

# The Barefoot-OCD Hypothesis: Could Grounding Through Barefoot Walking Modulate Neuroinflammation and Cognitive Rigidity in Obsessive-Compulsive Disorder?

**Abdullah Faisal Albukhari**

Faculty of Medicine, Rabigh, King Abdulaziz University, Saudi Arabia

Email: [Aabdulqaderalbukhari@stu.kau.edu.sa](mailto:Aabdulqaderalbukhari@stu.kau.edu.sa)

ORCID: <https://orcid.org/0009-0004-1482-4411>

---

## **Abstract**

### **Background:**

*Obsessive-Compulsive Disorder (OCD) is a persistent mental health condition marked by unwanted thoughts, compulsive actions, and considerable functional disruption. Despite the existence of both medication and psychotherapy options, a significant number of individuals continue to experience partial or total resistance to treatment. Recent studies indicate that factors such as neuroinflammation, irregularities in the hypothalamic-pituitary-adrenal (HPA) axis, and diminished cognitive flexibility may play a role in the pathophysiology of OCD.*

### **Objective:**

*This paper introduces an innovative hypothesis suggesting that grounding—particularly through walking barefoot on natural surfaces—might act as a non-invasive supplementary treatment for OCD by influencing essential biological and cognitive functions.*

### **Hypothesis:**

*Grounding has the potential to lower systemic and neuroinflammation, regulate cortisol levels, and improve prefrontal cortex activation via heightened somatosensory stimulation. These outcomes may aid in restoring the balance of cortico-striato-thalamo-cortical (CSTC) circuits and enhance cognitive flexibility among those with OCD.*

### **Conclusion:**

*While clinical research investigating grounding's impact on OCD has not yet been conducted, its positive safety profile and relevant mechanistic connections with recognized pathophysiological targets warrant additional exploration. This hypothesis lays the groundwork for future empirical studies and presents a promising avenue for integrative therapeutic approaches in managing OCD.*

**Keywords:** *OCD, Foot, Neuroinflammation, CSTC, cognitive*

---

## **1. INTRODUCTION**

Obsessive-Compulsive Disorder (OCD) is a persistent and debilitating mental health condition that affects around 2-3% of the worldwide population, typically manifesting during adolescence and often continuing into adulthood [1]. The disorder is characterized by ongoing obsessions and compulsions, which can result in considerable distress,

difficulties in occupational functioning, and a reduced quality of life [2]. Although the primary treatments involve selective serotonin reuptake inhibitors (SSRIs) and cognitive behavioral therapy (CBT), approximately 30-60% of individuals do not achieve complete remission, with many experiencing recurring symptoms despite receiving proper treatment [3,4].

Recent research indicates that the origins of OCD may be linked to factors beyond serotonin imbalances and structural issues within the cortico-striato-thalamo-cortical (CSTC) circuit. There is growing evidence supporting the influence of neuroinflammation, oxidative stress, and compromised neuroplasticity as significant contributors to the disorder's pathophysiology [5,6]. Increased levels of inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- $\alpha$ ), and C-reactive protein (CRP) have been observed in patients with OCD, suggesting a connection between immune system activation and both symptom severity and resistance to treatment [7,8].

Simultaneously, the practice known as grounding or earthing—where individuals make direct contact with the Earth's surface by walking barefoot on natural ground—has garnered scientific attention for its anti-inflammatory properties and effects on stress management. Controlled pilot studies indicate that grounding can reduce cortisol levels, stabilize circadian rhythms, lower pro-inflammatory cytokine levels, and enhance sleep quality [9,10]. These physiological changes may interact mechanistically with biological irregularities seen in OCD, indicating potential therapeutic implications.

This review introduces an innovative neurobiological hypothesis: that engaging in grounding through barefoot walking could mitigate OCD symptoms by addressing critical pathophysiological mechanisms such as chronic neuroinflammation, stress dysregulation, and cognitive inflexibility. By connecting research on immune-neural interactions in OCD with emerging findings on the holistic benefits of grounding practices, this hypothesis aims to encourage new avenues for integrative therapies that are low-risk.

