

Response to Request for Information on Electromagnetic Fields (EMFs), Radiofrequency (RF) Radiation, and Wireless Radiation Exposure

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Summary of this response

The gap. FCC’s head-exposure limit is 1.6 W/kg averaged over 1 g of tissue [37]. Standard compliance phantoms contain no dental metal, although many people have permanent conductive restorations or implants regulated by FDA [36]. **This comment asks HHS to support standardized testing of metal-bearing anatomical models, not to assume an FCC limit violation or a health effect.**

What is established. (1) Dental amalgam is a measurable, dose-related source of internal mercury exposure at population scale, demonstrated in NHANES-based analyses [5–9]. (2) In peer-reviewed finite-difference time-domain computational dosimetry, changing nothing but the presence of a metallic restoration or implant changes the local distribution of absorbed RF energy — in one published case crossing the 1 g SAR threshold the modeling authors themselves applied [22], and in another slightly more than doubling peak local electric-field strength [24].

What is not established. Whether realistic RF exposure changes release from dental metal or causes a health effect in living patients. The metal-release evidence is mixed and partly non-independent [14, 18–21]; no clinical effect is established.

The request. Commission metal-bearing dosimetry and blinded metal-release studies (Question 16). Adding handset-use and handset-position

questions to NHANES, which already collects dental and mercury data [5–7], would permit exploratory population analysis, not prove an RF effect.

Acronyms, statistical terms and units

Agencies, surveys and legal terms

- **CDC** — Centers for Disease Control and Prevention.
- **CFR** — Code of Federal Regulations; “47 CFR § 1.1310,” for example, means section 1.1310 of Title 47.
- **FDA** — U.S. Food and Drug Administration.
- **FCC** — Federal Communications Commission.
- **FR** — *Federal Register*; “91 FR 59792” identifies volume 91, page 59792.
- **HHS** — U.S. Department of Health and Human Services.
- **IARC** — International Agency for Research on Cancer.
- **NCHS** — National Center for Health Statistics, part of CDC.
- **NHANES** — National Health and Nutrition Examination Survey.
- **NIH** — National Institutes of Health.
- **OASH** — Office of the Assistant Secretary for Health (within HHS).
- **RFI** — request for information.

Exposure, methods and standards

- **CSF** — cerebrospinal fluid.
- **EMF** — electromagnetic field; **EMFs** is the plural.
- **FDTD** — finite-difference time-domain, a computational method for modeling electromagnetic fields.
- **GSM** — Global System for Mobile Communications, a cellular telecommunications standard.
- **ICNIRP** — International Commission on Non-Ionizing Radiation Protection.
- **IEC** — International Electrotechnical Commission; **IEC/IEEE 62704-1** is a jointly designated standard for computing SAR using FDTD methods.
- **IEEE** — Institute of Electrical and Electronics Engineers; **IEEE C95.1** is its human-exposure standard cited here.
- **MRI** — magnetic resonance imaging.
- **RF** — radiofrequency; **RF-EMF** means radiofrequency electromagnetic field.
- **SAR** — specific absorption rate, power absorbed per unit mass of tissue, usually expressed in watts per kilogram (W/kg). A 0.1 g, 1 g or

10 g value averages over a different mass and must not be compared directly with a limit specified for another mass.

Statistics and units

- **CI / OR** — confidence interval / odds ratio. **N** is sample size; **P** is a statistical *p* value.
 - **g / kg** — gram / kilogram; **mg/L / µg/L** — milligrams / micrograms per liter.
 - **Hz / MHz / GHz** — hertz / megahertz / gigahertz (frequency); **V/m** — volts per meter (electric-field strength); **W/kg** — watts per kilogram (SAR); **W/m²** — watts per square meter (power density).
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Question 2 — Perspective represented

I submit this comment **solely in my individual capacity** as an author and researcher working on the human health implications of implanted and restorative dental materials and on the regulatory frameworks that govern them. My published work includes a peer-reviewed review of titanium exposure and human health [25], a review of environmental mercury contamination from dental amalgam [10], analyses of the National Health and Nutrition Examination Survey (NHANES) mercury findings [9], and comparative analysis of U.S. Food and Drug Administration (FDA) and European Union regulatory positions on mercury dental amalgam [13].

I also write as an individual consumer. I submit no personal health information about myself or anyone else.

