Where Can They Hide?

A response to the U.S. Department of Health and Human Services Request for Information on RF/EMF exposure and human health

Docket: HHS-OASH-2026-0397

Respondent: Andrew Mills, individual respondent

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Perspective: Individual consumer; person reporting symptoms they associate with RF/EMF exposure; family member of another person reporting such symptoms.

Summary: listen, measure, test

This submission asks HHS to take three linked steps: listen carefully to people who report symptoms they associate with radiofrequency and other electromagnetic-field (RF/EMF) exposure; measure real-world exposure more accurately; and commission independent research capable of testing proposed explanations fairly.

I do not claim that RF/EMF exposure has been established as the cause of the symptoms described here, or that any single biological mechanism explains them. I do believe that people reporting substantial symptoms should be heard and their experiences recorded, while scientific investigation distinguishes association from causation.

I report neurological and mood-related effects that I associate with exposure, including irritability and anger that can arise in situations where I would not ordinarily expect such a response. Family members and friends have described different experiences that they associate with wireless exposure, including neurological symptoms, severe skin problems, eye pain and cardiac symptoms. These are personal reports, not clinical diagnoses or controlled experiments. Some people arrived at their suspicions through a painful process of elimination, observing symptoms in particular places and changes when suspected sources were reduced or switched off.

One research question I believe deserves targeted investigation concerns haemoglobin, oxygen handling and tissue oxygen delivery. Laboratory papers have reported changes in haemoglobin's oxygen affinity or the rate of its deoxygenation under particular experimental conditions. A 2025 sleep study also reported differences in consumer-wearable estimates of overnight oxygen saturation between periods with and without a mobile phone present. None of these findings, individually or together, establishes that ordinary RF exposure reduces arterial oxygen saturation or causes tissue hypoxia in people. They do, however, identify testable questions that should be independently replicated using verified exposure and clinically validated measurements.

I ask HHS to prioritise: (1) co-designed, individual-level, blinded studies of people reporting sensitivity; (2) independent research into haemoglobin function, oxygenation and other plausible mechanisms; (3) personal and environmental exposure measurement over realistic periods, including homes, schools and vehicles; (4) a standard reporting route for suspected adverse events; and (5) clear public disclosure of wireless transmitters and exposure information, alongside practical options to reduce non-essential transmissions where feasible.

1. Perspective and how to read this submission (RFI Q2)

I respond as an individual consumer and family member. I am a behavioural coach and former Chartered Valuation Surveyor based in the United Kingdom. I am not a clinician or laboratory scientist and do not present myself as one.

I distinguish between:

- **[P] Peer-reviewed research** - published research, with the limitations of each study stated.
- **[G] Government or regulatory material** - official guidance, reports, or legal decisions.
- **[L] Lived experience** - personal or anonymised accounts reported to me.
- **[O] Other material** - including a case account published in a specialist newsletter, which is not equivalent to a peer-reviewed controlled study.

The RFI invites individuals to share experiences and asks respondents to distinguish established evidence from emerging findings and uncertainty. This submission does so. No names or identifying health details of family members or friends are included.

Questions addressed: 2, 3, 5, 5(n)-(p), 6, 7, 8, 9, 10(b)-(h), 11(a), (b), (e) and (g), 12, 13(c)-(k), 14, 15(a)-(e), 16, 17 and 18.

2. A question before the evidence

Imagine a grandchild reading this record thirty years from now. She asks: what did we know in 2026, when people said they were becoming ill in environments filled with wireless technologies, and what did we do to find out?

I cannot honestly tell her that the cause has been established. The evidence does not justify that conclusion. But I would also not want to tell her that we treated uncertainty as a reason to stop asking questions, or left people who reported disabling symptoms to investigate their environments alone.

The request here is not for HHS to accept a predetermined explanation. It is for HHS to listen without prejudging, distinguish what is established from what remains uncertain, collect better exposure data and fund studies that can test specific hypotheses.

3. Reported experiences: symptoms, exposure changes and sleep (RFI Q3)

My account and accounts shared with me [L]

Over approximately eight years, I have associated some changes in my mood and neurological state with RF/EMF exposure. The change I notice most clearly is irritability or anger, including while driving, that can feel disproportionate to the circumstances. I have also noticed changes when reducing exposure, including through protective headwear and eyewear. These observations are unblinded and cannot establish causation.

A family member has reported more severe difficulties, with symptoms associated with particular places and improvement when a nearby Wi-Fi source was switched off. Other family members and friends have described different symptoms that they associate with exposure: neurological effects; severe skin irritation, dryness and skin that reportedly cracked or bled; pain involving the eyes; and cardiac symptoms. I cannot independently establish the cause of these symptoms, and they should not be assumed to share one mechanism. Their severity nevertheless makes accurate clinical assessment and fair investigation important.

