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Review

Neurobiological Effects of the Functional Magnetic Stimulation Chair: What Do We Know and What Do We Still Have to Discover? A Narrative Review

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Abstract

This narrative literature review aims to examine and hypothesize about the neurobiological effects of the Tesla Functional Magnetic Stimulation (FMS) chair through a comprehensive search of PubMed, Google Scholar, Scopus, Web of Science, and arXiv databases. A critical finding emerged: published research specifically investigating the Tesla FMS chair and its neurobiological mechanisms is extremely limited. Among the papers identified and ranked by relevance, only one study directly examined a Tesla-branded FMS chair system, which investigated a 3 Tesla electromagnetic chair for treating female urinary incontinence. The vast majority of the retrieved literature pertains to transcranial magnetic stimulation (TMS), a distinct neuromodulation technique that targets cortical brain regions rather than peripheral nervous system structures. This review also provides a detailed analysis of the available evidence on general FMS technology, contextualizes findings within the broader magnetic stimulation literature, and identifies critical gaps requiring future investigation. The limited evidence suggests FMS chairs operate through peripheral nerve depolarization and sacral neuromodulation, but comprehensive neurobiological characterization is lacking the published literature and requires more high-quality studies. This review should be interpreted as a hypothesis-generating narrative synthesis rather than as a definitive mechanistic or efficacy assessment. The comparison with TMS is used only as a conceptual framework. At the same time, the specific mechanisms of chair-based FMS remain to be directly demonstrated through dedicated peripheral, spinal, and supraspinal investigations.

Keywords

Magnetic stimulation; neuromodulation; pelvic floor disorders; urinary incontinence; neurophysiology; functional magnetic stimulation; neurobiological mechanisms

1. Introduction

Functional Magnetic Stimulation (FMS) can be framed within the broader field of non-invasive neuromodulation techniques that exploit time-varying magnetic fields to induce electric currents in neural tissue, thereby depolarizing membranes and modulating neural excitability. Although most mechanistic knowledge on magnetic neuromodulation derives from Transcranial Magnetic Stimulation (TMS), converging evidence indicates that electromagnetic induction can influence neural circuits across different anatomical levels, depending on stimulation parameters, field geometry, and target structures [1, 2]. From a biophysical perspective, magnetic stimulation induces electric fields that can trigger action potentials and activity-dependent plasticity through repeated application. Computational and experimental models have demonstrated that such stimulation can engage mechanisms analogous to synaptic plasticity, including changes in excitability and network dynamics, which may evolve as functions of stimulation intensity, frequency, and temporal

patterning [2, 3]. These principles underpin the rationale for extending magnetic stimulation beyond the cortex to peripheral and spinal neural targets. In this context, chair-based FMS systems represent a specific technological application of magnetic neuromodulation, designed to deliver repetitive electromagnetic stimulation to peripheral neuromuscular structures. At the same time, the patient remains seated and fully clothed. Although originally developed for clinical use rather than mechanistic investigation, these systems conceptually share key features with established TMS paradigms, including frequency-dependent effects and the potential induction of lasting neuromodulatory changes through repeated stimulation sessions [1, 4]. The extensive TMS literature provides important insights into how magnetic stimulation can modulate neural circuits and produce clinically meaningful effects, particularly in neuropsychiatric conditions. Decades of research have shown that TMS can alter neural activity at both local and network levels, producing measurable changes in brain function and behavior [4, 5]. A more recent paper has further highlighted that even low-intensity magnetic stimulation may induce significant neurobiological effects, challenging traditional assumptions about stimulation thresholds and expanding the conceptual framework of magnetic neuromodulation [1].

Despite these advances, the translation of mechanistic knowledge from TMS to peripheral applications such as FMS chairs remains incomplete. While theoretical models and closed-loop stimulation frameworks suggest that adaptive, parameter-specific modulation of neural circuits is feasible across different stimulation targets [3], direct evidence characterizing the neurobiological effects of FMS chair systems is sparse. As a result, the mechanisms by which FMS chairs may influence peripheral nerves, spinal reflexes, and potentially supraspinal circuits remain largely speculative.

This narrative review has been conducted to comprehensively evaluate published evidence on the neurobiological effects of the Tesla FMS chair. Given the very limited literature specifically on the Tesla FMS chair, this review also included the main evidence on other forms of magnetic stimulation, such as transcranial stimulation and functional stimulation, to highlight potential analogies and identify any similar effects from a biological and neurophysiological point of view.

It should be emphasized that analogies with TMS are used in this review only as a theoretical neurobiological reference and not as evidence of equivalent mechanisms. TMS primarily targets cortical neurons and distributed cortico-subcortical networks, whereas chair-based FMS mainly stimulates peripheral nerves, sacral roots, pelvic floor motor units, and spinal reflex circuits.

The objective is to synthesize available evidence, identify mechanistic insights, contextualize findings within the broader magnetic stimulation literature, and highlight critical knowledge gaps requiring future investigation.

