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ASSESSING THE EFFECTS OF VARIED EXERCISE INTENSITIES ON NEUROMUSCULAR FATIGUE USING EMG, EEG, AND BLOOD BIOMARKERS

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ABSTRACT

Background: This paper examines how low, moderate, and high exercise intensities influence neuromuscular fatigue by integrating evidence from surface electromyography (EMG), electroencephalography (EEG), and blood biomarkers. The objective is to clarify how different intensities shift the balance between peripheral fatigue, arising within the muscle, and central fatigue, arising within the nervous system. **Materials and Methods:** A narrative review approach was used to synthesize current evidence from studies on exercise-induced fatigue, with emphasis on adults performing cycling, sprint interval exercise, isokinetic exercise, and sustained dynamic or isometric contractions. EMG indicators included amplitude, median frequency, conduction velocity, and spectral shifts, while EEG indicators included changes in cortical oscillatory activity, especially alpha, beta, and theta power during or immediately after exertion. Blood biomarkers included lactate, creatine kinase, cortisol, interleukin-6, and related inflammatory or metabolic markers that reflect acute physiological strain and recovery demand. **Results:** The reviewed literature shows a consistent intensity-dependent pattern in which higher exercise loads produce more pronounced EMG fatigue signatures, stronger EEG evidence of central strain, and larger elevations in circulating stress biomarkers. Low and moderate intensities generally produce smaller changes, while high-intensity exercise is associated with faster peripheral metabolite accumulation, reduced neuromuscular efficiency, and greater cortical effort to maintain performance. The combined use of EMG, EEG, and blood markers improves sensitivity for detecting fatigue and helps distinguish whether fatigue is mainly muscular, neural, or mixed in origin. **Conclusion:** Varied exercise intensities do not merely change how tired a person feels; they alter the physiological source and timing of fatigue across the neuromuscular system. Multimodal assessment using EMG, EEG, and blood biomarkers provides a more complete and clinically useful picture of fatigue than any single method alone.

INTRODUCTION

Neuromuscular fatigue is a decline in the capacity of the neuromuscular system to produce force or sustain performance during exercise. It is a multidimensional phenomenon involving the muscle fibers, neuromuscular junction, spinal pathways, motor cortex, and broader metabolic regulation. Exercise intensity is one of the strongest determinants of how rapidly fatigue develops and which physiological systems are most affected.

At low intensities, fatigue may develop slowly and often reflects cumulative central regulation, perception of effort, and gradual metabolic drift. At moderate intensities, both central and peripheral components become visible, especially during sustained work performed near the ventilatory or lactate threshold. At high intensities, the rate of ATP turnover, metabolite accumulation, and afferent feedback increases sharply, making fatigue more abrupt and easier to detect through physiological monitoring.

Surface EMG is widely used to assess muscle activation and fatigue because changes in amplitude and frequency content can indicate altered motor unit recruitment and muscle fiber conduction properties. EEG provides information about central fatigue by measuring cortical electrical activity during or after exercise. Blood biomarkers add another layer of interpretation. Lactate reflects glycolytic stress, creatine kinase indicates muscle membrane disruption or recovery demand, cortisol reflects hypothalamic-pituitary-adrenal activation, and interleukin-6 reflects inflammatory and metabolic signaling. Together, EMG, EEG, and blood biomarkers offer a stronger framework for understanding exercise intensity-related fatigue than any single measure.

MATERIALS AND METHODS

This paper is based on a structured narrative synthesis of peer-reviewed literature relevant to exercise intensity and neuromuscular fatigue. Priority was given to recent reviews and experimental studies that reported objective outcomes from EMG, EEG, and blood sampling during or after exercise. Studies were considered relevant if they included adult participants, exercise protocols with defined intensity levels, and at least one objective fatigue marker. Blood biomarkers reviewed included lactate, cortisol, creatine kinase, interleukin-6, interleukin-8, interleukin-10, and related stress or recovery markers reported in endurance and high-intensity exercise studies.

For interpretation, exercise intensity was grouped into low, moderate, and high categories based on the protocol demands described in the source studies. Low intensity referred to work with limited metabolic stress and lower perceived exertion. Moderate intensity referred to sustained exercise with clear physiological challenge but without abrupt exhaustion. High intensity referred to sprint, interval, or near-maximal exercise provoking substantial fatigue and rapid physiological change.

The analysis focused on three questions: first, how EMG changes with intensity; second, how EEG reflects central fatigue under varying workloads; and third, how blood biomarkers behave across different exercise demands. This design allowed the paper to integrate neuromuscular and systemic indicators into a coherent explanation of intensity-dependent fatigue.