Two reports involving sleep also prompted me to ask whether this subject deserves better study. In one, friends told me that their six-week-old baby had not slept through the night during the first six weeks. After both parents switched off their mobile phones and household Wi-Fi overnight, they reported that the baby slept through for the first time. In a separate account, a family member who had experienced significant sleep difficulties reported sleeping through the night for the first time when household Wi-Fi and mobile phones were switched off.

These are uncontrolled observations, not evidence that RF exposure caused the earlier sleep difficulties. Several sources were changed at once; exposures were not measured; and sleep naturally varies, particularly in very young infants. They should not be treated as proof. They do, however, illustrate the kind of repeated, well-documented question that objective sleep measures and carefully controlled exposure studies could examine.

A reported skin case and the Ginkgo question [O]

A case account by Mary Redmayne, published in *ES-UK Newsletter*, Spring 2022, Vol. 19, No. 3, pp. 8-9, described a teenage boy with severe recurring skin inflammation, itching, scabbing and bleeding. The account reported that symptoms appeared after visits to town and worsened after returning to school, where wireless sources were present. The family reportedly had no Wi-Fi, smart devices or cordless phones at home and kept mobile-phone use to a minimum. After consultation with health professionals, the family tried Ginkgo biloba; the newsletter reports substantial improvement in the skin over the following months, although pain associated with being in or having been in town or school reportedly continued.

This is a single, uncontrolled case account, not a peer-reviewed clinical trial. The reported exposure was not quantified, the skin condition's cause was not established, and the account cannot show that RF/EMF caused the condition or that Ginkgo caused the improvement. Most importantly for the hypothesis discussed below, no blood oxygen, haemoglobin function or tissue oxygenation was measured.

The case nevertheless suggests a question, not a conclusion: could research into symptoms attributed to RF/EMF usefully measure more than exposure and self-reported symptoms, including relevant physiological variables? A small crossover study in ten healthy volunteers reported changes in red-cell aggregation and increased nail-fold capillary blood flow after Ginkgo biloba, but it did not show that oxygenation had been abnormally low before treatment [18]. Even if a substance alters circulation or microcirculation, a response to it does not logically prove that a person previously had an oxygen deficit. Ginkgo has several proposed biological actions, and improvement in one symptom cannot identify which action, if any, explains the change. I include the case because it led me to consider this research question, not because it proves the mechanism.

The RFI asks for lived experience, and these reports are offered on that basis. I ask HHS to develop a standardised, privacy-protecting method to record such reports, including exposure source, timing, changes made, symptom course, relevant clinical assessments and the limits of what can be inferred.

4. What is established, and what remains open (RFI Q5, Q6, Q12, Q14)

RF energy can heat biological tissue at sufficiently high exposure. The ICNIRP 2020 guidelines cover 100 kHz to 300 GHz and include exposure restrictions and methods of assessment for this frequency range [1]. The existence of established thermal effects is not in dispute. The research question is whether the current evidence and assessment frameworks adequately address every relevant outcome and realistic exposure pattern raised by this RFI, including chronic, multi-source exposure, particular modulation characteristics, sensitive populations and potential environmental effects.

The U.S. National Toxicology Program reported clear evidence of carcinogenic activity based on malignant heart schwannomas in male rats exposed to GSM- and CDMA-modulated RF radiation under its experimental conditions. The female-rat findings were equivocal, and the findings did not establish the same pattern across all animal groups. The exposures were prolonged whole-body exposures at levels not directly equivalent to ordinary mobile-phone use; the studies do not provide a human risk estimate [2]. A separate Ramazzini Institute lifetime rat study reported an increase in the same rare tumour type among male rats in its highest-exposure group, but event counts were small and the study had methodological limitations [3]. These animal results merit careful evaluation without being presented as proof of human harm at everyday exposure levels.

The 2021 U.S. Court of Appeals decision in *Environmental Health Trust v. FCC* required the FCC to address specified issues more adequately, including long-term exposure, children, the ubiquity of wireless devices and environmental effects. The court did not itself determine that the FCC limits were unsafe or decide the underlying scientific debate [4]. In September 2026 the FCC opened a further comment process on the remanded issues; that proceeding is separate from this HHS RFI [5].

