Understanding human physiological responses to RF electromagnetic fields: research priorities

Response to the Department of Health and Human Services (HHS) Request for Information on Electromagnetic Fields (EMFs), Radiofrequency (RF) Radiation, and Wireless Radiation Exposure (FR Doc. 2026-19252; Docket HHS-OASH-2026-0397)

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Summary statement: We recommend that HHS prioritize research on the temporal structure of radiofrequency electromagnetic field (RF-EMF) exposure and interindividual variability in physiological responses.

Perspective (Question 2): academic researchers responding as individuals (closest listed category: research entity). Affiliations are given for identification only. The views expressed are our own and not those of our institutions or of any scientific society in which we hold a role. Questions addressed: 2, 5(o), 5(p), 8, 11(g), 16 and 17. Basis: peer-reviewed research, guidelines and standards and a trial registration.

Within our area of expertise, we recommend that HHS prioritize research on the temporal structure of RF-EMF exposure and interindividual variability in physiological responses (Question 16). Several laboratories have reported small changes in the human electroencephalogram (EEG) during or after exposure to pulse-modulated RF-EMF, mainly in alpha power during wakefulness and spindle-frequency power during sleep. No adverse health effect has been linked to them. It is not known whether these changes are reproducible, which exposure characteristics produce them, in whom they occur or whether they matter for health. We address only questions within our research expertise, not other endpoints such as cancer or fertility. We make no recommendation on exposure limits or precautionary measures.

Our first priority (Questions 5(o), 5(p), 8 and 16) is to determine whether temporal exposure characteristics influence physiological responses. RF exposures are commonly described by carrier frequency, field strength, specific absorption rate (SAR) and duration. Communication signals also have temporal characteristics, including amplitude modulation, pulsing, frame structure and changing transmission patterns. These parameters warrant systematic testing. Previous studies have examined their relevance, but have not established whether any temporal characteristic consistently influences physiological responses. The frequencies of these temporal patterns overlap with those of physiological signals in the brain.

Hinrikus and colleagues varied modulation while maintaining a 450-MHz carrier. In 13 volunteers, they reported increased alpha and beta EEG power with 14- and 21-Hz modulation, but not with 7-Hz modulation [1]. A later report from the same laboratory, spanning 7–1000 Hz, classified 0 to 31% of participants as affected, depending on modulation frequency [2]. Schmid and colleagues investigated sleep EEG after different pulse-modulated exposures [3,4]. These and related findings support further controlled comparisons between modulated and non-modulated signals,

but do not establish a general modulation-specific mechanism. Event-related designs may detect transient responses that resting-state measures miss. Carrubba and colleagues developed a nonlinear recurrence analysis for this purpose and applied it to low-frequency magnetic and electric fields [5,6]. Independent replication using an RF-EMF stimulus and direct comparisons with resting-state EEG methods are needed.

A systematic review of 51 studies of waking EEG and event-related potentials, by one of us and colleagues, rated none as having a low overall risk of bias, mainly because only one reported a prespecified analysis plan [7]. Its only nominally significant result among 13 pooled comparisons was a small increase in eyes-open alpha power with a GSM-like (2G) signal (standardized mean difference (SMD) 0.16, 95% confidence interval 0.01 to 0.32, $p = 0.04$). Four of the five pooled studies came from one research group and the fifth from the group of one of us. One of the four has since been corrected and no longer shows a statistically significant effect on alpha power [8]. In the review's sensitivity analysis excluding that study, the estimated SMD was 0.15 (95% confidence interval −0.02 to 0.31; $p = 0.08$) [7]. This excludes the study rather than incorporating its corrected data. Of the 26 resting-EEG studies in the review that analyzed alpha power, 17 reported a change, 14 of them an increase, but methodological heterogeneity prevented pooling most of them [7]. The pooled finding concerns a signal with a defined pulse structure but does not establish modulation as the cause. Moreover, newer protocols were studied less frequently, precluding a reliable comparison of responsiveness across generations.

In 5G, the temporal pattern of exposure depends on the band and the device: time- or frequency-division duplex, periodic synchronization bursts, beam steering and the phone’s scheduled, power-controlled uplink, with the resulting exposure pattern depending on network configuration and traffic [9,10]. Claims about the presence or absence of a “5G effect” therefore require precise specification of the tested exposure and cannot automatically be generalized to other configurations.