## **2. NEUROBIOLOGY OF OCD**

### **2.1. Dysfunction of CSTC Circuitry**

Dysfunction within the cortico-striato-thalamo-cortical (CSTC) circuit is central to the pathophysiology of OCD, as this circuit regulates cognitive control, habit formation, and behavioral inhibition. Numerous functional neuroimaging studies have consistently reported hyperactivity in this circuit, particularly involving the orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), caudate nucleus, and thalamus [11]. This heightened activity is believed to disrupt the brain's ability to detect errors and filter irrelevant information, resulting in intrusive thoughts (obsessions) and repetitive actions (compulsions) [12].

Research employing PET and fMRI techniques has shown increased glucose metabolism in CSTC areas, notably in the caudate and OFC, associating this hypermetabolism with greater symptom severity. Critically, treatments that alleviate symptoms of OCD—such

as SSRIs and cognitive behavioral therapy (CBT)—seem to normalize CSTC activity, underscoring its significance as a target for therapeutic intervention [13].

## **2.2. Neuroinflammation's Role**

Emerging research points to neuroinflammation as a contributing factor in the development of OCD. Studies focusing on peripheral markers have revealed elevated levels of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- $\alpha$ ), and C-reactive protein (CRP) among both pediatric and adult populations diagnosed with OCD [14,15]. These cytokines can compromise the integrity of the blood-brain barrier, activate microglia, and disrupt neurotransmitter balance.

In vivo imaging utilizing [11C]PBR28 positron emission tomography—a marker indicating activated microglia—has shown increased glial activity within the CSTC loop in individuals with OCD when compared to control subjects, providing concrete evidence of central inflammation [16]. This observation suggests that immune processes might contribute to the persistence or worsening of CSTC dysfunction.

## **2.3. Cognitive Rigidity and Dysregulation in Prefrontal-Striatal Connectivity**

A fundamental characteristic of OCD is cognitive rigidity—the difficulty in adapting thought patterns or controlling compulsive behaviors. Neuropsychological assessments indicate that this inflexibility is linked to diminished adaptability within the dorsolateral prefrontal cortex (DLPFC) alongside disrupted connectivity between frontostriatal regions [17]. Such deficits lead to an unyielding adherence to rituals and intrusive thoughts despite their recognition as irrational.

Structural imaging findings reveal a decrease in cortical thickness within both the DLPFC and ACC as well as altered white matter integrity along frontostriatal pathways, reinforcing a possible neurodevelopmental basis for the rigidity observed in OCD patients [18].

## **2.4. Constraints of Current Treatment Options**

While SSRIs and CBT are recognized as effective therapies for OCD, a considerable number of patients remain only partially responsive or fully treatment-resistant. In cases of OCD, SSRIs typically necessitate higher dosages and extended treatment periods compared to their use in depression; their effectiveness may also be hindered by underlying inflammatory or structural issues [19].

For instances where standard treatments prove inadequate, augmentation strategies including low-dose atypical antipsychotics, glutamatergic agents, or neuromodulation techniques such as repetitive transcranial magnetic stimulation (rTMS) or deep brain stimulation (DBS) are employed; however, these approaches may come with adverse effects, substantial costs, and limited accessibility [20]. These challenges highlight an urgent requirement for additional low-risk therapeutic options that address both neuroinflammatory factors and cognitive aspects without introducing significant side effects.

### **3. GROUNDING PHYSIOLOGY AND EVIDENCE**

#### **3.1. Mechanism of Grounding: Electron Transfer and Antioxidant Effects**

Grounding, also referred to as earthing, involves making direct physical contact with the Earth's surface, typically by walking barefoot on natural elements such as soil, grass, or sand. The Earth's surface possesses a net negative electric potential, enabling the body to take in free electrons upon contact with the ground [21]. These electrons are believed to function as natural antioxidants, counteracting reactive oxygen species (ROS) and stabilizing bioelectrical systems, which may lead to decreased oxidative stress and cellular damage [22].

Research in biophysics indicates that the transfer of electrons might influence redox-regulated transcription factors and diminish inflammatory gene expression [23]. This mechanism is consistent with emerging theories regarding psychiatric disorders where redox imbalance and neuroimmune activation are pivotal.

#### **3.2. Impact on Inflammatory Markers**

Initial clinical trials and laboratory research have indicated that grounding correlates with lowered levels of inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- $\alpha$ ), and C-reactive protein (CRP) [24,25]. One study found that subjects experiencing delayed-onset muscle soreness (DOMS) who slept while grounded exhibited lower white blood cell counts and reduced pain compared to those who did not ground themselves [26].

