

Dr. Luis M. Valdés
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U.S. Department of Health and Human Services

Clinical observations on electromagnetic hypersensitivity and the amplification of tinnitus

Dear Sir or Madam,

I am writing to request that research and clinical evaluation of electromagnetic hypersensitivity (EHS) give specific attention to tinnitus, particularly sudden onset or otherwise unexplained worsening in people with sustained exposure close to electrical and wireless devices. My concern is a potentially modifiable amplifier of symptoms in a susceptible patient, even when electromagnetic fields did not cause the original auditory disorder.

Through my clinical work and the five tinnitus manuscripts I have coauthored with electromagnetic radiation researcher Joaquín Machado through EFEIA, I have developed a consistent position: we should distinguish the factors that initiate tinnitus from those that intensify or sustain it. An environmental contribution to amplification deserves investigation on its own terms. It need not explain every case or every aspect of the underlying hearing condition to be clinically relevant.

1 What I observe in clinical practice

I am seeing more patients who describe abrupt tinnitus onset or a marked escalation that is not adequately explained by the history available at presentation. This is a clinical impression within my practice, not a population incidence estimate. Some patients are younger than the conventional picture of age related hearing loss would suggest; others have established hearing impairment but develop a disproportionate increase in tinnitus distress, sleep disruption, or sound intolerance.

When I examine their technological habits and environments, I repeatedly encounter prolonged proximity to powered or transmitting devices: phones and chargers near the head during sleep, devices used close to the ears, wireless equipment beside the bed, electrically powered beds, or nearby electrical machinery. These histories raise a question that is often absent from the usual assessment. The relevant exposure must then be measured; the presence of a device alone does not establish an elevated dose or a harmful exposure.

Our clinical manuscripts describe approximately five years of observational work with dozens of tinnitus patients, using the Tinnitus Handicap Inventory (THI) and audiological assessment, including extended high frequency testing where appropriate. In selected cases, managing the exposure environment alongside usual care was followed by a progressive reduction in tinnitus burden and associated symptoms. This recurring sequence is the reason I consider the environmental question worth investigating. [1]

One illustrative case involved a 70 year old woman with severe hearing impairment and tinnitus in the catastrophic THI category. An environmental assessment identified nearby air conditioning equipment and electrical transients in her residential setting. Measures addressing proximity and the electrical environment, integrated with audiological management, were followed by gradual improvement. This is an uncontrolled observation; it supports a testable amplification hypothesis rather than establishing which intervention caused the improvement. [1]

2 My interpretation of EHS and tinnitus

I regard the overlap with EHS as a possible susceptibility and amplification pattern. Patients reporting environmental sensitivity may also describe tinnitus, poor sleep, heightened arousal, fatigue, and difficulty tolerating sensory input. Tinnitus in such a patient should not automatically be attributed to EMF, and the EHS label does not establish the mechanism. Equally, an initial auditory diagnosis should not close inquiry into potentially modifiable factors sustaining the patient's distress.

My working interpretation is that some exposures may add to the burden of an already sensitized auditory and nervous system. Central auditory gain provides one useful framework: altered processing can increase the prominence of internally generated auditory activity. What our manuscripts propose, and what remains to be tested, is whether particular near source electromagnetic exposures can further modulate that system under real living conditions. [1]

The distinction matters clinically. A patient may retain the hearing loss or other condition associated with tinnitus while experiencing less intrusive tinnitus after an aggravating factor is addressed. That would be meaningful even if the environmental factor never caused the original damage. My observations are consistent with this possibility, but spontaneous fluctuation, habituation, expectation, improved sleep, and concurrent treatments must be considered before a causal interpretation is made.

Published case control research has reported an association between self reported EHS and tinnitus. Importantly, that study did not demonstrate an association between its individual EMF exposure measures and tinnitus. I interpret it as evidence that the clinical overlap merits study, not as proof that exposure causes or amplifies tinnitus. [2]

3 The environment and the patient must be assessed together

The bedroom deserves particular attention because it combines proximity, long periods of occupancy, and the need for restorative sleep. A useful assessment records device location, actual operating states, charging, electrical equipment, and the timing of symptoms. It also evaluates light, sound, screen use, insomnia, stress, medication, and acoustic exposure, so that a change in tinnitus is not attributed to EMF when another factor better explains it.