A 2011 review found that most comparisons of modulated and unmodulated fields showed no modulation-specific effects, with a few exceptions in the human central nervous system [11]. Huber and colleagues found EEG changes with pulse-modulated but not continuous-wave exposure [12], whereas Perentos and colleagues found no significant difference between the two [13]. No established mechanism explains a modulation-specific effect. Living cells and tissues showed no consistent second harmonic of an 883-MHz signal [14], but this test does not address heating. Rectification of an 800-MHz signal, applied from a probe 10 to 15 micrometers away, was reported in artificial membranes with channel-forming peptides or pores but has not been independently replicated [15]. A thermal mechanism is plausible, because exposure at 1 to 2 W/kg engaged thermoregulation [16]. Recording artifacts must also be excluded.

Interestingly, the temporal pattern is also studied as an experimental variable for other stimuli, such as 40-Hz flickering light or combined light and sound [17,18]. The same applies to other components of the electromagnetic field: oscillating magnetic fields and pulsed electric fields. Reviews of rhythmic transcranial magnetic stimulation and transcranial pulsed current stimulation describe physiological responses that depend on temporal stimulation parameters [19,20]. This analogy does not imply a shared mechanism with RF-EMF, but it places research on RF-EMF effects in a context broader than safety alone. For one established RF effect, the microwave auditory effect, short pulses act through their energy per pulse rather than time-averaged power. ICNIRP found no evidence that it affects health under realistic exposure conditions [21,22].

Current limits (FCC, 47 CFR 1.1310; ICNIRP 2020 [22]; IEEE C95.1-2019 [23]) protect against the effects that the standard-setting bodies consider established, mainly tissue heating and, at lower frequencies, nerve stimulation. Heating limits are mainly time-averaged and ICNIRP and IEEE also limit the energy of single pulses [22,23]. ICNIRP regards the EEG changes described above as reliably shown at SAR values below 2 W/kg, with no evidence of adverse health effects [22]. The meta-analysis and the subsequent correction to one of its included studies provide weaker support for effects in waking EEG [7,8]. The research we propose would test whether specific temporal characteristics produce such changes, not their health relevance. On Question 5(p), current evidence is insufficient to decide whether modulation or pulsing needs a separate classification. The distinction between thermal and non-thermal effects also requires care. Detectable heating does not establish that an EEG response is entirely thermally mediated and the absence of measurable heating does not exclude a thermal contribution.

Our second priority (Questions 11(g) and 16) is variability between and within individuals. Group averages may obscure individual responses, while responsiveness might also vary between sessions in the same person. Apparent responders can arise through chance, measurement error or multiple testing. Individual differences in exposure effects must be distinguished from within-person variability and measurement error using appropriate control measurements [24]. Repeated active and sham sessions can help distinguish stable differences between people from variation in responsiveness within the same person. A retest of 20 participants found the increase in sleep EEG power mainly in those who had responded previously [25], whereas another study of 20 men exposed twice reported exposure-related delta-theta power increases but no reproducible individual responses [26]. Two studies found no greater EEG response in adolescents than in adults [27,28]. We therefore recommend repeated, randomized, double-blind active and sham sessions, with prespecified EEG outcomes and response criteria. Repeated sham measurements should quantify within-person variability; candidate responses should be confirmed in independent sessions and laboratories. Experiments should vary temporal characteristics while keeping the carrier frequency, duration, exposure geometry and time-averaged SAR distribution constant. They should report peak and time-averaged exposure metrics, duty cycle and temperatures [29] and include a non-RF heating control. EEG leads can pick up and demodulate RF, yet only 17 of 51 studies tested for this [7]. Artifacts, blinding and carryover therefore need explicit checks, including phantom tests. Positive and null results should be reported with effect sizes and uncertainty.

A longer-term aim is to establish whether a reproducible individual EEG response to RF-EMF exists. If such a response exists, it could be compared between controls and people reporting electromagnetic hypersensitivity (EHS). In provocation studies reviewed for WHO, neither the general population nor people reporting EHS detected exposure better than chance and symptoms followed perceived rather than actual exposure. These were short exposures, mostly in young, healthy volunteers [30]. On Question 11(g), current evidence therefore does not show that people reporting EHS respond differently. An EEG measure would complement provocation studies because it does not depend on perception. It would allow a possible physiological correlate of EHS to be investigated without presupposing that such a correlate exists and without attributing symptoms to RF-EMF on the basis of EEG changes alone.

NIH could fund preregistered multi-laboratory studies coordinated with European programs (Question 17). These studies could use a common protocol, independent exposure verification by FDA and blinded analysis and involve investigators who interpret the evidence differently.

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