Additionally, research involving grounded patch applications showed a decrease in blood viscosity, implying enhanced circulation and diminished inflammatory load [27]. These outcomes support the hypothesis that grounding may mitigate chronic low-grade inflammation — a characteristic feature of psychiatric conditions like OCD.

#### **3.3. Impact on HPA Axis and Cortisol**

The hypothalamic-pituitary-adrenal (HPA) axis plays a crucial role in regulating cortisol levels and stress responses; it is often dysregulated in individuals with OCD and anxiety disorders. Grounding has been observed to normalize daily cortisol rhythms, favor parasympathetic activity, and lower nighttime cortisol levels—factors that may elucidate reports of improved sleep quality and mood among those who practice grounding [28].

In a controlled study lasting eight weeks, participants who slept grounded reported lower levels of stress and fatigue alongside a healthier pattern in their cortisol secretion [29]. Given the tendency for hyperactivity within the HPA axis among OCD patients, grounding could provide restorative effects through this mechanism that buffers stress.

#### **3.4. Sensory-Cognitive Pathways**

In addition to biochemical influences, grounding activates mechanoreceptors located in the feet, stimulating the somatosensory cortex and enhancing bodily awareness. Research focused on sensorimotor neurorehabilitation has shown that walking barefoot increases

proprioceptive feedback while potentially enhancing cortical plasticity within motor pathways and prefrontal circuits [30].

This information is particularly pertinent when considering OCD since cognitive rigidity coupled with impaired flexibility can partly stem from insufficient sensory integration and prefrontal regulation. Thus, the sensory experience associated with barefoot walking may indirectly promote improvements in executive function that could help lessen repetitive or compulsive behaviors [31].

## **4. LINKING GROUNDING TO OCD PATHOPHYSIOLOGY**

### **4.1. Hypothesized Reduction in Neuroinflammation**

Research indicates that neuroinflammation plays a role in the development of OCD, suggesting that grounding might serve as a physiological method to alleviate this inflammation. Evidence shows that grounding reduces systemic levels of IL-6, TNF- $\alpha$ , and CRP—biomarkers that are elevated among individuals with OCD [32,33]. Additionally, the activation of microglia within the cortico-striato-thalamo-cortical (CSTC) circuit has been documented in OCD patients [34], indicating that the anti-inflammatory properties of grounding could potentially yield benefits for the central nervous system.

By restoring electrical homeostasis, grounding may also stabilize cellular signaling and diminish abnormal immune responses. Consequently, this practice could mitigate immune-related disturbances within neurotransmitter systems such as glutamate and dopamine, which are both associated with the manifestation of OCD symptoms [35].

### **4.2. Modulation of Cognitive Rigidity via PFC Activation**

Grounding might enhance the functioning of the prefrontal cortex (PFC) by reducing cortisol levels and increasing parasympathetic activity [36]. The PFC—particularly its dorsolateral and ventromedial areas—is crucial for cognitive flexibility, decision-making, and behavioral inhibition; all functions that are often compromised in OCD patients [37].

Research has demonstrated that somatosensory stimulation from walking barefoot on textured natural surfaces can activate PFC regions through processes involving multisensory integration. This activation may lead to improvements in set-shifting abilities, inhibition of compulsive behaviors, and decreased perseveration [38]. Such enhancements have the potential to address cognitive rigidity, a fundamental neuropsychological challenge faced by those with OCD.

### **4.3. Restoration of CSTC Balance through Stress and Sensory Input**

Chronic stress is known to heighten hyperactivity within CSTC circuits, resulting in repetitive behavioral patterns. Grounding has been shown to normalize activity in the HPA axis while lowering sympathetic tone and enhancing heart rate variability—indicators of improved stress resilience [39]. By diminishing systemic stress responses, grounding may contribute to reducing excessive cortical excitation and facilitating rebalancing within CSTC circuits.

Moreover, sensory input derived from barefoot walking activates brainstem and thalamic relay pathways, offering a mechanism to influence thalamo-cortical dynamics. This sensory engagement may support adaptive neural plasticity within networks relevant to OCD treatment [40].