On reported electromagnetic sensitivity, systematic reviews of blinded or double-blinded provocation studies have generally not found a consistent group-level ability to identify exposure or a reliable causal relationship between exposure and reported symptoms in the experimental conditions studied [6]. This evidence must be taken seriously. It does not establish that symptoms are imaginary, and it does not answer every possible question about individualised responses, long exposure durations, changing real-world environments or mechanisms that have not been adequately tested. It supports carefully designed studies rather than presuming either causation or absence of any possible effect.

I do not claim that RF/EMF causes autism, ADHD, heart disease, skin disorders or any other specific diagnosis. Nor do I infer causation from increases in diagnosis rates. The appropriate question is whether the relevant exposure and health outcomes have been measured well enough to test clearly defined hypotheses.

5. Haemoglobin, oxygen handling and tissue oxygen delivery: a research priority (RFI Q12, Q16, Q18)

I ask HHS to examine this hypothesis specifically, because it is testable and could help distinguish possible biological pathways. Haemoglobin transports oxygen from the lungs to tissues. Its oxygen affinity, the rate at which oxygen is released, arterial oxygen saturation and actual tissue oxygen delivery are related but distinct measurements. They must not be treated as interchangeable.

What the studies report

- **Mousavy et al. (2009) [7] [P].** In a laboratory study of isolated human haemoglobin, exposure to 910 and 940 MHz RF was reported to alter haemoglobin structure and oxygen affinity. This was an in-vitro result: the study did not measure blood oxygen saturation or oxygen delivery in living people.
- **Muehsam et al. (2013) [8] [P].** In a cell-free assay using a chemical reducing agent to initiate haemoglobin deoxygenation, the authors reported a faster rate of deoxygenation following exposure to a particular pulse-modulated RF signal or a static magnetic field. The authors proposed that, if the effect were replicated at cellular and tissue levels, altered oxygen availability might be relevant to some biological effects. This result does **not** show that RF exposure reduces oxygenation in a living person; the authors' discussion includes the possibility of enhanced oxygen availability.
- **Chowdhury et al. (2021) [9] [P].** Raman spectroscopy of human red blood cells exposed to specified GSM signals produced spectral changes interpreted by the authors as consistent with altered haemoglobin-oxygen interaction. Again, these laboratory findings do not establish a clinically significant reduction in oxygenation in people during everyday exposure.
- **Slezáková et al. (2025) [10] [P].** A small study using consumer wearable devices reported differences in estimated overnight oxygen-saturation values between periods with and without a mobile phone present. The device-based outcome is preliminary; the study did not directly quantify RF exposure or establish that the differences were caused by RF, nor did it establish clinical hypoxia.

The Muehsam paper also discloses partial support from a company involved in therapeutic EMF devices and consulting relationships for some authors; these disclosures are another reason to seek independent replication. These findings are not a single, consistent demonstration of an RF-induced oxygen deficit. They involve different signals, exposure conditions, preparations and outcome measures. In particular, faster haemoglobin deoxygenation in a cell-free assay is not equivalent to lower arterial oxygen saturation. Reduced haemoglobin oxygen affinity can change oxygen loading and unloading in ways that depend on physiological context; it does not automatically mean that the body receives less oxygen.

What the Ginkgo account does and does not add

The Redmayne newsletter case is an additional reported observation in which skin improved after Ginkgo biloba was introduced, while exposure-associated pain was still reported. It contains no oxygen measurements and cannot identify the mechanism of improvement. Ginkgo has multiple proposed biological actions; therefore, the improvement cannot be used to infer that RF had reduced oxygenation, or that Ginkgo corrected such a reduction.

Taken together, the laboratory papers and the case account justify asking whether the relationship between RF/EMF exposure and oxygen handling is worth investigating. They do not establish the answer. That distinction should guide the research design.

The research I ask HHS to commission

HHS should fund a preregistered, independent programme that tests specified frequencies and signal characteristics under verified exposure and sham conditions. Initial studies should replicate the laboratory findings before extending them to more complex systems. Where justified, follow-up research should distinguish and measure:

1. haemoglobin oxygen affinity and oxygen-dissociation curves;

2. red-cell morphology and function, haemoglobin concentration and haematocrit;

3. arterial oxygen saturation and, where ethically and practically appropriate, blood-gas measures;

4. relevant measures of tissue perfusion or oxygen delivery, rather than assuming these from SpO2 alone;

5. temperature and other potential confounders; and

6. whether any physiological changes are reproducible, dose-related, dependent on frequency or modulation, persistent after exposure, and associated with symptoms or impaired function.

The studies should use sufficient sample sizes, validated equipment, blinded outcome assessment, sham controls, declared conflicts of interest and independent replication. Any result should be reported whether positive, null or inconsistent. A measurable biological response should not be labelled harmful without evidence of adverse significance; equally, a proposed mechanism should not be excluded without suitable tests.

