

Comment of Len Ber, MD

Medical Director, Civilian Registry for Diagnosed Havana Syndrome Patients

Docket: HHS-OASH-2026-0397. Request for Information on Electromagnetic Fields (EMFs), Radiofrequency (RF) Radiation, and Wireless Radiation Exposure. 91 FR 59792 (FR Doc. 2026-19252), 21 September 2026.

Submitted by: Len Ber, MD, Medical Director, Civilian Registry for Diagnosed Havana Syndrome Patients

Date: 15 October 2026

Main points. Diagnosed civilian Havana Syndrome / Anomalous Health Incident (AHI) patients in the United States have no federal case definition, reporting pathway, or clinician guidance. This comment makes two related but separate points:

1. **Recognize AHI1 as a novel clinical phenotype.** I ask HHS to recognize the core clinical phenotype (AHI1), also known as Havana Syndrome, as a novel phenotype diagnosed by expert physicians on objective findings, without deciding its cause or attribution. The Intelligence Community (IC) Experts Panel described its four core characteristics as a combination that "is distinctly unusual, is unreported elsewhere in the medical literature" [1, FOIA pp. 007–008]; the 2024 NIH studies defined AHI1 by those characteristics [2,3]; and diagnosed civilian patients are documented in the registry I direct [4,5] (Section 2). HHS should give diagnosed civilians a public-health home.
2. **Current safety standards are not adequate for the exposure parameters attributed to this condition.** Directed, pulsed RF energy is the leading scientific explanation advanced for Havana Syndrome/AHI1, and expert judgment has moved from plausibility toward probability: the National Academies called it "the most plausible mechanism" in 2020 [6], the IC Experts Panel found pulsed RF a plausible explanation of the core characteristics in 2022 [7; 1, FOIA p. 008], and in 2025 Giordano and colleagues wrote: "It is now widely accepted that directed energy exposure is the most probable cause" [8]. This comment addresses the pulsed-RF exposure parameters at the person, such as peak power density, field strength, pulse width, and repetition rate, as described by the IC Experts Panel, Dagro and colleagues, and others [1,9]. The federal RF limits that HHS and FDA rely on are time-averaged and designed to prevent tissue heating; they set no separate limits on peak power density, pulse width, pulse repetition frequency (PRF), energy per pulse, or modulation [10–12]. This comment concerns their health effects, not their source.

Section 1. Respondent and the Civilian Registry [Q2]

[Q2] I submit this comment as the Medical Director of the Civilian Registry for Diagnosed Havana Syndrome Patients (CRHS). (In this comment, civilians are people outside the federal workforce and their families; federal employees are also civilians in U.S. Government Accountability Office (GAO) usage [13].) I established the registry in August 2024, in the absence of federal civilian surveillance, to document and analyze physician-diagnosed cases in the U.S. civilian population arising from incidents on U.S. soil [4,5]. It has issued two public reports: the January 2025 interim report [4] and the January 2026 update [5]. My perspective comes from monitoring clinical diagnoses of these patients and the medical literature. This comment contains no personally identifiable or personal health information.

[Q2, Q11(g)] The registry counts as diagnosed only patients who provide medical documentation of a physician diagnosis of Havana Syndrome or an equivalent diagnosis [5]:

- **January 2025 report:** Seven diagnosed civilians (three male, four female), including one minor, in five states (Arkansas, Illinois, Colorado, New York, and Rhode Island), diagnosed between 2020 and 2024 [4,5,14].
- **January 2026 update:** Fourteen diagnosed civilians, double the January 2025 count. Seventy percent were female; median age rose from 50 to 53; the registry still included one diagnosed teenager. Florida had the most diagnosed patients, followed by New York. New diagnoses averaged 3.2 per year in 2022–2025. All diagnosed patients reported recurrent or ongoing incidents. One is on permanent disability attributed to Havana Syndrome [5].
- **As of this writing (October 2026),** the registry includes 21 physician-diagnosed civilian patients.

These are counts of diagnosed patients who chose to come forward, not prevalence estimates, and U.S. health agencies have issued no case definition.