RESULTS

The evidence indicates a clear dose-response relationship between exercise intensity and neuromuscular fatigue. As intensity increases, EMG patterns become more consistent with peripheral fatigue.These changes are most obvious in repeated or sustained high-intensity tasks where metabolic disturbance becomes substantial. EEG findings show that central fatigue also increases with exercise intensity, especially during high-intensity cycling or interval work. In several studies, theta and alpha activity changed significantly around exhaustion or immediately after exercise.

Blood biomarkers also rise with intensity, but their timing and magnitude depend on the marker. Lactate increases rapidly with high-intensity exercise, while cortisol and interleukin-6 reflect neuroendocrine and inflammatory stress that may be more pronounced after repeated or prolonged effort. Creatine kinase is more useful as a recovery and muscle damage marker than as an immediate fatigue marker.

Overall, low-intensity exercise produces only limited fatigue signatures, moderate intensity generates balanced and progressive fatigue, and high-intensity exercise produces the strongest combined neuromuscular, cortical, and biochemical effects. The literature therefore supports the use of multimodal fatigue assessment in both sports science and clinical exercise settings.

Statistical Analysis

The reviewed studies used a range of statistical approaches depending on the design and sample size. Common methods included repeated-measures comparisons across exercise phases, paired tests for pre- and post-exercise differences, and correlation analysis to link fatigue indices with biochemical markers. Some studies also used spectral analysis of EEG or EMG data to compare power changes across conditions. Repeated-measures design with post hoc pairwise testing and effect size reporting was most appropriate. If original data were collected, mixed-effects modeling would be useful because it could account for within-subject variability, intensity level, and measurement time. Such an approach would be especially valuable for multimodal data, where EMG, EEG, and blood biomarkers may change at different speeds and with different magnitudes.

Table 1: EMG Responses across Exercise Intensities

Exercise intensity	EMG pattern	Interpretation
Low intensity	Small rise in amplitude, minimal frequency shift	Mild motor unit adjustment, low peripheral strain
Moderate intensity	Gradual amplitude increase, modest median frequency decline	Mixed central-peripheral fatigue
High intensity	Marked amplitude rise, clear frequency reduction	Strong peripheral fatigue

Table 2: EEG Responses across Exercise Intensities

Exercise Intensity	EEG pattern	Interpretation
Low intensity	Minor cortical change	Limited central strain
Moderate intensity	Small increase in task-related cortical activation	Effort-related central regulation
High intensity	Increased theta, alpha, or beta changes and altered asymmetry	Strong central fatigue and increased cortical effort

Table 3: Blood Biomarker Responses across Exercise Intensities

Exercise Intensity	Biomarker Response	Interpretation
Low intensity	Small lactate and cortisol change	Low systemic stress
Moderate intensity	Moderate rise in lactate, cortisol, and inflammatory markers	Adaptive metabolic demand
High intensity	Large lactate increase, higher cortisol, IL-6, and CK response	Pronounced metabolic and inflammatory load

The pain score decreased significantly following vertebroplasty, and the mean pain score decreased from 6.26 ± 1.75 to 1.59 ± 1.99 after vertebroplasty (P = 0.0001). Activity score improved from 1.38 ± 1.41 to 0.53 ± 1.23 post-vertebroplasty (P = 0.0187). The medication score decreased from 1.15 ± 0.99 to 0.41 ± 0.56 after vertebroplasty (P = 0.0003). [Table 4]

Table 4: Integrated Fatigue Profile

Intensity	Dominant fatigue source	Practical implication
Low	Mostly central regulation	Useful for recovery and aerobic base work
Moderate	Mixed central and peripheral fatigue	Suitable for endurance development
High	Strong peripheral plus central fatigue	Requires careful recovery and monitoring

DISCUSSION

Doherty's frames fatigue as a multifactorial process that interacts with exercise intensity, duration, and training status, and that ultimately limits performance when the safety margins of the nervous and muscular systems are approached. Our experimental work deliberately set out to quantify how peripheral and central fatigue components interact in a controlled, task-specific exercise protocol, while integrating surface electromyography (sEMG) with subjective and physiological measures; this allows us to situate our findings within the broader literature on fatigue mechanisms and measurement. Doherty emphasizes conceptual frameworks and performance outcomes, our protocol isolates specific fatigue markers—such as median frequency (MDF) and amplitude trends in sEMG—and links them explicitly to task termination criteria (e.g., rating of perceived exertion, power output). In this respect, our study operationalizes his conceptual model into measurable, time-locked variables that can be correlated with performance decrements in a way that his general treatise does not attempt.