2. Materials and Methods

2.1 Search Strategy

This research was designed as a narrative review, and it was carried out in accordance with the Scale for the Assessment of Narrative Review Articles (SANRA), which guarantees methodological strength, transparency, and coherence in narrative synthesis [6]. A comprehensive, non-systematic literature review was conducted between December the 2nd, 2025, and January the 7th, 2026. The literature search was executed across five databases and search platforms: PubMed/MEDLINE, Scopus, Web of Science, Google Scholar, and arXiv. A combination of controlled vocabulary and free-

text terms was used. Particularly, search strings incorporated terms related to “Tesla Functional magnetic stimulation chair”, “FMS chair”, “functional magnetic stimulation”, combined with neurobiological terms including “neurobiological effects”, “neural mechanisms”, “brain activity”, and “neuroscience”. Boolean operators and filters were applied to further refine the results.

2.2 Eligibility Criteria

Included studies encompassed original research (both preclinical and clinical), systematic, scoping, and narrative reviews relevant to the neurobiological effects of the Tesla FMS chair. Priority was given to studies addressing mechanistic and neuromodulatory pathways at peripheral and central levels; the search period ranged from database inception to January the 7th, 2026; publications with available abstracts or full text were considered eligible. Exclusion criteria were the following: articles not directly related to neurobiological effects, from non-peer-reviewed sources.

2.3 Data Extraction and Synthesis

All the relevant data from eligible studies were extracted narratively, focusing on biological and neurophysiological mechanisms underlying the effects produced by the Tesla FMS chair. The reference list of each of the included articles was also screened for possible relevant sources. Being a narrative review, this paper provides just a conceptual description of the available evidence; therefore, no systematic research nor quantitative synthesis (meta-analysis) was attempted.

2.4 Quality Assurance

To ensure methodological soundness, the literature selection and the synthesis process were independently cross-checked against SANRA recommendations. Emphasis was placed on scientific reasoning, critical analysis, and balanced reporting, while avoiding selective citation or overinterpretation. All these aspects are shown in Table 1.

Table 1 Adherence of this review to the SANRA domains.

SANRA Domain	Requirement	Adherence in this Review
1. Justification of the article’s importance for the readership	The topic should be relevant and timely.	Functional Magnetic Stimulation chairs are increasingly used in clinical practice for pelvic floor disorders; however, their neurobiological mechanisms remain poorly characterized. Addressing this gap is highly relevant for clinicians and researchers seeking mechanistically informed neuromodulation therapies.
2. Statement of concrete aims or formulation of questions	Clear articulation of the review’s purpose.	The aim of this narrative review is explicitly stated: to synthesize available evidence on the neurobiological effects of the Tesla FMS chair, contextualize findings within the broader magnetic stimulation literature, and identify critical mechanistic knowledge gaps.

3. Description of the literature search	Transparent description of sources and search approach.	A comprehensive narrative search was conducted across PubMed/MEDLINE, Scopus, Web of Science, Google Scholar, and arXiv, using predefined terms related to Tesla FMS chairs, functional magnetic stimulation, and neurobiological mechanisms, with clearly defined inclusion and exclusion criteria.
4. Referencing	Appropriate, comprehensive, and up-to-date references.	References include peer-reviewed clinical and preclinical studies, narrative and systematic reviews, and foundational neuroscience literature on magnetic stimulation, prioritizing recent and high-impact publications where available.
5. Scientific reasoning	Logical organization and critical interpretation of findings.	Evidence is organized across conceptual domains (direct FMS chair evidence, peripheral neuromodulation, spinal and supraspinal mechanisms, and plasticity-related processes), with critical discussion of limitations, speculative mechanisms, and comparison with established TMS literature.
6. Appropriate presentation of data	Clear structure and synthesis of material.	Findings are presented in a structured narrative form with thematic sections and subsections; mechanistic hypotheses are clearly distinguished from empirically validated evidence, and key limitations and future research directions are highlighted.

FMS, Functional Magnetic Stimulation; SANRA, Scale for the Assessment of Narrative Review Articles; TMS, Transcranial Magnetic Stimulation.

3. Results

The initial search yielded 250 papers across all databases. After deduplication, 108 papers remained. These papers were subsequently ranked by three reviewers (S.C.L., G.F., F.Q.), highlighting those specific to the research query regarding neurobiological effects of the Tesla FMS chair. The top papers by relevance score (n = 37) were selected for detailed analysis and synthesis in this review. Any disagreements were resolved by consulting the opinions of two additional expert reviewers (M.M. and A.B.). The results have been summarized in two paragraphs, which allow us to organize and highlight the main findings and are mentioned below.

3.1 Overview of Magnetic Stimulation Technologies

3.1.1 Transcranial Magnetic Stimulation (TMS)

Transcranial Magnetic Stimulation (TMS) is a well-established non-invasive brain stimulation technique that uses rapidly changing magnetic fields to induce electrical currents in cortical neurons. TMS has been extensively studied for both research and therapeutic applications, particularly in psychiatry and neurology. The neurobiological effects of TMS are well documented and include

modulation of cortical excitability, alterations in functional connectivity, neurotransmitter release, and the induction of neuroplastic changes [7-9].

