular injury (Noël et al., 2022).

Traditionally, research on vibration exposure has focused on musculoskeletal outcomes, including spinal degeneration and lower back pain (Griffin, 1998). However, increasing evidence indicates that vibration-induced effects extend beyond the musculoskeletal system and involve neurological, vascular, and molecular alterations (Yan et al., 2015;Tang et al., 2026; Noël et al., 2022; Zimmerman et al., 2024).

The peripheral effects of HAV are well established and include sensorineural impairment, carpal tunnel syndrome, and vibration-induced vascular dysfunction, including secondary Raynaud's phenomenon (Tang et al., 2026; Noël et al., 2022; Zimmerman et al., 2024). These disorders are associated with nerve compression, endothelial dysfunction, impaired microcirculation, and altered vascular reactivity, reflecting the combined effects of mechanical stress and biological injury.

At the mechanistic level, vibration exposure has been linked to oxidative and vascular alterations. A multiscale vascular model showed that vibration reduced arterial wall shear stress (WSS) by approximately threefold at 40 m/s², with this effect remaining evident across 31–400 Hz; moreover, wall shear stress decreased logarithmically as vibration amplitude increased (Noël et al., 2022). *Reduced WSS is considered a marker of endothelial dysfunction and impaired microcirculation, which may promote oxidative stress, inflammation, and progressive vascular damage*. The same model simulated approximately 30% arterial stenosis after exposure of 4 hours per day for 10 years, supporting a dose–response concept in chronic vibration injury (Noël et al., 2022).

These findings strengthen the view that vibration exposure induces cumulative biological effects beyond simple mechanical strain. Mechanical vibrations can propagate throughout the body and may reach the cranial compartment, where they could exert repetitive low-intensity stress on brain tissue. Transmission of vibration energy to the head is influenced by factors such as vibration frequency, body posture, and resonance characteristics of the spine. Low-frequency vibrations may propagate through the axial skeleton and generate measurable head motion, providing a plausible biomechanical basis for investigating potential CNS effects of chronic WBV exposure. Although direct evidence for central injury remains limited, the established peripheral vascular, neural, and molecular effects of chronic vibration exposure support the plausibility of broader systemic consequences, including subclinical central injury under prolonged exposure (Yan et al., 2015; Griffin, 1998; Giza & Hovda, 2014; Tang et al., 2026; Noël et al., 2022).

In occupational environments, vibration exposure often coexists with physical fatigue, noise, and psychological strain, which may further increase susceptibility to neurological dysfunction. Despite these considerations, current occupational regulations remain focused mainly on musculoskeletal risk, while the potential neurological consequences of long-term vibration exposure are still largely overlooked (Griffin, 1998; Noël et al., 2022).

5. Human Evidence from Occupational Studies

Human evidence on the neurological effects of whole-body vibration (WBV) remains limited; however, observations from occupational groups such as drivers and heavy machinery operators suggest potential central effects (Griffin, 1998). Reported findings include fatigue, reduced attention, impaired cognitive performance, as well as balance disturbances, and subtle neurobehavioral changes. Moreover, longitudinal epidemiological studies specifically examining central nervous system outcomes in WBV-exposed workers remain scarce, limiting assessment of long-term neurological risk.

Although a direct causal relationship has not yet been established, these observations are consistent with the hypothesis of repetitive low-intensity brain stress. Evidence from mild traumatic brain injury indicates that repeated minor mechanical insults can lead to cumulative neuronal damage and increase the risk of long-term neurological impairment and neurodegeneration (Maas et al., 2017; Simon et al., 2017; Hay et al., 2015; Haarbauer-Krupa et al., 2021).

In addition, oxidative stress biomarkers may serve as early indicators of neuronal dysfunction. Such biomarkers may enable the detection of subclinical alterations before the onset of overt clinical symptoms (Ionescu et al., 2025; Ismail et al., 2020; Maciejczyk et al., 2020; Nijakowski et al., 2024; Jiao et al., 2024).

However, the available evidence remains largely indirect, and direct demonstrations of WBV-induced CNS injury in human populations are currently lacking.

6. Occupational Risk Management and Regulatory Gaps

Current occupational health regulations addressing whole-body vibration are primarily focused on industries such as transportation and construction, with emphasis placed mainly on musculoskeletal outcomes and spinal disorders (Griffin, 1998).

However, the potential neurological effects of vibration exposure remain largely overlooked. Existing guidelines do not include the systematic evaluation of central nervous system involvement, nor do they recommend the use of biological monitoring for early detection of subclinical alterations.

Moreover, there are no standardised protocols for assessing oxidative stress biomarkers or neurofunctional impairment in exposed workers. This lack of integration limits the ability to identify early-stage biological changes and delays the implementation of preventive measures.

Consequently, a significant gap persists in occupational health practice. Without updated frameworks that incorporate neurological risk assessment, subtle but progressive brain-related effects of WBV exposure may remain undetected until more advanced stages (Griffin, 1998; Ionescu et al., 2025).

7. Discussion and Recommendations

This review proposes that long-term occupational exposure to WBV may represent a potential underrecognised risk factor for central nervous system dysfunction. While existing research has predominantly focused on musculoskeletal outcomes, emerging mechanistic evidence suggests that vibration-induced mechanical forces may exert cumulative effects on brain tissue through pathways analogous to those described in traumatic brain injury.

Key mechanisms described above are central to the proposed model linking chronic WBV exposure to potential CNS effects. In particular, BBB dysfunction represents a critical event in brain injury pathophysiology, contributing to neuroinflammation, vascular dysregulation, and progressive neuronal damage (Sivandzade et al., 2020). Similar oxidative and vascular alterations have been reported in tissues exposed to vibration, supporting the biological plausibility that chronic WBV exposure may contribute to comparable effects within the CNS (Yan et al., 2015; Tang et al., 2026). However, it must be emphasised that direct evidence remains limited, and current understanding is largely extrapolated from experimental models and related neurological conditions rather than WBV-specific human studies.