Our redox manuscripts also examine oxidative and nitrosative stress, glutathione related defenses, and the biological condition of the patient. In my clinical approach, these considerations accompany environmental assessment. Observed improvement with combined interventions cannot isolate their separate effects, and evidence concerning antioxidant protection in other hearing disorders cannot be assumed to establish treatment efficacy for chronic tinnitus. [1]

Our fifth manuscript explores abrupt onset through a model of preexisting vulnerability and a possible triggering event. For this letter, the relevant point is that sudden onset should prompt a careful history of preceding conditions and exposures. It does not establish an electromagnetic cause or prove that different sudden onset syndromes share one mechanism. [1]

Environmental investigation must accompany appropriate medical and audiological evaluation. Sudden hearing loss requires urgent medical attention, and environmental measures must not delay assessment or treatment. Established tinnitus care, including hearing rehabilitation and behavioral support where indicated, remains part of patient management. [3,4]

4 What I ask HHS to support

First, include tinnitus and sound intolerance as explicit outcomes in research on EHS and environmental exposure. Distinguish new onset, exacerbation of existing tinnitus, perceived loudness, distress, and functional impairment, rather than combining them into a single symptom category.

Second, support prospective clinical registries with standardized audiology, repeated THI or comparable validated patient reported measures, symptom diaries, sleep assessment, medication and acoustic histories, and measured exposure. Analyze clinically defined subgroups, including patients reporting close device proximity and nocturnal worsening, without assuming that these features establish EHS.

Third, fund independent studies of environmental management using real and sham interventions where feasible, randomized or counterbalanced order, blinded assessment, verified exposure changes, and adequate follow up. Record concurrent care and assess expectation. Improvement should be evaluated against a suitable comparison, rather than inferred from an uncontrolled before and after sequence.

Fourth, ensure that exposure characterization addresses low frequency electric and magnetic fields as well as relevant RF sources, with the spatial and temporal resolution needed to describe actual use. A questionnaire about call duration or device ownership should not substitute for measuring the specific environmental hypothesis under investigation.

Fifth, develop professional education and a differential assessment pathway connecting tinnitus care, environmental measurement, sleep, and psychological support. Nocebo and conditioning should be assessed respectfully as possible contributors; they should neither be dismissed nor used to avoid investigating other explanations. Patient reports should guide inquiry without being treated as proof of exposure causation.

My request is for a rigorous way to test a recurring clinical observation: in some susceptible patients, the electromagnetic environment may be an amplifier of tinnitus even when it is not the primary cause. Recognizing that distinction could improve study design and identify a modifiable contributor for patients whose symptoms remain difficult to manage. I would welcome the opportunity to discuss our clinical reasoning and explore appropriately consented, independently reviewed research collaboration.

Sincerely,

Dr. Luis M. Valdés

Selected references and supporting manuscripts

1. Valdés LM, Machado JJ. Five EFEIA tinnitus working manuscripts supplied for this letter: redox mechanisms; central gain and an observational case; glutathione and LEDNA; the bedroom environment; and abrupt onset and susceptible phenotypes. Parts 1 to 5. Working discussion manuscripts; clinical observations and proposed mechanisms, not controlled evidence of treatment efficacy.
2. Landgrebe M, Frick U, Hauser S, Hajak G, Langguth B. Association of tinnitus and electromagnetic hypersensitivity: hints for a shared pathophysiology? PLoS One. 2009;4(3):e5026. <https://doi.org/10.1371/journal.pone.0005026>
3. National Institute on Deafness and Other Communication Disorders. Tinnitus. <https://www.nidcd.nih.gov/health/tinnitus>
4. National Institute on Deafness and Other Communication Disorders. Sudden Deafness. <https://www.nidcd.nih.gov/health/sudden-deafness>