Repetitive TMS (rTMS) protocols can induce lasting changes in neural activity through mechanisms analogous to long-term potentiation (LTP) and long-term depression (LTD). High-frequency rTMS (≥5 Hz) typically increases cortical excitability, while low-frequency rTMS (≤1 Hz) generally decreases it [10, 11]. These effects are mediated by multiple mechanisms including modulation of GABAergic and glutamatergic neurotransmission, NMDA receptor-dependent plasticity, and activation of molecular pathways involving brain-derived neurotrophic factor (BDNF) and immediate-early genes [12-14].

As a consequence, the clinical applications of these effects have focused primarily on neurological and psychiatric pathologies, with highly encouraging results. A meta-analysis by Bai et al. examined the effects of specific TMS protocols on modulating cortical excitability in patients with stroke and found that these protocols could be effective in facilitating motor recovery [15]. Similar high-quality studies suggested other important beneficial effects for stroke patients. Indeed, TMS has been shown to significantly help them in the treatment of common complaints, such as neglect, aphasia, and cognitive impairment [16, 17]. A meta-analysis by Pagali et al showed that TMS provides favorable evidence of improved cognition in patients suffering from mild cognitive impairment and Alzheimer's disease [18]. A recent network meta-analysis by Vinod et al suggested that TMS targeting the prefrontal cortex could be useful for patients with resistant obsessive-compulsive disorder [19]. TMS represents a valid solution for major depressive disorders both alone and in combination with antidepressant drugs [20, 21]. The translation of the neurostimulation effects described above into clinical benefits, therefore, appears well-established in the literature and represents an incentive to further investigate the effects of stimulation applied to other body regions.

3.1.2 Functional Magnetic Stimulation (FMS)

Functional Magnetic Stimulation (FMS) applies similar electromagnetic principles to TMS but targets peripheral nervous system structures rather than the brain [22]. FMS devices generate time-varying magnetic fields that penetrate tissue and induce electrical currents capable of depolarizing peripheral nerves and motor neurons. Beyond chair-based systems, FMS has been investigated in several clinical and experimental contexts, including peripheral nerve stimulation, neuromuscular activation in post-stroke rehabilitation, prevention of muscle atrophy, and facilitation of motor recovery through stimulation of spinal roots and limb muscles [23, 24]. Specifically, focal and repetitive peripheral magnetic stimulation protocols have been applied directly over limb muscles and peripheral nerves to enhance neuromuscular excitability, modulate spinal reflex pathways, reduce spasticity, and promote motor recovery in neurological and musculoskeletal conditions. These applications demonstrate that FMS can exert neuromodulatory effects beyond pure muscle strengthening, engaging afferent pathways and sensorimotor integration mechanisms [25, 26].

Compared with other forms of peripheral magnetic stimulation, chair-based FMS shares the capacity to recruit mixed afferent and efferent pathways and to induce muscle contractions without contact electrodes. However, important differences should be acknowledged. Focal or repetitive peripheral magnetic stimulation is usually applied over selected peripheral nerves or limb muscles, allowing more anatomically targeted stimulation. In contrast, FMS chair systems deliver a broader

electromagnetic field to the pelvic and sacral region. Consequently, evidence from non-chair peripheral magnetic stimulation supports the general biological plausibility of peripheral neuromodulation. Still, it cannot be directly transferred to chair-based pelvic protocols without dedicated mechanistic studies.

Unlike TMS, which requires precise coil positioning over specific cortical regions, FMS chair systems deliver broader field exposure to pelvic and sacral regions while patients remain seated and fully clothed, thereby reducing variability related to electrode placement or skin-electrode impedance, so it represents a specific form of functional magnetic stimulation as it is applied to a peculiar region and is not affected by potential barriers between the magnetic field generator and the body.

The fundamental biophysical mechanism involves Faraday's law of electromagnetic induction: a changing magnetic field induces an electric field in conductive tissue (Figure 1). When this induced electric field exceeds the threshold for neural depolarization, action potentials are generated in nerve fibers and muscle tissue [27]. The depth of penetration and spatial distribution of the induced electric field depend on magnetic field strength, pulse frequency, and coil geometry.