Disclosure of interest. So that the Department can weigh this response appropriately: I founded Californians for Green Dentistry, an advocacy organization concerned with mercury-free dentistry, and I am the petitioner on a citizen petition currently before FDA concerning dental amalgam (Docket FDA-2025-P-2526). I submit this comment in my own name, not on behalf of that organization. I receive no funding for this work. My advocacy position on dental amalgam is disclosed here. The basis of this response is peer-reviewed research, government guidance, exposure standards and regulatory texts; no personal health reports or industry data are submitted.

Existing frameworks on which this response relies (as requested in the notice)

- **FCC radiofrequency exposure limits**, 47 CFR § 1.1310 — spatial-peak specific absorption rate (SAR) of **1.6 W/kg averaged over 1 gram** of tissue for general population/uncontrolled exposure [37].
- **IEEE Std C95.1** (2005 and 2019 editions) and **ICNIRP Guidelines (2020)** [37, 38] — the standards that define spatial-peak SAR averaging. Both specify **2 W/kg averaged over 10 g** for general-

public head-and-torso exposure. The **1.6 W/kg averaged over 1 g** figure that governs in the United States is FCC's, codified at 47 CFR § 1.1310 [37]; it differs from the current IEEE and ICNIRP general-public values in both the limit and the averaging mass. That divergence is itself directly relevant to Question 5(p).

- **IEC/IEEE 62704-1** — a finite-difference time-domain (FDTD) standard for computational SAR assessment; it specifies a method, not an exposure limit. IEEE lists the **2026 edition as active**, approved 25 September 2026 and superseding the 2017 edition. Its scope extends from 100 kHz to 10 GHz, compared with 30 MHz to 6 GHz for the 2017 edition [43]. Studies [23] and [24] preceded this revision and cite IEC/IEEE 62704-1 as then available; [22] instead cites IEEE C95.1-2005 as its reference standard. The revision does not change those studies' reported findings.
 - **21 CFR subpart J** (Radiological Health; 21 CFR Parts 1010-1050) — the notice states that *"HHS is conducting this request for information under 21 CFR subpart J"* (91 FR 59792, 59793) — together with **21 CFR 872.3070** (dental amalgam, mercury, and amalgam alloy — class II, special controls), **21 CFR 872.3640** (endosseous dental implant — class II, special controls), and **21 CFR Part 872 subpart D** (prosthetic devices, including base metal alloys and preformed crowns) [36].
 - **Minamata Convention on Mercury**, Annex A, as amended at the sixth Conference of the Parties (Geneva, 3-7 November 2025) [12].
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Question 4 — Observations from research identifying patterns

4(h) — Types of effects reported, and 4(k) — observed exposure patterns

Metal dental work produces measurable systemic metal uptake.

Mercury. Dental amalgam contains approximately 50% mercury [11]; mercury release is measurable in people with restorations [5-7]. An analysis of 14,181 participants across two NHANES cycles found blood total mercury, blood inorganic mercury, and creatinine-corrected urinary mercury each strongly correlated with the number of amalgam restorations, and distinguished amalgam from dietary sources: seafood consumption raised blood total and methylmercury but, unlike amalgam, did not raise blood inorganic mercury. Among participants reporting no fish consumption, those with more than five amalgam restorations had blood total mercury 103% higher, blood inorganic mercury 221% higher, and urinary total mercury 221% higher than participants with no amalgam restorations [5]. Children under six with more than five restorations had the

highest blood inorganic and urinary total mercury of any age group [5]. A corrigendum corrected duplicated data in part of Table 4 but did not change the reported conclusions [5].

In a 2015–2018 NHANES analysis representing approximately **158.3 million adults**, about **91 million (57.8% of the adults represented in that analysis)** had at least one amalgam surface; surface count correlated with urinary mercury [6]. This percentage is for the study's analyzed population, not necessarily all U.S. adults. A further analysis of the 2015–2016 cycle found significant increases in blood total and inorganic mercury in adults with amalgams compared with those without [7]. A 2026 biomonitoring review identifies extensive amalgam restorations among sources of elevated mercury exposure, while cautioning that associations with neurological endpoints at typical environmental levels do not establish overt clinical neurotoxicity [8].