Section 2. Request: Recognize the AHI1 Clinical Phenotype [Q4(h), Q4(i), Q4(l), Q10(a), Q11(g), Q17(b), Q17(c)]

[Q4(h), Q4(i)] "Anomalous Health Incident" is an administrative label used for incident reporting and benefit eligibility, which is limited to covered federal employees and their dependents (for example under FECA Bulletin 22-03 [15]; the HAVANA Act itself covers "qualifying injuries to the brain" [16]). It is not a diagnosis. Within the cases carrying that label is a clinical phenotype, AHI1, that expert physicians have diagnosed as Havana Syndrome or an equivalent diagnosis on objective findings and that had not been described before 2016. The 2024 NIH studies defined AHI1 as cases consistent with the four core characteristics used by the IC Experts Panel [2,3]; the Panel found that "A disturbance of the auditory or vestibular components of the ear could explain the core characteristics" [1, FOIA p. 017]. AHI1, as I use it, is the core-characteristics subset with objective otolithic and other findings. AHI2 refers to reported AHIs that do not fit the four core characteristics and lack otolithic findings. The

evidence for AHI1:

(a) Core characteristics. The Panel described acute onset of audio-vestibular sensory phenomena, sometimes including sound or pressure in only one ear or on one side of the head; other nearly simultaneous signs and symptoms such as vertigo, loss of balance, and ear pain; a strong sense of locality or directionality; and the absence of known environmental or medical conditions that could explain them [1, FOIA p. 007]. It found that this combination "is distinctly unusual, is unreported elsewhere in the medical literature," and that several aspects of "this unique neurosensory syndrome" make a functional neurological disorder unlikely [1, FOIA p. 008]. What makes it unique is not any single symptom but the fact that they occur together, which is unprecedented.

(b) Pressure, vibration, and "buffeting" at onset. In the original Havana cases, a pressure-like or vibratory sensation accompanying the onset sound, likened by patients to air "baffling" [sic] inside a moving car with the windows partially rolled down, was reported as "pressurelike (n = 9, 43%) or vibratory (n = 3, 14%)" among the 21 personnel evaluated at the University of Pennsylvania [17]; the National Academies reported that, of the 35 Havana individuals assessed at Penn, 16 reported "a sensation of pressure or vibration in the head" [6]. These overlapping cohorts predate the AHI1 definition; the Penn series, as part of Verma et al.'s cohort [18], in my assessment meets its criteria retrospectively, and the figures are not pooled. A peer-reviewed article describes, from the first-person perspective of a diagnosed civilian: "To power through the most severe attacks, I would scream in order to 'push' against extremely painful sensation of buffeting. It feels as if your head is being squeezed in a vibrating vice" [19, p. 5]. Buffeting or vibrating pressure is the most distinctive sensation in AHI1 patients and is not commonly described in the medical symptom literature.

(c) Early blood biomarkers. The Panel reported "early, transient elevations in biomarkers suggestive of cellular injury to the nervous system" in "the small number of affected individuals who were tested within an appropriate time period" [1, FOIA p. 008]. The markers were glial fibrillary acidic protein (GFAP) and neurofilament light chain (NfL), with a time course matching mild traumatic brain injury though at apparently lower levels; it called these data preliminary [1, FOIA pp. 014–015]. The Panel recommended testing "as soon as possible after an event, ideally within hours" [1, FOIA p. 009], and its appendices state that ubiquitin carboxyl-terminal hydrolase L1 (UCHL1), a brain injury biomarker that "was not included in assays performed to date" [1, FOIA p. 114], "should be included in any diagnostic panel" [1, FOIA p. 074].

(d) Otolithic injury. The defining objective finding is otolithic. In the University of Miami acute series, 25 individuals who reported a localized noise or pressure were compared with 10 asymptomatic roommates; "100% of individuals had an otolithic abnormality and evidence of cognitive dysfunction," and the authors described "the universal nature of vestibular injuries" [20]. In the University of Pennsylvania series, objective findings included vestibular abnormalities in 81% [17].