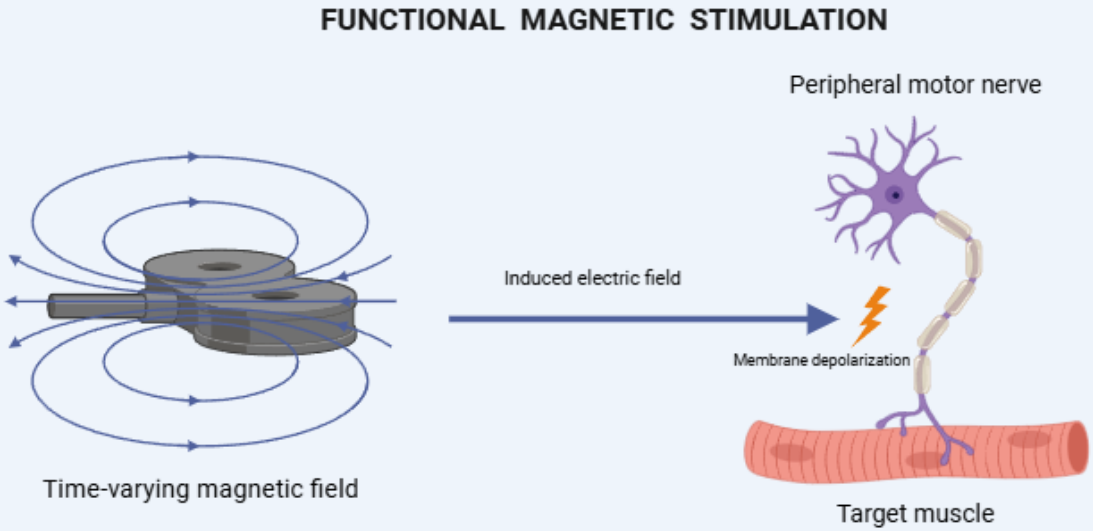

Figure 1 Biophysical principle of Functional Magnetic Stimulation.

It is critical to distinguish between TMS and FMS chair technologies, as they target fundamentally different neural structures and operate through distinct anatomical pathways (Figure 2). TMS directly stimulates cortical neurons in the brain, affecting cognitive, motor, and affective functions by modulating cortical circuits and cortico-subcortical networks [28, 29]. In contrast, FMS chairs target peripheral structures including sacral nerve roots (S2-S4), pudendal nerve branches, pelvic floor muscles, and associated autonomic pathways [30]. Importantly, stimulation of spinal and sacral nerve roots using non-chair-based magnetic stimulation has been previously explored in experimental and clinical settings, particularly in the context of lower urinary tract dysfunction and reflex modulation. These earlier applications provide a conceptual and neurophysiological foundation for current chair-based FMS systems, although they typically employed focal coils and targeted stimulation rather than broad-field exposure [31].

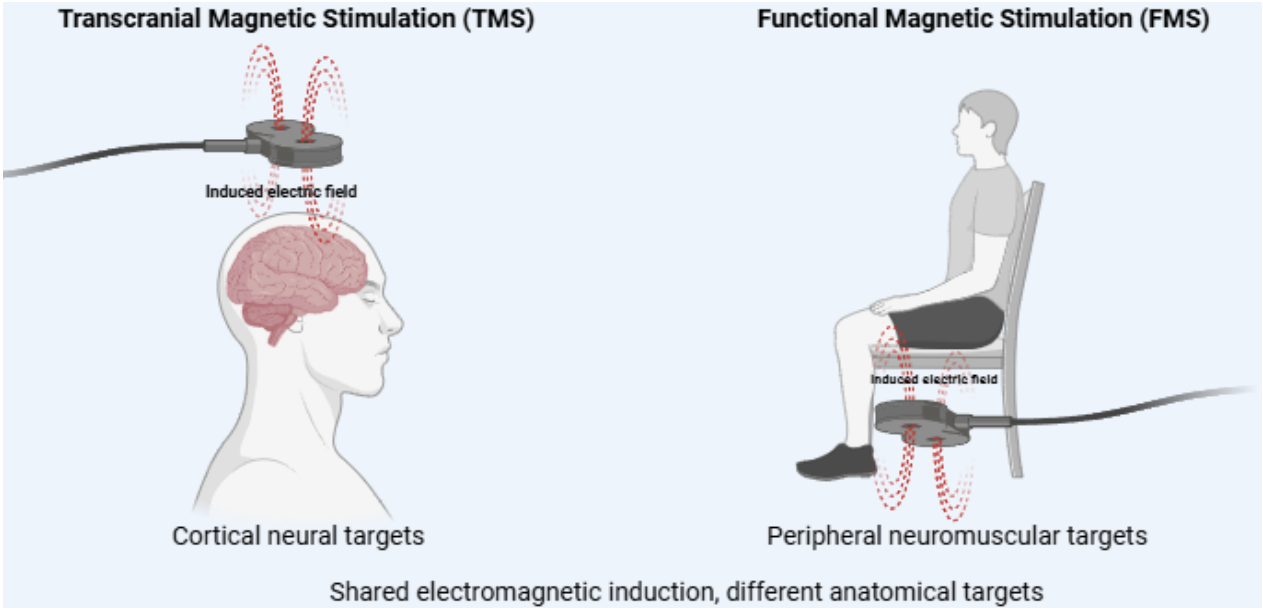

Figure 2 Conceptual comparison between Transcranial Magnetic Stimulation and Functional Magnetic Stimulation chair systems.

The neurobiological effects of TMS have been extensively characterized using neuroimaging techniques including functional MRI (fMRI), electroencephalography (EEG), positron emission tomography (PET), and magnetoencephalography (MEG) [28, 32]. These studies have revealed TMS-induced changes in regional cerebral blood flow, glucose metabolism, neurotransmitter receptor availability, functional connectivity patterns, and oscillatory brain dynamics [23]. By contrast, neurobiological characterization of FMS chair effects using comparable neuroimaging or electrophysiological methods is notably absent from the published literature.