#### 4.4. Integrative Model (Conceptual Framework)

We propose a conceptual framework (Figure 1) illustrating how grounding influences OCD pathophysiology through several interconnected mechanisms:

- Electron influx diminishes both systemic and neuroinflammation.
- Sensory input activates somatosensory pathways as well as prefrontal connections.
- Modulation of stress contributes to restoring balance within CSTC circuits via HPA axis regulation.

The cumulative effects of these mechanisms may lead to reduced obsessions, improved cognitive flexibility, and disruption of compulsive feedback loops.

While this model remains theoretical at present, it is consistent with emerging frameworks surrounding neuroimmune interactions and embodied cognition, underscoring the promise held by non-invasive somatic interventions in psychiatric treatment. A proposed integrative model illustrates how grounding may modulate obsessive-compulsive symptoms through its effects on inflammatory processes, CSTC circuitry, and prefrontal cortical function (Figure 1).

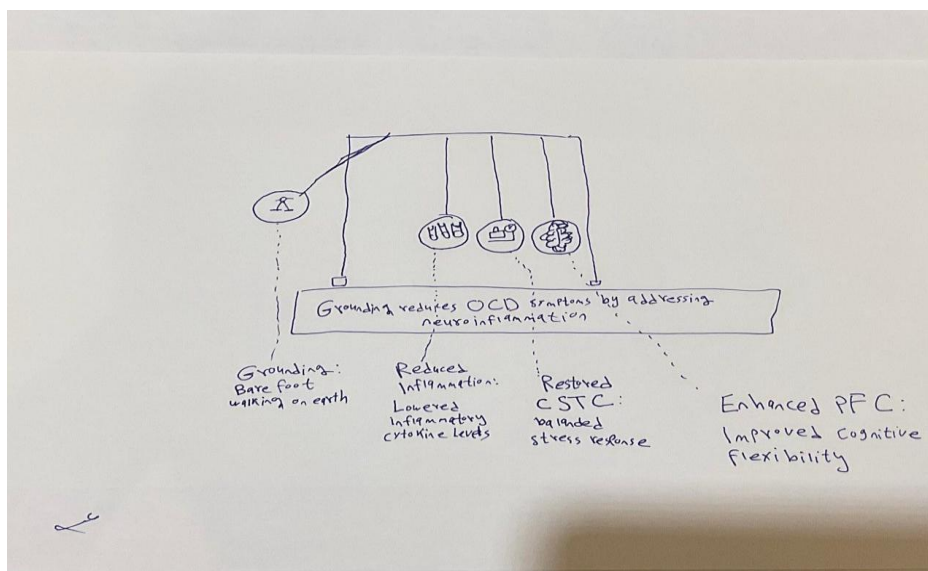


Figure 1 Conceptual model illustrating the proposed mechanism by which grounding (barefoot walking on earth) may reduce OCD symptoms.

Grounding may lower systemic inflammation, restore CSTC balance, and enhance prefrontal cognitive flexibility, ultimately contributing to symptomatic improvement.

## 5. CLINICAL IMPLICATIONS AND LIMITATIONS

While there is strong mechanistic reasoning and physiological evidence supporting grounding as an intervention for reducing inflammation and regulating neurological functions, no randomized controlled trials (RCTs) have been conducted to assess its impact on individuals with obsessive-compulsive disorder (OCD). This lack of research is particularly notable given the substantial prevalence of OCD that does not respond to treatment, highlighting the necessity for non-invasive, low-risk supplementary therapies alongside existing treatments.

Grounding has demonstrated safety and tolerability in both healthy individuals and those suffering from chronic pain, insomnia, and cardiovascular issues [41]. Trials involving direct barefoot contact or conductive systems have not reported any severe adverse effects, indicating that grounding may be a suitable option for psychiatric patients with minimal associated medical risks [42].

Nevertheless, several methodological challenges obstruct the clinical application of grounding research:

Most current studies are limited in scale, do not employ blinding methods, and lack control groups.

The physical nature of grounding practices—such as walking barefoot or using patches—makes it difficult to implement blinding procedures in placebo-controlled trial designs [43].

Variability in study protocols (including aspects like duration, type of surface used, indoor versus outdoor settings, and time of day) diminishes both reproducibility and generalizability.

Additionally, individual variations in baseline levels of inflammation, psychological distress, and sensitivity to sensory inputs may affect therapeutic outcomes; these factors need to be considered in future study designs.