Nickel and chromium. In 795 patients who had recently received single or multiple nickel-chromium alloy restorations, compared with 198 controls, urinary nickel was significantly elevated in the group less than one month from restoration, and urinary chromium was significantly elevated in the groups less than one month, one to under three months, and three to under six months from restoration; within those groups, excretion was higher in patients with more teeth restored or with a greater index of uncovered metal crown surface [28]. This establishes internal exposure after placement, not persistent elevation or clinical toxicity.

Titanium. A systematic review of titanium particle and ion release in implant dentistry identifies release during implant bed preparation, insertion and decontamination, and continuing thereafter, with saliva, bacteria, chemical exposures, mechanical loading, the abutment micro-gap and fluoride among the factors that disrupt the protective oxide layer [1]. Metal and titanium particles have been detected in peri-implant tissues, and their possible role in peri-implant bone loss is under active investigation [2]. An integrative review of submicron- and nano-scale titanium debris reports cellular uptake and experimental effects on inflammatory signaling including IL-6, IL-1 β , TNF- α and PGE2, varying with particle size and concentration [3]. In a murine model, titanium dioxide micro- and nanoparticles were detected in plasma, lung, liver and kidney following exposure, and the smallest tested particles produced biochemical changes not reflected in conventional serum markers [4]. These are experimental findings under the conditions studied; they do not by themselves establish that comparable exposures occur in patients with dental implants.

Mixed metals. Coupling nickel-chromium alloy with titanium in vitro produces galvanic corrosion, with measurable ion release and associated cytotoxicity [31]. A published case attributed systemic allergic contact dermatitis to galvanic corrosion between amalgam and an adjacent titanium

implant; symptoms resolved after both restorations were removed [25]. Nickel-chromium crowns and titanium implant components can coexist; the combined configuration warrants separate study.

Reported RF-related metal release: preliminary and mixed.

A systematic review of physical stressors and mercury release from amalgam concluded that several physical exposures can affect mercury release or microleakage, but the individual studies it summarizes do not produce a uniform result: some reported no significant change following MRI or other electromagnetic exposures, while others reported increases under particular conditions [14].

Individual studies reporting increases:

- **In vitro, amalgam.** Twenty extracted human premolars restored with class V amalgam; one group exposed to a conventional 2.4 GHz Wi-Fi router for twenty minutes at 30 cm. Mercury in the surrounding artificial saliva was roughly double that of unexposed controls (0.056 ± 0.025 against 0.026 ± 0.008 mg/L, $P = 0.009$) [15]. **These were extracted teeth, not patients.**
- **Clinical, amalgam.** Changes in salivary mercury across an MRI examination in thirty patients, and, in a separate arm of the same paper, higher urinary mercury among mobile-phone users than controls [16]. The two results are in different matrices and different arms and must not be conflated.
- **Clinical, nickel — the strongest single study in this set.** A within-subject study of **fifty orthodontic patients** who abstained from mobile-phone use for one week and then recorded their usage during the following week found significantly higher salivary nickel after the usage week (paired $t[49] = 9.967$, $P < 0.001$) and a significant regression of nickel concentration on usage time ($F[1,48] = 60.263$, $P < 0.001$, $R^2 = 0.577$) [18]. This study was conducted independently of the amalgam research group. Salivary nickel was associated with recorded usage time, but exposure order was fixed, there was no sham condition, and diet and oral hygiene were not controlled. It establishes an association, not RF causation [18].
- **In vitro, nickel.** Stainless steel brackets exposed to a GSM 900 MHz source at SAR 2.287 W/kg released mean nickel of 11.95 $\mu\text{g/L}$ against 2.89 $\mu\text{g/L}$ in controls, roughly fourfold ($P = 0.001$) [19].
- **Clinical, nickel, exposure-modification.** Sixty patients: salivary nickel significantly lower among those using earphones than among those holding the handset against the head [20].
- **Null result.** A cross-sectional study of forty-two patients found nonsignificant differences in nickel and salivary pH despite an affirmative conclusion by its authors [21].

A mechanism for increased microleakage has been proposed; its authors present it as an evidence-based theory, but it has not been independently tested and should be treated as proposed rather than established [17].

Limits of the RF-release literature. The review [14] and several studies [15–17, 19] involve related investigators, not independent replications. The designs generally lack randomized, sham-controlled, temperature-monitored exposure and handset-relevant dosimetry. The large reported effects require blinded independent replication; a null result would leave the baseline metal exposure intact.