3.2 Tesla FMS Chair and Its Proposed Neurobiological Effects

The only identified clinical study directly investigating a Tesla-branded FMS chair system was conducted by Braga et al. (2022), who examined the efficacy of a 3 Tesla electromagnetic chair for treating female urinary incontinence [33]. This prospective study included 35 patients with stress urinary incontinence (SUI) and 40 with overactive bladder (OAB) symptoms. Participants received 20-minute FMS treatments twice weekly for 8 weeks.

Treatment parameters were condition-specific: SUI patients received stimulation at 35 Hz with a 12-second duration and 300-microsecond pulse width, while OAB patients received 10 Hz frequency with 12-second duration and 250 microsecond pulse width. Clinical outcomes were assessed using validated questionnaires including the Patient Global Impression of Improvement (PGI-I) Scale, Urogenital Distress Inventory (UDI-6), Incontinence Impact Questionnaire (IIQ-7), International Consultation on Incontinence Questionnaire-Short Form (ICIQ-SF), and Overactive Bladder Questionnaire Short Form (OAB-q SF).

Results demonstrated subjective cure rates of 47% for SUI and 50% for OAB, with improvement rates of 68.3% and 70%, respectively. These findings suggest potential therapeutic efficacy, though the study lacked a control group and relied exclusively on subjective patient-reported outcomes without objective neurophysiological measurements [33].

Braga et al. proposed several neurobiological mechanisms underlying FMS chair effects, though these remain largely theoretical given the absence of direct neurophysiological measurements in their study [33]. Among the proposed mechanisms, the first one is peripheral nerve depolarization. In fact, FMS induces controlled depolarization of peripheral nerves through electromagnetic induction, generating electrical activity that activates terminal motor nerve fibers and motor end plates. This process leads to pelvic muscle contraction, which may build muscle strength and endurance over repeated treatment sessions [33]. The second possible effect is represented by sacral neuromodulation. Indeed, the electromagnetic field targets sacral nerve roots S2-S4, which provide sensory and motor innervation to the bladder, urethra, and pelvic floor muscles. Stimulation of these roots is proposed to modulate reflex pathways controlling bladder function [33]. Finally, FMS is supposed to determine modulation of afferent and efferent pathways. On one side, FMS targets afferent branches of the pudendal nerve, which carry sensory information from the pelvic region to the spinal cord and brain. Activation of these afferent pathways is hypothesized to inhibit detrusor muscle activity through central reflex mechanisms, potentially suppressing involuntary bladder contractions in OAB [33]. On the other hand, stimulation of efferent nerve branches strengthens pelvic floor muscles and urethral sphincters. This enhanced muscle tone may improve continence through the "guarding reflex", whereby increased urethral pressure prevents urine leakage during activities that increase intra-abdominal pressure [33]. As a result, the combination of modulation of afferent and efferent pathways is proposed to inhibit detrusor muscle overactivity, thereby reducing urgency and frequency symptoms in OAB patients [33]. Although these mechanisms remain speculative, as the study did not employ neurophysiological recordings, neuroimaging, or electromyographic measurements to directly verify these proposed pathways, they are also discussed and deepened in two important systematic reviews on magnetic stimulation for treating pelvic floor disorders, providing a conceptual framework for understanding its putative neurophysiological effects.

The review by Lukanović et al. was published in 2021 and analyzed clinical studies on magnetic stimulation for urinary incontinence and other pelvic floor dysfunctions, emphasizing that FMS should not be regarded solely as a passive muscle-strengthening modality, but rather as a form of noninvasive neuromodulation [34]. According to this paper, time-varying magnetic fields induce electrical currents in deep pelvic tissues in accordance with Faraday's law, leading to depolarization of peripheral nerves (particularly the pudendal nerve and sacral nerve roots S2-S4) rather than direct muscle fiber stimulation. Repeated activation of motor nerve terminals and motor end plates is proposed to enhance pelvic floor muscle strength and endurance. At the same time, simultaneous stimulation of sensory afferents may improve proprioceptive input and voluntary motor control. Importantly, the authors highlight the potential modulation of spinal reflex circuits involved in bladder-sphincter coordination, suggesting that FMS may influence detrusor activity and urethral closure mechanisms through reflex pathways rather than purely mechanical effects [34]. A more recent systematic review by Antić et al. focused specifically on urgency urinary incontinence (UUI) further expanded this neurophysiological interpretation by distinguishing frequency-dependent effects of magnetic stimulation [35]. This review proposed that low-frequency stimulation (typically 10-20 Hz) preferentially engages inhibitory reflex mechanisms, particularly relevant for UUI and OAB symptoms, whereas higher frequencies (around 35-50 Hz) primarily facilitate motor recruitment and pelvic floor strengthening, which may be more effective for stress urinary incontinence. From a neurobiological perspective, afferent activation of pudendal nerve fibers is hypothesized to inhibit

detrusor overactivity via central reflex pathways at the spinal and supraspinal levels. In contrast, concurrent efferent activation increases urethral sphincter tone and pelvic floor muscle contraction through the guarding reflex [35]. Additionally, the review discusses possible neuromodulatory effects on pain perception and sensory processing, invoking mechanisms analogous to spinal gate control [35]. Despite these coherent mechanistic models, both reviews underscore that current evidence remains largely indirect, as most clinical studies lack objective neurophysiological endpoints such as electromyography, urodynamics with neuro-reflex assessment, or functional neuroimaging. Consequently, while the proposed mechanisms could provide biological plausibility for the clinical effects of FMS chairs, they warrant validation through rigorously designed studies incorporating direct measures of neural and neuromuscular function (Figure 3).