(e) Oculomotor and pupillary findings. In the same University of Pennsylvania series, clinical examination raised concern for oculomotor dysfunction in 15 individuals (71%), prompting referral to neuro-optometry; formal testing confirmed convergence insufficiency (52%), abnormal smooth pursuit (52%), and saccadic dysfunction (47%) [17]. In the acute Miami cohort, filmed eye-movement testing included smooth pursuit, horizontal, predictive, and anti-saccades, optokinetic response, and vergence [20, p. 124]; 13 of 25 affected patients (52%) had an abnormal anti-saccade error rate, and central vestibular testing (optokinetic, saccadic, and fixation tests) was abnormal in 9 (36%) [20, p. 127]. A later retrospective analysis of quantitative video-oculography of vergence and pupil responses in 19 of these Havana diplomatic personnel and family members, tested in the acute period, showed patterns "markedly different" from 17 patients with acute mild traumatic brain injury and from 62 controls; a discriminant classifier separated the groups with 91.8% overall accuracy (81.6% cross-validated) and "no confusion" between the Havana and concussion groups [21].

(f) Brain imaging and network-level disruptions. Advanced MRI by Verma et al. (2019) in 40 U.S. patients showed significantly smaller whole-brain white-matter volume and differences in cerebellar microstructure and functional connectivity compared with controls, and higher fractional anisotropy in the splenium of the corpus callosum at an uncorrected threshold [18]. In Canadian diplomats and family members posted in Havana, fixel-based diffusion analysis showed reduced fibre density in the fornix and splenium in the exposed group ($n = 16$), correlated with persistent migraine, tinnitus, sound sensitivity, and fatigue [22]; the same group's earlier multimodal study, posted as a preprint, reported transient "paroxysmal slow-wave events" on magnetoencephalography (MEG) in exposed individuals, "diffusely distributed in both hemispheres, more prominently on the right" [23]. My peer-reviewed re-analysis of the AHI1 rows of the published NIH neuroimaging data (43 AHI1 participants versus 48 controls) found significantly reduced salience-network connectivity (adjusted $p \approx 0.02\text{--}0.03$) and nominal (uncorrected) midline white-matter changes, including a small ($\sim 2\%$) return-to-axis probability decrease in the splenium [24, p. 9]. It reports that these changes parallel Verma et al., both pointing to "key midline and long-range white matter tracts (notably the corpus callosum, cerebellar connections, and cingulum)" as "common loci of change" [24, p. 10; 18], though the matched structures and measures differ. The splenium of the corpus callosum is thus the one anatomical structure all three studies converge on, though the direction of change differs (higher anisotropy in [18], lower fibre density and return-to-axis probability in [22] and [24]), and the cohorts were analyzed separately and are not pooled (my analysis; see Section 8).

(g) Cognitive deficits. Cognitive deficits begin during or around the incidents and are chronic, persistent, in some reports progressive. In the acute Miami series, examined "approximately 4 to 60 days after the most recent exposure" [20, p. 125], "All of the individuals noticed unsteadiness and cognitive symptoms (feelings of disorientation, lack of mental clarity, slower speed of processing, difficulty sustaining attention) within 18 hours of the exposure that produced ear pain" [20, p. 127], and testing of 9 individuals showed decrements "on measures of verbal fluency, working memory and sustained attention/vigilance" [20]. In the University of

Pennsylvania series, evaluated a mean of 203 days after exposure, "Persistent cognitive manifestations were reported by 17 individuals (81%)" [17, p. E4]; multidisciplinary evaluations raised concern for cognitive impairment in 16 individuals (76%), and the 6 with complete neuropsychological testing there "all had significant areas of cognitive weakness and/or impairment" [17]. In Canadian diplomats, a preprint reported "significantly lower performance in spatial working memory" [23]. In my re-analysis, the syndrome's sudden onset is "followed by chronic deficits" [24, p. 8], and I reported that civilians "are continuing to suffer progressive neurocognitive debilitation" [24, p. 12].

Defining this phenotype has been a serious and complex undertaking. Giordano and colleagues wrote that "The diagnostic process for Havana Syndrome AHIs remains complex and multifactorial" and proposed "modular artificial intelligence approaches, particularly those leveraging machine learning and predictive analytics," to integrate neuroimaging, cognitive, auditory/vestibular, and biomarker data [8, p. 1]; they noted that "diagnostic criteria continue to evolve" [8, p. 3]. Giordano has also written that AHIs "have presented a complex diagnostic challenge for both clinicians and government entities" and that "Diagnostic efforts have been hindered by heterogeneity in symptom reporting and the absence of standardized assessment tools" [25, p. 1]. None of these efforts currently sits in the public health domain.