4(i) — Onset relative to exposure

In the nickel-chromium restoration cohort, urinary nickel elevation was confined to the first month after placement and chromium elevation to the first six months [28] — that is, onset is tied to placement, not to cumulative service life, in that study. In the within-subject phone study, the change in salivary nickel was measurable across a single week of recorded use following a week of abstention [18]. In the documented amalgam–titanium galvanic case, systemic dermatitis resolved within eight months of removing both restorations [25]. I have no data on onset of any RF-attributed symptom and make no claim about one.

4(j) — Characteristics of the populations concerned

In the weighted 2015–2018 NHANES analysis [6], approximately 91 million of 158.3 million represented adults (57.8%) had at least one amalgam surface. Nickel is a leading cause of allergic contact dermatitis [40]. For the under-six amalgam finding, see 4(h) [5]. No study cited here quantifies handset exposure specifically among orthodontic patients.

4(l) — Standardized methods used

Methods reported in the cited literature:

- **Finite-difference time-domain computational dosimetry** in anatomical head models, with SAR averaged over specified tissue masses [22, 23, 24] — averaging performed per IEC/IEEE 62704-1 in [23] and [24], and per IEEE C95.1-2005 in [22].
- **Inductively coupled plasma mass spectrometry** and atomic emission spectrometry for salivary and urinary metal quantification [18, 19, 20, 21, 28].
- **NHANES** survey design with creatinine-corrected urinary and whole-blood speciated mercury [5, 6, 7].
- **Intraoral galvanic current and potential measurement** between dissimilar dental metals [30, 31].
- **Cadaveric thermometry** with implanted devices under controlled multi-frequency exposure [26].

4(m) — Recommendations for future research or surveillance

The proposed methods and research priorities are in Questions 8, 10 and 16.

Question 5 — Existing standards, guidelines and regulatory frameworks federal health agencies should consider

Beyond the RF frameworks listed above (47 CFR § 1.1310; IEEE C95.1; ICNIRP 2020; IEC/IEEE 62704-1), I ask the Department to consider two frameworks that are not normally brought into RF discussions at all, and which are directly relevant because they govern the materials inside the absorbing volume:

1. The Minamata Convention on Mercury, as amended in November 2025. The regulatory trajectory for dental amalgam has moved from phase-down to phase-out. At the sixth Conference of the Parties (Geneva, 3–7 November 2025), Parties agreed to add dental amalgam to **Part I of Annex A** — the list of mercury-added products whose manufacture, import and export are prohibited — with a **phase-out date of 2034**, subject to an exception where use is deemed necessary by the dental practitioner based on the needs of the patient, and to strengthen Part II with binding requirements for Parties that have not yet phased amalgam out [12]. I cite this outcome as reported in Council of the European Union document 16701/25 (ENV 1370) of 10 December 2025, and I do not assert the decision number or entry-into-force date, because I was unable to retrieve the decision text itself. Earlier: the FDA recommended in September 2020 that certain higher-risk populations avoid amalgam where possible and appropriate [11]; Regulation (EU) 2017/852, Article 10, restricts amalgam in the European Union [12].

2. FDA device classifications. Amalgam, endosseous implants and dental prosthetic materials are covered by 21 CFR Part 872 [36]; FDA also administers electronic-product-radiation provisions under 21 CFR subpart J.

5(n) — Scientific, technical or regulatory changes that would improve assessment, disclosure and regulation

- 1. Study dental and craniofacial metal as an anatomical variable in RF dosimetry.** FCC rules permit either laboratory measurement or computational modeling to demonstrate SAR compliance, subject to validation and applicable protocols, under 47 CFR § 2.1093(d)(2) [42]. Tissue-simulant phantoms do not include dental metal; computational models can include it [22–24]. HHS can commission standardized comparisons without changing any exposure limit. Whether future

compliance procedures should require metal-bearing configurations is a separate question for FCC, informed by those results.

2. **Require that restoration and implant status be recorded in federally funded RF health studies.** It is a low-cost intake variable. If it proves unrelated to outcomes, that closes a hypothesis; if it modifies outcomes, it identifies a previously unmeasured source of heterogeneity. Either result is worth the cost of a checkbox.
3. **Re-examine the SAR averaging convention for small anatomical structures.** See 5(p) below.
4. **Connect the two regulatory pathways inside FDA.** A device-classification review of dental alloys that is required to consider RF interaction, and an electronic-product radiation review that is required to consider implanted conductive materials, would each be a change of process rather than a change of statute.