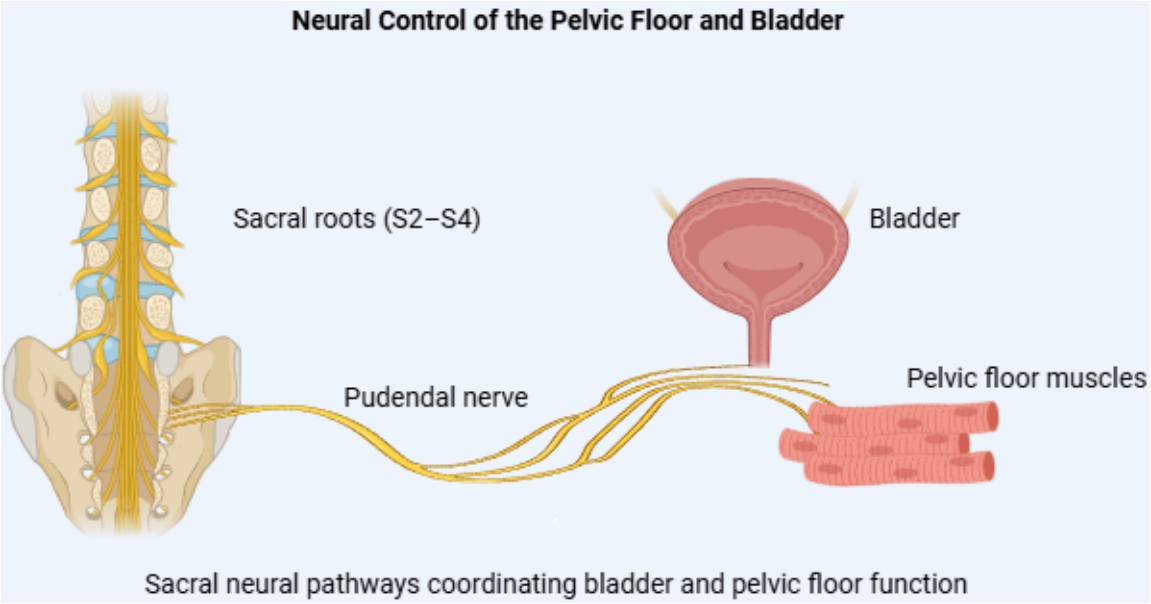

Figure 3 Neural pathways involved in pelvic floor and bladder control targeted by FMS chair stimulation.

3.3 What about Plasticity and Molecular Mechanisms?

Since direct evidence for Tesla FMS chair-induced neuroplasticity or molecular effects is currently lacking, the TMS literature can serve only as an indirect conceptual comparator. This extrapolation must be considered with caution: cortical TMS engages complex cortico-cortical and cortico-subcortical networks, whereas chair-based FMS primarily targets peripheral nerves, sacral roots, pelvic floor motor units, and spinal reflex circuits. Therefore, any discussion of plasticity in FMS should be interpreted as a hypothesis to be tested, not as evidence of mechanisms equivalent to TMS. Similar inferential approaches have been adopted in other areas of neuromodulation research, such as cancer-related pain, where mechanistic hypotheses on neural plasticity and central sensitization have been derived from broader neurobiological frameworks in the absence of direct experimental evidence [36].

In fact, the TMS literature provides extensive evidence for neuroplastic changes induced by repetitive magnetic stimulation, though it remains unknown whether similar mechanisms operate in peripheral FMS.

Firstly, repetitive TMS seems to induce LTP-like and LTD-like changes in synaptic strength, mediated by NMDA receptor activation and calcium-dependent signaling cascades [12, 14, 37]. High-frequency stimulation (≥5 Hz) typically enhances synaptic efficacy, while low-frequency stimulation (≤1 Hz) reduces it. The frequency-specific parameters used in FMS chair protocols (35 Hz for SUI, 10 Hz for OAB) suggest potential for inducing differential plasticity effects [33].

Besides, TMS could activate molecular pathways critical for neuroplasticity, including phosphorylation of ribosomal protein S6, expression of immediate early genes (Arc, c-Fos), and upregulation of Brain-Derived Neurotrophic Factor (BDNF) and its receptor Tropomyosin Receptor Kinase B (TrkB) [12, 14, 37]. These molecular changes support structural remodeling of synapses and long-term functional modifications. Whether peripheral FMS induces similar molecular changes in spinal or supraspinal circuits remains to be investigated.