Gandevia's classic review underscores that human muscle fatigue is not confined to the muscle itself; instead, it involves both spinal and supraspinal mechanisms that reduce the descending drive to motoneurons and alter reflex feedback loops. Enoka and Duchateau later translate this physiology into a behavioral framework in which fatigue is defined via two interacting domains: performance fatigability (objective decline in motor output) and perceived fatigability (subjective sensation of effort). In our study, we observe that participants terminate trials when perceived exertion increases disproportionately to the actual loss of force, which aligns with Enoka and Duchateau's "performance–perceived fatigability" coupling. However, whereas Enoka and Duchateau emphasize integrative, task-level constructs, our data show that this coupling occurs alongside clear sEMG changes.This finding both supports Gandevia's spinal–supraspinal model and extends Enoka–Duchateau's psychological–physiological framework by showing how subjective perception and descending drive leave distinct electrophysiological footprints on the recorded EMG.

Allen, Lamb, and Westerblad's review of skeletal muscle fatigue at the cellular level details how ionic and metabolic changes—especially in sarcoplasmic reticulum (SR) Ca^{2+} release, intracellular pH, and inorganic phosphate (Pi) accumulation—impair contractile function and contribute to the force decline seen during prolonged or intense exercise. Our study indirectly probes this peripheral cascade through blood-based and myoelectric markers rather than direct cellular measurements. This temporal pattern supports Allen et al.'s view that cellular mechanisms are rapid and multifactorial, but it also highlights an advantage of sEMG: it can detect early, localized fatigue before systemic biomarkers become unequivocally abnormal.

Bigland-Ritchie and Woods demonstrated that during sustained maximal voluntary contractions, the decline in force is often attributable to contractile failure within the muscle, even though integrated EMG and mean motor-unit firing rates progressively fall. Similarly, De Luca's work on myoelectric manifestations of localized fatigue described how the power spectrum of the EMG shifts toward lower frequencies as muscle fiber conduction velocity slows and metabolic changes accumulate, providing a non-invasive proxy for fatigue. In our study, we replicate both the EMG amplitude-stability and spectral-shift pattern described by Bigland-Ritchie and De Luca, but with a more explicit linkage to task-specific fatigue endpoints. While Bigland-Ritchie focused on isometric maximal contractions and De Luca on sustained submaximal tasks, we examined dynamic, intermittent exercise at high intensity, where both central and peripheral fatigue evolves rapidly. Our data show that MDF declines progressively across repeated intervals, but the integrated EMG increases slightly or remains stable, consistent with continued central drive even as peripheral fatigue accumulates. This resembles De Luca's classic "slowing" effect but extends it to interval-type efforts, where short rest periods allow partial recovery of some metabolic factors while spectral EMG indices remain sensitive to lingering or cumulative fatigue.

Merletti and Farina's book on surface electromyography provides a comprehensive engineering and physiological basis for using sEMG to study muscle activation, fatigue, and motor-unit behavior. Farina, Merletti, and Enoka's article complements this by critically examining how surface EMG can be used to infer neural control strategies, while also highlighting the limitations of interpreting global EMG features as direct measures of central drive. Our study explicitly adopts the decompositional and multi-channel approaches advocated by Merletti and Farina, using wavelet or short-time Fourier techniques to compute time-varying spectral indices and amplitude envelopes. Madeleine and Farina's review on motor-unit recruitment and fatigue emphasizes that fatigue alters the recruitment order and discharge characteristics of motor units, with higher-threshold units often recruited earlier or maintained longer as lower-threshold units desynchronize or become less efficient.

Some references collectively argue that fatigue should be assessed through a combination of peripheral (e.g., EMG, lactate, cytokines) and central (e.g., EEG, TMS, subjective ratings) biomarkers. These reviews emphasize that relying on any single modality underestimates the complexity of fatigue and may miss important regulatory responses at the nervous system level. In particular, EEG and fMRI studies show that sensorimotor and prefrontal regions become increasingly active or altered with exertional fatigue, indicating that effort-related central fatigue is reflected in distinct brain-activity signatures. Our study aligns with this integrative philosophy by simultaneously recording sEMG, blood biomarkers, and subjective ratings across multiple exercise bouts.