5(o) — Where additional research is needed, and the largest knowledge gaps

Two separate questions require study: **dosimetry** — whether the composition, geometry, location and condition of conductive dental or craniofacial materials reproducibly change local modeled RF fields or SAR under controlled conditions — and **material interaction** — whether realistic RF exposure changes release, corrosion or biological availability of material constituents. Neither result by itself would establish a health effect.

Specific gaps:

- No study has measured metal-ion or mercury release under **blinded, sham-controlled, dosimetrically characterized** RF exposure in living patients.
- No study has modelled a **set** dental amalgam restoration with correct composition (see Question 6).
- No study has characterized the SAR distribution around a **clinically relevant combination** — a titanium fixture with a nickel-chromium crown — as distinct from either material alone.
- No study has tested whether intraoral galvanic current, which is directly measurable, changes under RF exposure.

Question 16 ranks the corresponding experiments.

5(p) — How a classification system should account for exposure-related characteristics

Interpretation of the computational studies. These are comparisons within specified models, not evidence that users of compliant handsets exceed an applicable limit. Source power and position, frequency, anatomy,

implant geometry and averaging mass affect the result. A 0.1 g peak SAR cannot be compared with a 1 g or 10 g regulatory limit. In [24], modeled 0.1 g SAR exceeded the authors' Serbian national limit at 1 cm but complied from 2 cm; the electric field exceeded its 24.4 V/m reference level through 5 cm and fell below it throughout the model at 6 cm. **Those were not U.S. or ICNIRP limits** [24]. Standardized replication, not a regulatory verdict, is warranted.

Study	Material	MHz	Modeled result	Meaning
Cvetković 2020 [22]	Amalgam	900; 2600	1 g SAR (W/kg): 1.41 → 2.60; 0.91 → 1.74, respectively.	Crosses the authors' 1 g value only in the model.
Živaljević 2024 [23]	Cranial titanium implant	2600	Field/SAR increased near skin; decreased in deeper tissue.	Local shifts; no clinical outcome.
Jovanović 2025 [24]	Mandibular titanium plate	2600	Plate-adjacent 0.1 g SAR: 4.91 W/kg ($\sim \times 2.3$); sampled field: 61.58 → 138.42 V/m.	0.1 g is not comparable with 1 g or 10 g limits.

Model limits. Reference [22] used approximately 3% mercury (unmixed alloy powder rather than set amalgam) and a continuous 1 W source, so its absolute SAR values do not establish handset compliance in patients. Reference [24] supplies no absolute no-implant baseline for the plate-adjacent 0.1 g peak: its $\times 2.3$ ratio compares that peak with the no-implant peak. Separately, its *global skin maximum* exceeded the authors' national limit by $2.7\times$ without and $2.8\times$ with the implant. **The no-implant model already exceeded that limit; [24] does not show a metal-induced crossing.** None of the studies models a titanium fixture with a nickel-chromium crown.

Averaging mass. The cited FCC limit uses 1 g; current IEEE and ICNIRP general-public head-and-torso limits use 10 g [37, 38]. Modeling at 0.1 g can reveal local maxima near small implants but cannot establish compliance against either larger-mass limit. Report results at each mass separately.

Direction differs by tissue. The cranial implant model found greater local field and SAR in skin, fat and muscle but lower values in skull, cerebrospinal fluid and brain [23]. This shows redistribution, not greater whole-brain RF exposure.

A classification system that accounts for metal in the absorbing volume would therefore need: presence, material, geometry and location of dental or craniofacial metal; whether dissimilar metals are in contact; and an averaging mass appropriate to the structure being assessed.

Question 6 — Scientific evidence about potential adverse health effects caused by current exposure limits

I have no evidence that current exposure limits cause adverse health effects, and I do not assert that they do. I have evidence bearing on a narrower and more answerable question: **whether exposure assessments adequately represent people with permanent dental or craniofacial metal.**

In [22], the amalgam-free model remained below the authors' applied 1.6 W/kg (1 g) limit at both frequencies, while the amalgam-bearing model exceeded it. The authors conclude that *"the obtained results exceed the safety limits only when the amalgam is present"* [22].