Human data linking WBV exposure to CNS outcomes are scarce and primarily observational. Reported findings in occupational settings, including fatigue, reduced attention, and subtle cognitive changes, are suggestive but insufficient to establish causality (Griffin, 1998). These effects may be influenced by confounding factors such as noise exposure, physical workload, and psychosocial stress. In addition, heterogeneity in vibration characteristics—particularly frequency, amplitude, and exposure duration—limits comparability across studies and complicates the identification of clear exposure–response relationships.

An important consideration is the context-dependent nature of vibration effects. While chronic occupational exposure may be detrimental, experimental evidence indicates that controlled WBV can exert neuroprotective effects under specific conditions. For example, WBV has been shown to attenuate brain damage and modulate neuroinflammatory responses following TBI in experimental models (Chen et al., 2022). This apparent duality underscores the importance of exposure parameters, suggesting that the biological impact of vibration depends on intensity,

duration, and cumulative exposure. Distinguishing between harmful chronic exposure and controlled therapeutic application is therefore essential for accurate risk interpretation.

Despite these limitations, the convergence of mechanistic insights and indirect occupational observations suggests that WBV warrants further investigation as a potential systemic stressor with neurological implications. Current occupational health frameworks predominantly address musculoskeletal risks and do not adequately incorporate CNS-related outcomes, representing a significant gap in risk assessment (Griffin, 1998).

From a preventive perspective, reducing vibration exposure remains fundamental. Engineering interventions, including improved seat suspension systems, vibration-damping technologies, and optimized equipment design, should be prioritised to minimize transmission of mechanical forces. Regular maintenance of machinery and ergonomic optimization—such as appropriate seating alignment and posture—may further reduce exposure.

Occupational health strategies should incorporate systematic exposure monitoring and adherence to established standards such as ISO 2631. However, these frameworks may require revision to account for potential neurological effects. Limiting cumulative exposure through task rotation, workload management, and scheduled rest periods may help mitigate long-term risk.

The integration of neurofunctional assessment into occupational health evaluations represents a promising approach for early detection. Sensitive tools, including digital and virtual reality–based platforms, may enable the identification of subtle cognitive and functional impairments not captured by conventional methods (Bogdanova et al., 2024; Reneker et al., 2025; Salawu et al., 2026). In parallel, oxidative stress biomarkers may provide early indicators of subclinical alterations, although their clinical applicability in occupational settings requires further validation (Ionescu et al., 2025; Ismail et al., 2020; Maciejczyk et al., 2020; Nijakowski et al., 2024; Jiao et al., 2024). Furthermore, these biomarkers are not specific to WBV-related injury and may be influenced by age, lifestyle factors, comorbidities, and other occupational exposures.

Future research should prioritise well-designed human studies integrating quantitative exposure assessment with longitudinal neurological and biochemical outcomes. Prospective cohort studies in high-risk occupational groups are particularly needed to clarify causal relationships and establish exposure thresholds.

In conclusion, although direct evidence remains limited, the available data support a biologically plausible link between chronic WBV exposure and subclinical CNS effects. Addressing this potential risk through targeted research, improved monitoring strategies, and updated occupational guidelines may contribute to more comprehensive protection of exposed populations.

8. Limitations

This review has several limitations. First, the proposed framework relies largely on indirect evidence derived from traumatic brain injury research, hand–arm vibration studies, and experimental models rather than direct human WBV studies. Second, the included studies are heterogeneous in design, exposure characteristics, and outcome measures. Finally, no quantitative analysis was performed due to the conceptual nature of this narrative review.

9. Conclusions

Whole-body vibration (WBV) remains a common occupational exposure traditionally associated with musculoskeletal disorders; however, the evidence synthesised in this review raises the possibility that its biological impact may extend to the central nervous system. Converging mechanistic data indicate several pathways implicated in traumatic brain injury may also be relevant in the context of chronic vibration exposure.

Although direct human evidence remains limited, these underlying mechanisms provide biological plausibility for the hypothesis that repeated low-intensity mechanical stress may contribute to cumulative, subclinical neuronal alterations. At the same time, experimental findings demonstrating potential neuroprotective effects of controlled WBV highlight the importance of

exposure characteristics, suggesting that the balance between adaptive and deleterious responses is determined by intensity, duration, and cumulative load.

A key implication of this framework is the need to move beyond conventional clinical endpoints toward earlier and more sensitive detection strategies. In this context, redox biomarkers—including malondialdehyde, superoxide dismutase, glutathione peroxidase, and catalase—represent promising tools for identifying subclinical oxidative imbalance and early molecular changes preceding overt neurological dysfunction. However, their application in occupational settings requires further validation, including the establishment of standardised protocols and clinically meaningful thresholds.

In parallel, advances in neurofunctional assessment—particularly the use of digital platforms and virtual reality–based tools—offer new opportunities to detect subtle cognitive and sensorimotor alterations that may not be captured by traditional evaluation methods. The integration of such approaches into occupational health practice may enhance early detection and improve risk stratification in exposed populations.

Taken together, these considerations highlight a critical gap in current occupational health paradigms, where potential neurological effects of WBV remain insufficiently addressed. Future research should adopt integrative designs combining exposure assessment, biomarker analysis, and advanced functional evaluation in longitudinal human studies. Further human studies are needed to determine the clinical relevance of WBV-related neurological effects and to guide future monitoring strategies and occupational safety recommendations.

Data Availability Statement

The data supporting the conclusions of this article are included within the article and its references. No new datasets were generated or analysed for this study.

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