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U.S. Department of Health and Human Services (HHS)

**Re: Request to review the criteria used to evaluate and interpret electromagnetic hypersensitivity (EHS) — clinical observations and proposal from the EFEIA Foundation**

Dear Sir or Madam,

I am writing as Director and co-founder of the EFEIA Institute & Foundation, together with Dr. Robert Marshall Leal Lue, PhD in clinical psychology, Dr. Pilar Muñoz-Calero (Fundación Alborada) and other specialists, to request a comprehensive review of the criteria used to date to design, evaluate and interpret studies on electromagnetic hypersensitivity.

Dr. Muñoz-Calero has set out the methodological limitations of provocation studies and the emerging biological evidence. I fully endorse her letter. My purpose here is complementary: to describe, from direct clinical observation of a large number of cases, a mechanism that current study designs do not account for, and that systematically leads EHS patients to be classified as having a psychological condition.

## 1. My professional background

For 15 years I have worked as a researcher of electromagnetic radiation in human environments, focused on exposure assessment, electromagnetic hygiene and the clinical presentation of people who report sensitivity to these exposures. I am the author of *Electromagnetic Pollution* and *Electromagnetic Hygiene*, and I have published peer-reviewed work in the Journal of Applied Biotechnology & Bioengineering.

My clinical observations rest on two bodies of experience:

- **Longitudinal cohort, 2014–2018.** Together with Dr. Robert Marshall Leal Lue, PhD in clinical psychology and co-author of our article on this longitudinal study, I followed 357 patients with EHS over four years in Aruba. This follow-up allowed us to observe onset, course, relapses and response to environmental and clinical interventions over time, not in isolated sessions.
- **International consultant network.** Through EFEIA's network of international consultants — functional medicine physicians, psychiatrists, psychologists, osteopaths, audiologists, clinical nutritionists, health coaches and other health professionals — I have reviewed or directly observed more than 1,500 cases of EHS over the years.

These are clinical and observational data, not controlled trials. I offer them for what they are: a large, consistent body of first-hand observation that should inform how controlled studies are designed and interpreted.

## 2. Our position and clinical observations

In our experience, EHS behaves neither as a purely psychological phenomenon nor as a simple dose–response toxicity. It presents as a sensitized, partly conditioned stress response of a susceptible organism, with a recognizable phenotype. My reasons are as follows.

**a) The nocebo effect is real, and it is part of the picture — not the explanation of it.** We fully acknowledge that negative expectations can produce symptoms attributed to electromagnetic fields (Bräscher et al., 2017). However, qualitative work by Dieudonné (2016) found that in many patients symptoms preceded the attribution to EMF, which is inconsistent with a purely expectation-driven onset. Our clinical observation is consistent with this sequence: physical distress first, attribution afterwards.

**b) Clinically, we observe a conditioned response with features similar to post-traumatic stress.** After one or more episodes in which a real exposure coincided with genuine physiological distress, many patients develop an association between the source and the malaise. From then on we observe hypervigilance, anticipatory autonomic activation, avoidance and symptom amplification in response to cues — the sight of an antenna, a router light, the knowledge that a device is present — even when the emitter is switched off. This is consistent with established models of interoceptive learning and symptom perception (Van den Bergh et al., 2017).

The consequence for interpretation is decisive. In post-traumatic stress, no clinician concludes that the original trauma never happened because the patient reacts to a harmless reminder. Yet in EHS research, a reaction to a sham exposure is routinely read as proof that the condition has a psychological origin. A conditioned response to an inactive cue does not demonstrate that the original trigger was inert.

**c) Symptoms are not always caused by a source; often they are sustained by a sensitized body.** We have repeatedly observed patients whose symptoms persist or recur without a relevant active exposure, because the organism remains in a self-sustaining state of sensitization — what we describe clinically as a chemical loop of inflammatory, oxidative and autonomic dysregulation. This fits the limbic kindling model proposed for multiple chemical sensitivity (Bell et al., 1992), the biomarker findings of Belpomme et al. (2015) and De Luca et al. (2014), and the framework of central sensitivity syndromes (Yunus, 2008). In this state the trigger becomes progressively less specific.

**d) Current provocation designs therefore produce a systematic classification bias.** When already sensitized and conditioned patients are tested with short blinded exposures and conscious perception as the endpoint, the design cannot separate three components: the environmental trigger, the physiological sensitization and the learned response. Almost every case is then sorted into the psychological category. This is not a finding about the nature of EHS; it is a property of the method.

**e) EHS has a specific phenotype, but it should not be treated as a disease in itself.** We understand EHS as a biological response of a susceptible organism to an environment whose characteristics predispose that response. The appropriate clinical target is the interaction of three factors — the exposure environment, the biological terrain of the patient and the conditioned response — rather than any one of them alone.

## 3. The EFEIA approach: differential assessment before attribution

To address this, we developed a preliminary clinical protocol from the 2014–2018 cohort. Dr. Leal Lue led the research within the NOXTAK project that established a clinical psychological protocol for determining the degree of nocebo involvement; that work gave rise to the EFEIA Differential Diagnosis Protocol. It is now a mandatory part of EFEIA's intake questionnaire across our consultant network. Its purpose is to determine what is actually happening in each patient before any attribution is made — environmental, physiological or psychological.

The protocol assesses:

1. **Exposure history and onset** — the temporal relationship between real exposures and the first episodes of symptoms.
2. **Degree of psychological and conditioned involvement** — a clinical estimate of anticipatory anxiety, hypervigilance, avoidance and trauma-like features, which allows us to identify nocebo-driven presentations and to measure how much of the current picture is learned response.
3. **Physiological sensitization and comorbidity** — overlap with multiple chemical sensitivity, fibromyalgia and chronic fatigue syndrome.
4. **Objective markers where available** — for example, heart rate variability as an index of autonomic regulation.
5. **The actual exposure environment** — measured, not assumed, by trained consultants.

The result is a profile, not a label: it indicates in what proportion a given case is driven by the environment, by the patient's biological terrain and by conditioning. This is the broader view we propose. It accepts the nocebo literature, accepts the biological literature, and holds that EHS can only be understood when both are assessed in the same patient.

EFEIA is prepared to share the protocol and de-identified case data with independent investigators.

## 4. Our requests

1. That the criteria used to design and interpret EHS studies be reviewed, as Dr. Muñoz-Calero proposes, to include prolonged exposures, delayed responses, adequate washout periods and objective biological markers.

2. That participants be stratified before provocation testing by their degree of conditioned response and physiological sensitization, so that a learned reaction to a cue is not read as evidence about the original trigger.

3. That nocebo and conditioned responses be treated as components to be measured in each patient, not as a diagnosis of exclusion that closes the inquiry.
4. That independent research, free from conflicts of interest, be promoted on the interaction between conditioned stress responses and physiological sensitization, including its relationship with multiple chemical sensitivity and central sensitivity syndromes.
5. That clinical protocols and medical training be developed for differential assessment and integrated care — environmental remediation, physiological support and trauma-informed psychological care — without assigning a psychological origin by default.
6. That, while knowledge advances, the precautionary principle be applied, especially in hospitals, schools and settings where the most vulnerable people are present.

I remain at your disposal to expand on any aspect of this letter, to present our protocol and clinical data, and to collaborate in any way you consider appropriate.

Sincerely,

**Joaquín Machado**
Director and Co-founder, EFEIA Institute & Foundation
Researcher in electromagnetic radiation and environmental exposure

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