Clinical trials investigating the efficacy of grounding for OCD should utilize a standardized model for exposure and incorporate objective biomarkers (e.g., IL-6, CRP, salivary cortisol). Furthermore, changes in OCD severity should be assessed using validated instruments such as the Yale-Brown Obsessive Compulsive Scale (Y-BOCS). It would also be crucial to conduct longitudinal follow-ups to evaluate whether physiological changes align with lasting improvements in symptoms.

Given its favorable safety profile and initial indications of anti-inflammatory and neurocognitive advantages, grounding warrants consideration for pilot studies as an adjunctive approach—particularly among populations experiencing significant inflammatory responses or heightened stress sensitivity.

## 6. FUTURE DIRECTIONS

The theoretical framework that connects grounding to neuroimmune modulation in obsessive-compulsive disorder (OCD) presents several promising research opportunities.

In light of the lack of clinical trials, an essential step is to develop pilot studies aimed at evaluating both the feasibility and preliminary effectiveness of grounding as a supplementary treatment alongside standard OCD therapy.

### **Key Recommendations for Future Research:**

Conduct **Randomized Controlled Trials (RCTs)** that compare grounding methods (such as barefoot walking or conductive systems) against sham grounding, ensuring well-defined protocols regarding duration, frequency, and surface type.

Incorporate validated measures of OCD severity (e.g., Y-BOCS) as primary outcomes, alongside inflammatory biomarkers (e.g., IL-6, TNF- $\alpha$ , cortisol) as secondary outcomes to objectively assess biological responses.

Utilize **neuroimaging techniques** (e.g., fMRI, PET) to investigate potential alterations in CSTC circuitry and prefrontal activation resulting from grounding interventions.

Implement stratified analyses to pinpoint subgroups that may derive the most benefit, such as those with elevated baseline inflammation, high perceived stress levels, or significant cognitive rigidity.

Explore integration with mind-body practices like mindfulness, breathwork, or movement therapy to examine possible synergistic effects on sensory processing and neuroplasticity.

Conduct longitudinal studies to evaluate the sustainability of therapeutic effects, adherence to barefoot practices over time, and overall user acceptability.

Furthermore, qualitative research could provide insights into patient perceptions, obstacles, and motivators related to grounding—particularly among OCD patients who have heightened concerns about contamination or cleanliness—factors that may influence their engagement with barefoot interventions.

In conclusion, grounding is a biologically plausible intervention that is low-cost and safe; it deserves thorough empirical investigation. Although the current hypothesis remains tentative, it establishes a solid foundation for translational research that bridges psychiatry, neuroimmunology, and integrative medicine.

## **7. CONCLUSION**

Obsessive-Compulsive Disorder (OCD) is a multifaceted psychiatric disorder that frequently resists treatment, with increasing evidence highlighting the significance of neuroinflammation, cognitive inflexibility, and abnormalities in the CSTC circuitry. Grounding—especially through activities like walking barefoot on natural surfaces—appears to be a plausible non-invasive method that may impact these pathological areas via mechanisms such as immune system modulation, stress management, and sensory-cognitive integration.

This hypothesis paper suggests that grounding might function as a complementary strategy in treating OCD by lowering inflammatory cytokines, normalizing HPA axis functioning, and improving prefrontal cortex activity. Although clinical trials have not

yet investigated this concept, the integration of physiological, psychological, and neurobiological perspectives provides strong reasoning for thorough exploration.

While grounding may not serve as a definitive solution on its own, it shows potential as part of a comprehensive integrative approach to enhance outcomes for individuals with OCD—particularly those who do not respond well to traditional therapies. Future research is crucial to confirm its effectiveness, establish suitable protocols, and clarify its role within evidence-based mental health practices.