6. Real-world exposure: homes, schools, transport and vehicles (RFI Q7-Q9, Q13, Q15)

Everyday exposure may come from multiple sources, including phones, Wi-Fi, Bluetooth, wearables, smart meters, connected appliances, mobile infrastructure, satellite-based communications and vehicle telematics. The extent of exposure depends on the source, distance, frequency, power, duty cycle, signal conditions and how devices operate. I do not assume that all sources contribute equally or that more devices necessarily produce a fixed increase in personal exposure. That is why measurement is needed.

The RFI should prompt HHS to support time-resolved personal and environmental measurements across homes, schools, workplaces, healthcare settings and transport. Where feasible, measurements should attribute signals to their sources and record frequency, field strength or power density, duration, modulation, duty cycle and simultaneous exposures. Individual measurements should be paired with clear information about location, activity and device operation, while protecting privacy.

Connected vehicles and telematics

Vehicles deserve particular attention because occupants may be unable to leave an environment that they associate with symptoms. Modern vehicles can contain mobile modems, emergency-call systems, Wi-Fi hotspots, Bluetooth links, wireless charging, keyless entry and other connected systems. The configuration differs by make, model and year. A metal vehicle body can alter signal propagation, and a phone's transmission power can vary with reception conditions; it is not accurate to claim that every vehicle acts in the same way or that all onboard systems produce the same exposure.

Bluetooth hands-free operation also does not universally route a phone's cellular signal through an external vehicle antenna. That depends on the specific vehicle and equipment. Some systems have external cellular antennas or phone-coupling arrangements; others do not. Manufacturers should document what each system transmits, where its antennas are located, and which functions can be disabled.

I ask HHS to encourage independent measurements in representative vehicles, with phones and embedded systems tested separately and together under realistic reception, passenger and driving conditions. Results should be reported by source and compared with relevant exposure standards, without treating a measurement below a guideline as proof that a symptom mechanism exists or treating it as proof that no further question can be asked. Buyers and occupants should have understandable information and, where feasible, a way to disable non-essential transmitters while retaining safety-critical functions.

A comparable principle applies to homes and public buildings. Consumers, schools, healthcare providers and local authorities should be able to find clear information about relevant transmitters and measurements. Where practicable, wired alternatives and designated lower-wireless-use spaces could provide options for people who report sensitivity while research continues. Such arrangements should be evaluated for feasibility and accessibility, rather than presented as proof that wireless exposure causes symptoms. The practical question is simple: where a person cannot leave a classroom, workplace, train or vehicle, what information and reasonable choices are available to them?

7. Occupational monitoring, accommodation and public-health surveillance (RFI Q4, Q10, Q11, Q18)

Certain occupational tasks near transmitting antennas require advance planning, exposure assessment and controls to reduce exposure where necessary. Occupational practices are not proof that ordinary public exposure below a guideline causes illness; the exposures and tasks may be very different. They do, however, illustrate useful principles: identify the source, measure exposure, keep records, explain controls and have a plan for managing high-exposure conditions.

HHS should examine whether appropriate de-identified occupational monitoring data could contribute to longitudinal exposure research, recognising that workplace readings may not be comparable with personal exposure in homes or vehicles. It should also establish a standard adverse-event reporting route for people who suspect RF/EMF-related symptoms and a transparent procedure for considering reports of illness clusters. Reports should be treated as signals for investigation, not as confirmed causal cases.

A legal example of accommodation [G]

In EAM v East Sussex County Council [2022] UKUT 193 (AAC), the Upper Tribunal required an Education, Health and Care plan for a child whose reported electromagnetic sensitivity had been accepted as disabling on the case-specific evidence and who was hindered from using Wi-Fi-dependent computers for education. A wired internet connection was found to be educational provision. The decision illustrates that functional impairment attributed to electromagnetic sensitivity can be legally relevant to disability and practical accommodation, even while the scientific question of causation remains distinct [19].

The ruling should not be overstated: it did not establish a general medical diagnosis of EHS or determine that RF exposure generally causes symptoms. The judge adopted the earlier tribunal's disability analysis while noting a concern that parts of it rested on the local authority's failure to produce or challenge evidence. The judge also noted that the NHS did not recognise the child's condition as a medical one for the separate statutory question of whether a wired connection counted as healthcare provision. This was a case-specific decision under the law governing education and disability in England, not a scientific finding and not a precedent binding on U.S. agencies. It is relevant to HHS as a comparative example of the practical-access question.