Moreover, TMS seems to modulate multiple neurotransmitter systems including dopamine, serotonin, Gamma-Aminobutyric Acid (GABA), and glutamate [14, 28, 38, 39]. For example, prefrontal TMS increases dopamine release in mesostriatal and mesolimbic regions and affects serotonergic neurotransmission [39]. Analogous neurotransmitter changes may occur with FMS, particularly in spinal and brainstem circuits that control pelvic function, but direct evidence is lacking. This reasoning refers to another similar mechanism, namely the possible changes induced in functional connectivity.

Indeed, TMS alters functional connectivity within and between brain networks, as demonstrated by fMRI studies showing changes in the default mode network, central executive network, and other large-scale networks [13, 29, 33, 37]. These connectivity changes reflect reorganization of neural circuits and may contribute to therapeutic effects. Whether FMS induces a similar reorganization of spinal-supraspinal circuits that control pelvic function remains an important unanswered question.

4. Discussion

This narrative review reveals a paucity of published research specifically investigating the neurobiological effects of the Tesla FMS chair. Of all the papers identified through comprehensive database searches, only one study directly examined a Tesla FMS chair system [33], and this study focused on clinical outcomes rather than neurobiological mechanisms. No studies were identified that used neuroimaging, electrophysiological recordings, or molecular/cellular analyses to characterize FMS chair effects on neural tissue.

On the other hand, the extensive TMS literature provides a useful reference point for understanding potential FMS chair mechanisms, while also highlighting the limitations of extrapolating from cortical to peripheral stimulation.

Both TMS and FMS use electromagnetic induction to depolarize neural tissue. Both can induce frequency-dependent effects, with high-frequency stimulation generally producing excitatory effects and low-frequency stimulation producing inhibitory effects [11, 14, 33, 37]. Both may induce neuroplastic changes through repeated application, potentially involving LTP/LTD-like mechanisms [12, 14, 37].

Nevertheless, there are some important differences between TMS and FMS chair. While TMS directly stimulates cortical neurons and modulates complex cognitive, motor, and affective functions through cortico-cortical and cortico-subcortical networks [13, 29], FMS chairs target peripheral structures and modulate relatively simpler reflex circuits. However, supraspinal

involvement is possible [40]. TMS effects have been extensively characterized using neuroimaging and electrophysiology, while FMS chair effects remain largely uncharacterized. TMS requires precise coil positioning over specific cortical targets, while FMS chairs deliver broader-field exposure to pelvic/sacral regions.

The mechanistic insights from TMS research suggest hypotheses for FMS chair effects but cannot substitute for direct investigation. The distinct anatomical targets and functional outcomes of these technologies necessitate dedicated research programs to characterize the neurobiological mechanisms of FMS chair.

To clarify the critical differences and possible areas of overlap among the main magnetic stimulation modalities discussed in this review, a comparative summary is provided in Table 2.

Table 2 Comparative summary of TMS, peripheral magnetic stimulation, and chair-based FMS.

Feature	**TMS**	**Peripheral magnetic stimulation**	**Chair-based FMS**
Main target	Cortical neurons and cortico-subcortical networks	Peripheral nerves, nerve roots, and limb muscles	Sacral roots, pudendal pathways, pelvic floor muscles, and pelvic reflex circuits
Field selectivity	Relatively focal, depending on coil type and neuronavigation	Variable; often more focal than chair-based systems	Broad pelvic and sacral field exposure
Mechanistic evidence	Extensive neuroimaging, electrophysiological, and molecular evidence	Growing evidence on neuromuscular activation, spinal excitability, reflex modulation, and motor rehabilitation	Limited direct mechanistic evidence; mainly inferred from clinical outcomes and peripheral neuromodulation models
Clinical applications	Depression, stroke recovery, cognitive and neuropsychiatric disorders	Spasticity, motor recovery, peripheral neuromuscular rehabilitation	Urinary incontinence, overactive bladder, pelvic floor dysfunctions
Plasticity evidence	Well documented, including LTP/LTD-like effects, neurotransmitter modulation, and network reorganization.	Partially supported, especially for spinal excitability and sensorimotor integration	Hypothetical; requires direct neurophysiological and neuroimaging confirmation.
Critical interpretation	Established neuromodulation technique with validated cortical mechanisms	Useful comparator for peripheral neuromodulation	Promising but still empirically driven; mechanisms remain incompletely validated.

Thus, addressing the identified evidence gaps requires a systematic research program incorporating multiple methodological approaches.

Functional MRI studies should examine the effects of the FMS chair on brain activity in regions that control pelvic function (periaqueductal gray, pontine micturition center, insula, anterior cingulate cortex, prefrontal cortex). PET or SPECT studies could assess changes in cerebral blood flow, glucose metabolism, and neurotransmitter receptor availability. Such studies would reveal whether FMS induces supraspinal reorganization analogous to the effects of TMS on cortical networks [8, 28, 38]. Similarly, EEG recordings could characterize cortical responses to FMS chair stimulation, examining evoked potentials and oscillatory dynamics. Peripheral nerve conduction studies and electromyography could assess changes in nerve excitability and neuromuscular function. Spinal reflex testing could quantify modulation of sacral reflex circuits [40].