## References

1. Ruscio AM, Stein DJ, Chiu WT, Kessler RC. The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. *Mol Psychiatry*. 2010;15(1):53–63.
2. Fontenelle LF, Mendlowicz MV, Versiani M. The descriptive epidemiology of obsessive-compulsive disorder. *Prog Neuropsychopharmacol Biol Psychiatry*. 2006;30(3):327–337.
3. Pittenger C, Bloch MH. Pharmacological treatment of obsessive-compulsive disorder. *Psychiatr Clin North Am*. 2014;37(3):375–391.
4. Fineberg NA, Reghunandan S, Brown A, Pampaloni I. Pharmacotherapy of obsessive-compulsive disorder: evidence-based treatment and beyond. *Aust N Z J Psychiatry*. 2013;47(2):121–141.
5. Benedetti F, Aggio V, Pratesi ML, Greco G, Furlan R. Inflammation and depression: a review of recent findings. *Int J Neuropsychopharmacol*. 2020;23(10):679–690.
6. Leckman JF, King RA. PANDAS and the microbiome: implications for clinical research. *J Child Adolesc Psychopharmacol*. 2016;26(7):578–585.
7. Konuk N, Tekin IO, Ozturk U, Atik L, Erdogan A, Alptekin K. Plasma levels of tumor necrosis factor- $\alpha$  and interleukin-6 in obsessive-compulsive disorder. *Mediators Inflamm*. 2007;2007:65704.
8. Attwells, S., Setiawan, E., Wilson, A., Rusjan, P., Mizrahi, R., Miler, L., Xu, C., Richter, M., Kahn, A., Kish, S., Houle, S., Ravindran, L., & Meyer, J. (2017). Inflammation in the Neurocircuitry of Obsessive-Compulsive Disorder. *JAMA Psychiatry*, 74, 833–840. <https://doi.org/10.1001/jamapsychiatry.2017.1567>.
9. Saxena S, Rauch SL. Functional neuroimaging and the neuroanatomy of obsessive-compulsive disorder. *Psychiatr Clin North Am*. 2000;23(3):563–586.
10. Maia TV, Cooney RE, Peterson BS. The neural bases of obsessive-compulsive disorder in children and adults. *Dev Psychopathol*. 2008;20(4):1251–1278.
11. Fitzgerald KD, Welsh RC, Gehring WJ, et al. Error-related hyperactivity of the anterior cingulate cortex in obsessive-compulsive disorder. *Biol Psychiatry*. 2005;57(3):287–294.
12. Sayyah M, Boostani S, Pakseresht S, et al. Increased serum interleukin-6, interleukin-1 $\beta$ , and tumor necrosis factor- $\alpha$  levels in patients with obsessive-compulsive disorder. *Psychiatry Res*. 2015;229(1–2):322–327.
13. Rodriguez CI, Bender J Jr, Marcus SM, et al. Minocycline augmentation of pharmacotherapy in obsessive-compulsive disorder: an open-label trial. *J Clin Psychiatry*. 2010;71(9):1247–1249.
14. Attwells S, Setiawan E, Wilson AA, et al. Inflammation in the brain in patients with OCD: A positron emission tomography study using [ $^{11}\text{C}$ ]PBR28. *JAMA Psychiatry*. 2017;74(8):833–840.
15. Vaghi MM, Vértes PE, Kitzbichler MG, et al. Specific frontostriatal circuits for impaired cognitive flexibility and goal-directed planning in obsessive-compulsive disorder. *Biol Psychiatry*. 2017;81(8):708–717.
16. Radua J, van den Heuvel OA, Surguladze S, et al. Meta-analytical comparison of voxel-based morphometry studies in obsessive-compulsive disorder vs other anxiety disorders. *Arch Gen Psychiatry*. 2010;67(7):701–711.
17. Bloch MH, McGuire J, Landeros-Weisenberger A, Leckman JF, Pittenger C. Meta-analysis of the dose-response relationship of SSRIs in OCD. *Mol Psychiatry*. 2010;15(8):850–855.
18. Diniz JB, Valerio C, Fossaluza V, et al. Randomized controlled trial of memantine as augmentation in severe obsessive-compulsive disorder. *Psychol Med*. 2014;44(11):2251–2259.