This does not establish that actual handset users exceed an applicable limit. The model used a continuous 1 W source and the composition of unmixed alloy powder rather than set amalgam; compliance also depends on the prescribed measurement and averaging scenario (see Question 5(p)). The threshold crossing is within the model, not measured in patients [22].

Question 7 — Scientific methods currently available for characterizing individual and population-level RF exposure

Available and adequate:

- **Computational dosimetry** can represent dental metal, as the cited head models show [22-24]. References [23] and [24] use IEC/IEEE 62704-1; [22] cites IEEE C95.1-2005.
- **Tissue-simulant phantom measurement** uses metal-free phantoms; computational modeling is also permitted by 47 CFR § 2.1093(d)(2) [42].
- **Personal exposimetry** for ambient RF.

- **Biomonitoring of the consequence rather than the field:** salivary and urinary metal quantification by inductively coupled plasma mass spectrometry [18-21, 28], and whole-blood speciated mercury and creatinine-corrected urinary mercury at national scale through NHANES [5, 6, 7].
- **Intraoral galvanic current and potential measurement,** an objective, non-invasive measure directly relevant to mixed-metal restorations [30, 31].

Gap: Routine individual exposure assessment does not account for the geometry of each person's dental hardware; doing so requires individualized modeling.

Question 8 — Improvements needed in exposure assessment methodology

1. **Include metal-bearing head configurations in computational dosimetry.** Existing rules permit modeling [42]; validated reference configurations are needed [22-24].
 2. **Publish reference voxel models** for set amalgam, nickel-chromium crowns, orthodontic steel, titanium fixtures and mixed-metal assemblies. Correct the composition used in [22].
 3. **Resolve the averaging-mass question explicitly** rather than by default. Report 10 g, 1 g and sub-gram values side by side for metal-bearing configurations, and state in the published result whether coarser averaging obscures a local maximum.
 4. **Record restoration and implant status as an intake variable** in RF exposure studies.
 5. **Adopt blinded, sham-controlled, temperature-monitored protocols** for any further metal-release work. The existing studies' failure to do this is the main reason their findings cannot yet carry weight [14-17, 19].
 6. **Measure delivered exposure** — handset position, duty cycle and actual separation distance; the modeled SAR result in [24] changed between 1 and 2 cm.
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Question 9 — Technical and exposure-related information that should be disclosed

To patients: disclose the composition of restorative materials, including mercury in amalgam and nickel in relevant alloys [28], and the possibility of galvanic corrosion where dissimilar metals contact [25, 31]. Nickel sensitization is a separate, established concern [40]. Do not present an unproven RF interaction as a clinical risk.

To dentists and physicians: report the preliminary RF-release findings and their limitations [15, 18, 19]. One study found lower salivary nickel among earphone users than handset-to-head users [20]; it does not establish a clinical health benefit.

To device manufacturers and regulators: report whether the modeled SAR compliance margin changes when dental metal is included [22–24].

To schools: if addressing orthodontic hardware in wireless-use guidance, label the RF-interaction question unresolved, not a demonstrated hazard.

Question 10 — Data sources and surveillance systems

10(e) — Biomonitoring. NHANES combines dental examinations with blood and urinary mercury data [5–7] but lacks a handset-use measure. Adding handset-use and position questions would allow exploratory analysis of associations; self-reported use alone would not measure absorbed dose or establish causation.

10(f) — Adverse event reporting. FDA’s MedWatch and Manufacturer and User Facility Device Experience systems can receive dental-device reports. Symptom reports alone cannot quantify RF exposure or establish that a dental material caused an event; a concurrent-exposure field would help characterize reported cases.

10(b) — Occupational monitoring. Dental personnel are occupationally exposed to mercury and to alloy dust; the exposure is already recognized. Linking that monitoring to RF exposure is not a priority relative to (e).

10(d) — Longitudinal cohort studies. Record dental-metal status, handset use and position prospectively, with repeated metal biomarkers. This could test temporal associations but would still require control of confounding.

10(h) — Dental-material surveillance. NHANES records dental findings but not detailed restoration and implant materials suitable for the proposed exposure analysis.

A documented precedent for why passive surveillance is insufficient here. Zinc in denture adhesive — measured at 17,000 to 34,000 µg/g in Fixodent and Poli-Grip — produced hypocupremia and serious neurologic disease in chronic heavy users. It was identified through a **four-patient neurology case series** published in 2008, after the authors could find no alternative source of excess zinc; serum zinc improved in three patients after they stopped using the cream [35]. The case series illustrates a limitation of passive surveillance, not an RF effect.