Exposure assessment and risk evaluation should specifically consider children, pregnancy, people with implanted medical devices, workers with elevated occupational exposure and people who report substantial sensitivity. That does not mean presuming that every group is harmed; it means making sure that evidence and exposure methods are relevant to each population, and being explicit about where data are sparse.

8. Recommendations to HHS (RFI Q5, Q9-Q11, Q16-Q18)

I ask HHS to consider the following actions:

1. **Listen and record.** Develop a standard, privacy-protecting reporting pathway for suspected RF/EMF-associated symptoms, distinguishing self-attribution, clinical findings and confirmed diagnoses. Report anonymised patterns and limitations publicly.
2. **Test individual sensitivity fairly.** Commission preregistered, adequately powered, co-designed, double-blind provocation studies, with verified RF and sham exposures, clinically characterised participants, realistic durations, prespecified symptoms and objective measures where suitable. Replicate findings independently.
3. **Investigate oxygen handling.** Fund a staged programme to replicate and extend the laboratory haemoglobin and red-cell findings, distinguishing oxygen affinity and deoxygenation kinetics from arterial oxygen saturation and tissue oxygen delivery. Publish null and contradictory results as well as positive results.
4. **Measure exposure as people experience it.** Improve personal dosimetry and time-resolved measurements across homes, schools, workplaces and transport. Measure multiple sources, frequency and modulation characteristics, duration and proximity rather than relying exclusively on occasional area surveys.

5. **Study vehicles and disclose transmitters.** Conduct independent source-by-source measurements in connected vehicles and public transport. Encourage clear consumer information about onboard radios, antenna placement and the ability to disable non-essential transmissions.
6. **Build longitudinal evidence.** Support well-designed cohorts that connect repeated personal exposure measurements with carefully defined health outcomes, beginning where appropriate with pregnancy and childhood. Do not infer causation from diagnosis trends alone.
7. **Review standards transparently.** Compare the current FCC framework, ICNIRP 2020, IEEE standards and relevant international approaches. Publish the evidence considered, uncertainties, methods, conflicts of interest and criteria that would prompt guideline review. Give appropriate weight to independent research and to strong negative findings as well as positive findings.
8. **Coordinate responsibilities.** NIH should lead or support mechanistic and clinical research; CDC should develop surveillance and cluster-investigation methods; FDA should contribute to device-related testing and consumer information; and HHS should coordinate the overall programme with appropriate expertise and public reporting.
9. **Provide practical options while uncertainty is investigated.** Encourage accessible wired alternatives and lower-wireless-use options where feasible, especially in settings used by children and people who report sensitivity. Evaluate these measures on practicality, accessibility and evidence, without implying that an unproven mechanism has already been established.

Research priorities should be set by independent, multidisciplinary groups that include researchers who report evidence of effects and researchers whose findings are null or non-confirming, alongside clinical, engineering, statistical and public-health expertise. Protocols should be preregistered, funding and conflicts declared, data and methods shared where ethically possible, and independent replication planned from the outset.

What would change my view. If independent, preregistered, adequately powered studies using verified exposure, appropriate sham conditions and validated measurements repeatedly found no reproducible changes in haemoglobin function or oxygenation, and well-designed individual-level provocation studies found no reliable symptom response under relevant conditions, I would give those findings substantial weight. Conversely, isolated positive findings without independent replication would not, on their own, establish a general mechanism or a health risk.

9. Questions I hope HHS will answer

- What measurements would be sensitive enough to detect a reproducible biological effect if one exists, and are we collecting those measurements in real-world settings?
- Can selected RF exposures reproducibly alter haemoglobin function, oxygenation or tissue oxygen delivery in living systems, and are any observed changes adverse?
- Can studies designed with people who report sensitivity identify reproducible individual responses under verified blinded exposure conditions?
- What exposure information should be available to people in homes, schools, workplaces and vehicles, and what practical choices should they have where feasible?
- How will HHS make sure that both positive and null findings are independently tested and publicly reported?

Closing: the answer we hope to give

When that grandchild asks what we did, I hope we can say that we neither presumed danger nor assumed every relevant question had been answered. We listened to people's accounts without treating them as proof; we respected the evidence that did not confirm a causal relationship; we measured exposure more realistically; and we funded the studies needed to reduce uncertainty.

That is the responsibility I am asking HHS to take seriously. People should not have to become scientists to have their experiences recorded and fairly investigated. Nor should any of us confuse a compelling hypothesis with a proven explanation. The task is to find out, openly and rigorously, what the evidence can support—and to make sure that uncertainty does not become an excuse for inaction.

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