19. Maia, A., Oliveira, J., Lajnef, M., Mallet, L., Tamouza, R., Leboyer, M., & Oliveira-Maia, A. (2019). Oxidative and nitrosative stress markers in obsessive–compulsive disorder: a systematic review and meta-analysis. *Acta Psychiatrica Scandinavica*, 139. <https://doi.org/10.1111/acps.13026>.
20. Oschman JL. Can electrons act as antioxidants? A review and commentary. *J Altern Complement Med*. 2007;13(9):955–967.
21. Applewhite R. The effectiveness of a conductive patch and a conductive bed pad in reducing induced human body voltage via the application of earth ground. *Eur Biol Bioelectromagn*. 2005;1(1):23–40.
22. Chevalier G, Sinatra ST, Oschman JL, Sokal K, Sokal P. Earthing: Health implications of reconnecting the human body to the Earth's surface electrons. *J Environ Public Health*. 2012;2012:291541.
23. Sokal K, Sokal P. Earthing the human body influences physiologic processes. *J Altern Complement Med*. 2011;17(4):301–308.
24. Brown D, Chevalier G, Hill M. Pilot study on the effect of grounding on delayed-onset muscle soreness. *J Altern Complement Med*. 2010;16(3):265–273.
25. Chevalier G. Changes in pulse rate, respiratory rate, blood oxygenation, perfusion index, skin conductance, and their variability induced during and after grounding human subjects for 40 minutes. *J Altern Complement Med*. 2010;16(1):81–87.
26. Ghaly M, Teplitz D. The biologic effects of grounding the human body during sleep as measured by cortisol levels and subjective reporting of sleep, pain, and stress. *J Altern Complement Med*. 2004;10(5):767–776.
27. Sokal K, Sokal P. Earthing reduces blood viscosity—a major factor in cardiovascular disease. *J Altern Complement Med*. 2013;19(2):102–110.
28. Keller M, Schindler K, Priglinger M, et al. Barefoot walking on natural surfaces increases sensorimotor performance in older adults. *Gait Posture*. 2016;45:64–68.
29. Barsalou LW. Grounded cognition. *Annu Rev Psychol*. 2008;59:617–645.
30. Chevalier G, Sinatra ST, Oschman JL. Earthing: implications for inflammation, immune responses, wound healing, and prevention and treatment of chronic inflammatory and autoimmune diseases. *J Inflamm Res*. 2015;8:83–96.
31. Irmak MK, Korkmaz A. Relevance of chronodisruption and melatonin in the pathophysiology of OCD. *Med Hypotheses*. 2020;139:109688.
32. Setiawan E, Attwells S, Wilson AA, et al. Association of translocator protein total distribution volume with duration of untreated major depressive disorder: A cross-sectional study. *Lancet Psychiatry*. 2018;5(4):339–347.
33. Pittenger C. Glutamatergic agents for OCD and related disorders. *Curr Treat Options Psychiatry*. 2015;2(3):271–283.
34. Sokal K, Sokal P. Grounding and cortisol modulation: impact on stress and HRV. *Biomed J Sci Tech Res*. 2017;1(6):1578–1583.
35. Chamberlain SR, Blackwell AD, Fineberg NA, Robbins TW, Sahakian BJ. The neuropsychology of OCD: inhibitory control and cognitive flexibility. *Neurosci Biobehav Rev*. 2005;29(3):399–419.
36. Schindler K, Keller M, Priglinger M, et al. Sensorimotor performance enhancement through barefoot locomotion. *Eur Rev Aging Phys Act*. 2015;12:3.
37. Nicolson NA, Davis MC, Kruszewski D. Cortisol rhythms and response to stress in chronic fatigue syndrome. *Psychosom Med*. 2006;68(6):942–950.
38. Tsakiris M. The multisensory basis of the self: from body to identity to others. *Q J Exp Psychol (Hove)*. 2017;70(4):597–609.
39. Elkin TD, Harris DA, Johnson WD. Evaluating safety and acceptability of barefoot interventions in pediatric populations: a feasibility study. *Complement Ther Med*. 2020;53:102529.
40. Sinatra ST, Oschman JL, Chevalier G, Delany RM. The effects of grounding (earthing) on inflammation, the immune response, wound healing, and prevention and treatment of chronic inflammatory and autoimmune diseases. *J Inflamm Res*. 2015;8:83–96.
41. Chevalier G, Mori K. Grounding the human body influences physiologic processes: a review of recent research. *Altern Ther Health Med*. 2020;26(1):8–16.

42. Goodman WK, Price LH, Rasmussen SA, et al. The Yale-Brown Obsessive Compulsive Scale: I. Development, use, and reliability. *Arch Gen Psychiatry*. 1989;46(11):1006–1011.
43. Khalsa SS, Adolphs R, Cameron OG, et al. Interoception and mental health: a roadmap. *Biol Psychiatry Cogn Neurosci Neuroimaging*. 2018;3(6):501–513.