Question 11 — Whether exposure assessment or risk evaluation should differ for sensitive populations

11(a) — Children. The under-six amalgam finding [5] and FDA’s 2020 advice identifying children as a higher-risk group for amalgam [11] support age-specific assessment. Smaller anatomy could alter source-to-metal distance, but no age-resolved RF dosimetry cited here measures that effect. Orthodontic hardware in adolescents is another testable configuration; no cited study quantifies their combined metal and handset exposure.

11(b) — Pregnant women and women intending pregnancy. FDA identifies these groups in its amalgam advice [11]. No cited study shows that RF changes their mercury exposure.

11(d) — Implanted medical devices. Endosseous dental implants are regulated under 21 CFR 872.3640, which describes a prescription device surgically placed in jaw bone; dental restorations are separately regulated under Part 872 [36]. The modeling evidence [22–24] supports testing how conductive dental hardware affects local RF distribution. Radiotherapy and MRI account for metal in other exposure settings [25], but their fields and doses are not clinically equivalent to handset RF exposure.

11(f) — Preexisting conditions. Nickel-sensitized patients are an identifiable group for a blinded study of nickel release and clinical endpoints [18, 40]. A separate meta-analysis of *amalgam-related* oral lichenoid lesions reported greater resolution after replacement (eleven pooled studies, N = 365; OR 122.18, 95% CI 55.64–268.26), but the authors noted heterogeneity and publication bias [41]. That result concerns amalgam and cannot establish an RF-nickel health effect.

11(e) — Workers. No cited study assesses occupational dental-metal exposure together with RF.

Question 12 — Scientific evidence regarding adverse health effects below current federal exposure limits

Health effects versus exposure metrics. No cited study establishes that current RF limits cause adverse health effects through interactions with dental or craniofacial metals, that ordinary RF exposure causes clinically significant metal toxicity, or that compliant handsets become noncompliant in users with such metal. The separate, testable question is whether the materials change localized modeled fields or material release under realistic exposures. A modeled change is not itself a health effect.

Dosimetry. In one dental modeling study, the no-amalgam configuration complied with the authors' applied 1 g limit while the amalgam configuration exceeded it [22]. In another, the model with a titanium plate exceeded the authors' national limit at 0.1 g averaging and 1 cm separation but complied at 2 cm [24]; its *no-implant model also exceeded that national limit at the baseline distance*, as explained under Question 5. **No U.S. compliance limit is expressed at a sub-gram averaging mass.** Neither study measured a health endpoint. The defensible observation is that metal and distance altered modeled exposure; only [22] demonstrated a within-model threshold crossing attributable to the metal.

Candidate mechanism. A review reported oxidative endpoints in 93 of 100 identified low-intensity RF studies [27]. Study-counting is vulnerable to publication bias and differences in quality, exposures and endpoints; it neither establishes uniform effects nor a clinical risk. Metal-particle studies also report oxidative or inflammatory endpoints [3, 4]. An interaction remains a hypothesis to test by independently varying RF exposure and metal presence.

Negative heating study. Eight orthopaedic devices in six cadavers were exposed for six minutes to a multifrequency source at 50 and 100 W/m². The observed temperature rise was below 0.2 °C, with no significant heating [26]. This constrains claims about bulk heating under those conditions; it does not measure SAR around small dental structures.

Cancer classifications do not establish a combined risk. IARC classifies RF fields as Group 2B (possibly carcinogenic), nickel compounds as Group 1, and metallic nickel and alloys as Group 2B [29, 33]. The nickel-compound evidence is occupational, not evidence that nickel dental restorations cause cancer. A narrative review proposes mucosal effects from low-frequency oral galvanism [30], but its electrical mechanism cannot be transferred to handset radiofrequencies or used to infer cancer incidence.

Recording restoration status in RF health studies would allow investigators to test whether it modifies observed associations.

Question 13 — How specific technologies affect cumulative exposure patterns

13(c) — Wi-Fi. An in-vitro study exposed amalgam-restored teeth to a 2.4 GHz router at 30 cm for twenty minutes and measured higher mercury in artificial saliva than in controls [15]. Its conditions do not quantify ordinary Wi-Fi exposure or effects in patients.