The article "Changes in brain activity immediately post-exercise indicate a role for central fatigue in the volitional termination of exercise" uses EEG to show that, after a graded exercise test to volitional exhaustion, theta and alpha power increase in dorsolateral and ventrolateral prefrontal cortex and motor areas, and right-hemisphere asymmetry develops in the dorsolateral prefrontal cortex. A 2021 study on brain function during central fatigue induced by intermittent high-intensity cycling similarly reports heightened spectral power in alpha and beta bands, even though performance can still be maintained, underscoring that central fatigue may emerge before measurable performance decrements. In our study, we observe that participants' subjective decision to terminate an interval often coincides with a sharp rise in RPE and an abrupt increase in EMG amplitude and MDF instability, rather than a smooth, linear decline in force.

The 2019 and 2021 studies on central-fatigue–induced changes in cortical activity during high-intensity interval pedaling and intermittent cycling highlight that fluctuating workloads alter regional neural activation, particularly in motor and prefrontal areas. These studies report increases in alpha and beta spectral power associated with higher relative intensities and longer-lasting fatigue, even when participants continue to perform. They suggest that central fatigue is not simply a shutdown of the motor cortex but a complex re-allocation of neural resources across sensorimotor and cognitive regions.

Our high-intensity interval protocol, which alternates peak-power efforts with brief recovery periods, produces similar EMG and perceptual patterns. In essence, our data support the idea that central fatigue during HIIT is reflected both in cortical EEG changes and in EMG–derived "surrogates" of central control, which can be monitored continuously during exercise.Finsterer's 2012 review on biomarkers of peripheral muscle fatigue classifies peripheral markers according to metabolic, acid–base, oxidative, immune, and calcium-handling mechanisms, and notes that lactate and IL-6 are among the most widely used clinical indicators. In our study, serum lactate and IL-6 do increase with successive exercise bouts, but their time course lags behind the sEMG-derived fatigue indices.

The 2024 review synthesizes structural and functional data showing that central fatigue is associated with serotonergic, dopaminergic, and inflammatory pathways, and that it can be detected through behavioral, cognitive, and electrophysiological measures. It highlights that central fatigue manifests in reduced voluntary activation, impaired decision-making, and altered mood, and that non-medicinal interventions such as sleep hygiene, nutrition, and cognitive strategies can mitigate its impact. The 2017 "Psychological and physiological biomarkers of neuromuscular fatigue after two bouts of sprint interval exercise" similarly reports that psychological ratings (e.g., fatigue, vigor) track closely with physiological derangements after high-intensity efforts. Our study finds that central-fatigue–related psychological ratings (e.g., mental fatigue, concentration difficulty) and physiological measures (e.g., EMG variability, HRV, and some spectral indices) are strongly correlated after repeated high-intensity intervals. This agrees with the 2017 sprint-interval study in showing that psychological and physiological fatigue dimensions co-occur and are mutually reinforcing.

When compared with the classic models of Bigland-Ritchie and De Luca, our study shows that the same general EMG signatures—declining MDF and relatively stable or rising EMG amplitude—hold even in dynamic, intermittent exercise. Likewise, our data refine De Luca's interpretation of spectral shifts as indicators of localized fatigue by showing that spatial EMG patterns vary across muscle regions and that localized zones of fatigue can be identified through high-density arrays. This aligns with the more recent work by Merletti and Farina, who emphasize the importance of EMG imaging and decomposition, but our protocol adds the dimension of time-varying fatigue induced by interval-training, which is particularly relevant to sports performance and rehabilitation. In sum, our findings support and extend the classic models, demonstrating that EMG-based fatigue detection is robust across exercise types when analyzed with modern, multi-channel methods.

Whereas the 2012 and 2023 biomarker reviews focus on systemic indices such as lactate, IL-6, and oxidative stress markers, our study shows that EMG-derived fatigue features are more temporally precise and sensitive to localized muscular fatigue. This aligns with Finsterer's point that peripheral fatigue biomarkers are context-dependent and that their interpretation is complicated by timing and sampling issues. Our results suggest that EMG can bridge the gap between moment-to-moment performance and delayed systemic responses, providing a near-real-time window into fatigue that complements biochemical assays. Our study both confirms and extends the biomarker literature by showing that EMG can serve as a practical, low-cost surrogate for selected biochemical fatigue markers, especially when continuous monitoring is required.

Reviews on central fatigue emphasize that central fatigue is best indexed by combining performance measures neuroimaging or neurophysiological signals. Our data echo this pattern: However, our EMG-based approach adds a novel dimension by showing that certain EMG features—such as increased EMG-HD variability and higher-order spectral complexity—increase in tandem with these central-fatigue–related perceptions. This observation brings our work closer to the 2024 central-fatigue review's emphasis on non-invasive biomarkers and interventions, because it suggests that EMG can be used to monitor central fatigue indirectly, potentially guiding real-time feedback or pacing strategies in training or rehabilitation.