Then, some molecular and cellular studies should be needed. Animal model studies should examine molecular markers of plasticity (immediate-early genes, BDNF, synaptic proteins) in the spinal cord and brainstem following FMS. Histological analyses could assess structural changes in neural circuits. Such studies would determine whether FMS induces plasticity mechanisms similar to those observed with TMS [14, 41, 42].

Since the FMS chair is a physiotherapy device, a systematic investigation of stimulation parameters (frequency, intensity, pulse width, treatment duration, session frequency) is needed to optimize therapeutic efficacy and understand the mechanism of action. Frequency-specific effects should be examined, given evidence from TMS that different frequencies produce distinct neurobiological effects [10, 11, 37].

Moreover, longitudinal studies with extended follow-up periods should assess the durability of effects and examine whether repeated FMS treatment induces lasting neuroplastic changes; in particular, randomized controlled trials should incorporate neurobiological outcome measures alongside clinical endpoints.

4.1 Limitations

The limitations of this study are methodological. The paucity of literature directly related to the FMS chair made it impossible to conduct a systematic review. On the other hand, we believe this narrative review is essential because it allows us to delve deeper, albeit theoretically and speculatively, into the possible neurobiological effects of the Tesla chair, which is now commonly used in clinical practice, especially in the treatment of pelvic floor disorders and musculoskeletal pathologies.

Another limitation is that this review was intentionally designed as a narrative and hypothesis-generating synthesis rather than as a structured scoping review. Therefore, it does not aim to map the entire field of all available magnetic stimulation devices or protocols. Future scoping reviews should expand the scope beyond a single branded chair-based system and systematically compare Tesla FMS with other chair-based FMS devices and peripheral magnetic stimulation techniques. Nevertheless, the present review addresses a clinically relevant gap by critically discussing the limited mechanistic evidence currently available for Tesla FMS chair systems.

5. Conclusion

This narrative literature review reveals a critical gap in scientific understanding of the neurobiological effects of the Tesla Functional Magnetic Stimulation chair. Despite comprehensive searches across major biomedical databases and preprint repositories, only one published study directly investigated a Tesla FMS chair system, and it focused on clinical outcomes for urinary incontinence without mechanistic investigation. The proposed neurobiological mechanisms, including peripheral nerve depolarization, sacral neuromodulation, spinal reflex modulation, and muscle strengthening, remain largely theoretical and unvalidated by direct neurophysiological or neuroimaging evidence.

The extensive literature on transcranial magnetic stimulation provides a useful theoretical framework. It suggests potential mechanisms, but the distinct anatomical targets and functional outcomes of FMS chairs necessitate dedicated research programs. Critical priorities include neuroimaging studies to assess supraspinal effects, electrophysiological characterization of neural responses, molecular and cellular studies in animal models, systematic dose-response investigations, and mechanistic clinical trials incorporating neurobiological outcome measures.

The limited evidence available suggests that FMS chairs may produce therapeutic benefits for pelvic floor disorders through peripheral neuromodulation. Still, the neural circuits, molecular pathways, and plasticity mechanisms underlying these effects remain poorly understood. Addressing these knowledge gaps is essential for optimizing treatment protocols, identifying patient populations most likely to benefit, and advancing FMS chair technology from an empirical intervention to a mechanistically grounded therapeutic approach. Future research should prioritize rigorous mechanistic investigation using state-of-the-art neuroimaging, electrophysiological, and molecular techniques to elucidate the neurobiological effects of this promising but understudied neuromodulation technology.

In this perspective, the Tesla FMS chair should currently be regarded as a promising peripheral neuromodulation approach with preliminary clinical support, but with neurobiological mechanisms that remain largely hypothetical and require direct experimental validation.

Abbreviations

BDNF	Brain-Derived Neurotrophic Factor
EEG	Electroencephalography
fMRI	Functional Magnetic Resonance Imaging
FMS	Functional Magnetic Stimulation
GABA	Gamma-Aminobutyric Acid
Hz	Hertz
ICIQ-SF	International Consultation on Incontinence Questionnaire-Short Form
IIQ-7	Incontinence Impact Questionnaire-7
LTD	Long-Term Depression
LTP	Long-Term Potentiation
MEG	Magnetoencephalography
NMDA	N-Methyl-D-Aspartate
OAB	Overactive Bladder
OAB-q SF	Overactive Bladder Questionnaire Short Form

PET	Positron Emission Tomography
PGI-I	Patient Global Impression of Improvement
rTMS	Repetitive Transcranial Magnetic Stimulation
SANRA	Scale for the Assessment of Narrative Review Articles
SUI	Stress Urinary Incontinence
TMS	Transcranial Magnetic Stimulation
TrkB	Tropomyosin Receptor Kinase B
UDI-6	Urogenital Distress Inventory-6
UUI	Urgency Urinary Incontinence

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AI-Assisted Technologies Statement

During the preparation of this manuscript the authors used ChatGPT in order to improve the language. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the contents of the published article.

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