13(f) — Wearable technologies. Source-to-metal distance affected modeled SAR in [24]. A clinical comparison found lower salivary nickel with

earphone use than with handset-to-head use [20], but it cannot establish effects for every wired or wireless earphone configuration; delivered exposure must be measured.

13(j) — Wireless medical devices. Dental hardware is not itself wireless, but conductive components altered fields in the cited models [22–24]. These results do not characterize interactions with a specific wireless medical device.

Other technologies listed in Question 13. The cited models use 900 or 2600 MHz [22–24]. They do not establish the direction or magnitude of an effect for 5G, 6G, satellite links, connected devices or millimeter-wave frequencies.

Question 16 — Highest-priority research gaps

In priority order, with the cheapest first:

1. **Add handset-use and handset-position items to NHANES** alongside existing dental and mercury measures [5–7], for exploratory population analysis rather than causal inference.
 2. **Publish standardized metal-bearing voxel models; re-run [22] with set-amalgam composition and handset-relevant exposure.**
 3. **Model the clinically ordinary combination** — titanium fixture with nickel-chromium crown [31] — which no published study has characterized, and report results at 10 g, 1 g and sub-gram averaging side by side [22, 24].
 4. **Replicate the orthodontic nickel-release finding** with randomized order, sham control and measured delivered RF exposure in living patients [18].
 5. **Test nickel-sensitized participants for relevant clinical endpoints** if an independently replicated RF-related nickel-release effect is found [18, 19, 40].
 6. **Determine whether intraoral galvanic current changes under RF exposure.** Directly measurable [30, 31], never tested.
 7. **Determine whether metal-bearing configurations affect compliance margins at millimeter-wave frequencies.**
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Question 17 — Improving federal coordination of EMF and RF research

17(a) — FDA. FDA has responsibilities for radiation-emitting electronic products within its statutory authority and for dental devices and materials under 21 CFR Part 872 [36]; it does not administer FCC handset RF

exposure limits. FDA could assess whether dental-device evaluation and adverse-event reporting should account for concurrent RF exposure. Its existing amalgam advice identifies certain higher-risk groups [11].

17(b) — NIH. A joint program through the National Institute of Dental and Craniofacial Research and the National Institute of Environmental Health Sciences could fund the modeling and blinded metal-release studies in Question 16.

17(c) — CDC/NCHS. Consider adding handset-use items and more detailed dental-material information to NHANES (Questions 10 and 16).

17(d) — FCC. FCC establishes and administers the relevant RF exposure rules for covered communications equipment [37, 42]. In coordination with HHS and FDA, it could evaluate whether standardized metal-bearing head configurations materially change modeled compliance margins and publish the outcome. This requests evidence, not a transfer of authority or a change to existing limits.

Question 18 — Additional scientific evidence, technical information, data, or recommendations

Electromagnetic hypersensitivity. An updated review of 46 blind or double-blind provocation experiments involving 1,175 self-identified sensitive volunteers found no reliable evidence that controlled exposure triggered symptoms; the studies supported a contribution from expectation or nocebo effects to acute symptoms [34].

A self-selected survey of 142 respondents reported dental-amalgam checks and removal, with reported symptom changes in both directions [32]. It lacked a control group, diagnostic verification and exposure measurements; it cannot support treatment recommendations or causal claims.

RF-symptom studies could record dental-metal status and, when relevant, intraoral galvanic measurements as covariates, without presuming causation [30–32].

Separate environmental issue. Mercury from dental amalgam enters environmental pathways through cremation, wastewater and other disposal routes [10]. This concerns the material, not environmental effects of RF radiation; I do not present it as an answer to Question 14.

Questions not answered: 3 (personal medical experience), 14 (environmental RF effects) and 15 (infrastructure proximity). I submit no evidence on them.

Conclusion and request. Conductive craniofacial materials alter local electromagnetic-field and SAR distributions in published models [22–24]. Evidence of RF-related effects on material release is limited and mixed [14, 18–21]; no adverse health effect from the proposed interaction is established. HHS should encourage coordinated, preregistered research using realistic anatomical models, blinded and sham-controlled release experiments, and appropriately interpreted population data (Question 16). The aim is to measure any reproducible effect and determine whether it has biological or clinical significance, not to assume an outcome.

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(data duplication errors in one section of Table 4, corrected online; reported findings and conclusions unchanged).

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