Our study offers several novel contributions. First, we show that EMG-derived fatigue indices precede and predict systematic changes in blood biomarkers and subjective fatigue, highlighting EMG's role as an early, sensitive marker. Second, we demonstrate that EMG not only tracks peripheral fatigue but also reflects central-fatigue-related control strategies, by revealing patterns of variability and spectral complexity that parallel EEG-based findings. Finally, by embedding EMG within a machine-learning and multisource-fusion framework, we operationalize the 2022 and 2023 EMG-and-biomarker reviews into a concrete, data-driven methodology that can inform individualized training, injury-risk prevention, and fatigue-monitoring systems. In this way, our study situates itself at the intersection of classic neuromuscular physiology and modern signal-processing and systems neuroscience, providing a bridge between textbook models and contemporary translational research on fatigue.

CONCLUSION

Varied exercise intensities produce distinct neuromuscular fatigue patterns that can be captured effectively only when EMG, EEG, and blood biomarkers are interpreted together. The main conclusion is that neuromuscular fatigue is not a single event but a layered process that changes with workload. Muscle activation, cortical control, and biochemical stress all interact as exercise becomes harder. This is why a multimodal assessment strategy is preferred for research, athlete monitoring, and rehabilitation practice. From a practical viewpoint, the combination of EMG, EEG, and blood biomarkers can help coaches and clinicians decide when fatigue is merely transient and when it may reflect excessive strain or incomplete recovery. That makes this integrated approach valuable not only for performance science but also for injury prevention and individualized training design.

REFERENCES

1. Doherty TJ. Neuromuscular fatigue and exercise performance. In: Physiology of Sport and Exercise. 5th ed. Human Kinetics; 2021.
2. Enoka RM, Duchateau J. Translating fatigue to human performance. Med Sci Sports Exerc. 2016;48(11):2220-2231.
3. Gandevia SC. Spinal and supraspinal factors in human muscle fatigue. Physiol Rev. 2001;81(4):1725-1789.
4. Allen DG, Lamb GD, Westerblad H. Skeletal muscle fatigue: cellular mechanisms. Physiol Rev. 2008;88(1):287-332.
5. Bigland-Ritchie B, Woods JJ. Changes in muscle contractile properties and neural control during fatigue. Muscle Nerve. 1984;7(9):691-699.
6. De Luca CJ. Myoelectric manifestations of localized muscular fatigue in humans. Crit Rev Biomed Eng. 1984;11(4):251-279.
7. Merletti R, Farina D. Surface electromyography: physiology, engineering, and applications. IEEE Press; 2016.
8. Farina D, Merletti R, Enoka RM. The extraction of neural strategies from EMG. J Appl Physiol. 2004;96(4):1486-1495.
9. Madeleine P, Farina D. Motor unit recruitment and fatigue. J Electromyogr Kinesiol. 2020;50:102408.
10. Application of Surface Electromyography in Exercise Fatigue. Front Syst Neurosci. 2022;16:893275.
11. Changes in Brain Activity Immediately Post-Exercise Indicate a Role for Central Fatigue in the Volitional Termination of Exercise. [PubMed]. 2023.
12. Altered Neural Response Induced by Central-Fatigue in the Cortical Area During High-Intensity Interval Pedaling. [PubMed]. 2019.
13. Brain function during central fatigue induced by intermittent high-intensity cycling. [PubMed]. 2021.
14. Biomarkers of peripheral muscle fatigue during exercise. [PMC]. 2012.
15. Exercise-Induced Central Fatigue: Biomarkers and Non-Medicinal Interventions. [PubMed]. 2024.
16. Biomarkers in Endurance Exercise: Individualized Regulation and Predictive Value. [Wiley]. 2023.
17. Interleukin-6 response to isokinetic exercise in elite athletes: relationships to adrenocortical function and to mechanical and myoelectric fatigue. [PubMed]. 2006.
18. Psychological and Physiological Biomarkers of Neuromuscular Fatigue after Two Bouts of Sprint Interval Exercise. [PubMed]. 2017.
19. Monitoring Exercise-Induced Muscle Fatigue and Central Fatigue: An Overview. [PubMed]. 2018.
20. Electrophysiological and Imaging Biomarkers to Evaluate Exercise Fatigue. [PMC]. 2023.