[Q4(l), Q10(a), Q11(g)] The phenotype can be diagnosed without deciding its cause or attribution: from the history and distinct complaints (2(a)), and from objective otolithic, oculomotor (on clinical and neuro-optometric examination [17] and quantitative video-oculography [21]), cognitive, and imaging findings, with timely blood biomarkers when available. AHI1 must be analyzed separately from AHI2 and never pooled with it; pooling dilutes the signal [24].

[Q17(b), Q17(c)] Request. HHS, CDC, and NIH should formally recognize the Havana Syndrome AHI1 clinical phenotype as diagnosed by expert physicians on objective findings, so that civilian patients can be counted, referred, and studied without resolving attribution (Recommended HHS Action 1).

[Q4(h), Q4(i), Q17(c)] Naming. Havana Syndrome is the most widely used term, AHI is an administrative label, and AHI1 is a phenotype placeholder. The naming gap is real and runs across sources:

- The IC Experts Panel called the core characteristics "a preliminary definition of an AHI neurosensory syndrome," "reproducible, as well as unique," but left it preliminary and unnamed [1, FOIA p. 014].
- Clinical investigators have used descriptions only [17,18,20,21]; Hoffer and Balaban used "acquired neurosensory dysfunction" in their paper titles as a descriptive term, not as a proposed disease name [20,21]. CDC's 2019 investigation likewise described only "a symptom cluster" following "an unusual auditory or sensory event," with case definitions but no name [26].
- Even in recent articles on AHI treatment (one peer reviewed, one preprint), the condition

is not named; the vague administrative label AHI is used instead [27,28].

- Military medicine has used "unconventionally acquired brain injury (UBI)," a term defined in a 2020 U.S. Special Operations Command policy memorandum and described in a 2021 military medicine article [29]; the 2025 clinical guidance of the Defense Health Agency (DHA) uses "AHI" and states that "AHI has also been referred to as" Havana syndrome and "Unconventionally acquired brain injury (UBI)" [30, p. 3].
- The Department of Labor's FECA Bulletin 22-03 does not name the condition; it notes that the symptoms "have since been collectively referred to as Havana Syndrome," and, because "symptoms are not compensable under the FECA," directs claims examiners to accept "a diagnosed traumatic brain injury, ICD-10 S06.301A" [15]. That code denotes a focal traumatic brain injury with loss of consciousness, which is not among the core characteristics; the DHA guidance instead treats witnessed loss of consciousness as a red flag and codes AHI as R44.9 or R29.90, both symptom codes [30, pp. 5, 11]. A 2024 federal rule states: "There is no diagnostic code or criteria for AHIs in the International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM)" [31, p. 28634].
- On 10 July 2026, the Department of War (the Department of Defense, DoD) renamed its AHI Cross-Functional Team the Directed Energy Bio-Effects (DEBE) Cross-Functional Team, describing the change as applying "a more dedicated warfighter and medical focus" to "non-kinetic threats" [32]. That label names a mechanism without describing the clinical condition, and suggests that the cause may be regarded as established within military medicine; if so, the supporting evidence has not been shared with civilian medicine.
- Building on UBI, I proposed non-kinetic brain injury (NKBI) as a placeholder name [24, p. 8], supported by network-level disruptions and advanced neuroimaging findings rather than the macroscopic lesions expected in traumatic brain injury (TBI) or concussion [18,24]. The term fits with the Department's focus on non-kinetic threats while naming the clinical injury rather than a mechanism, when findings support a diffuse form of brain injury.

I ask HHS to adopt a phenotype-based name that does not presuppose a cause until additional data resolve it.

Section 3. Why This Comment Treats Havana Syndrome as a Pulsed-RF Exposure Problem [Q5(p), Q6, Q11(g), Q12]

[Q6, Q12] I do not ask HHS to decide attribution or source. I ask it to recognize the clinical phenotype (Section 2) and to recognize that expert bodies most often propose pulsed RF as the mechanism. That places Havana Syndrome under HHS's own question about non-thermal effects of pulsed and modulated signals:

1. The National Academies found that "directed pulsed RF energy ... appears to be the most plausible mechanism in explaining these cases among those that the committee

considered" [6].

2. The IC Experts Panel found bulk heating "an implausible pathway" and rated thermo-acoustic effects, pressure waves from "rapid thermal expansion of the affected tissue (but not bulk heating)," "a plausible pathway, with information gaps" [1, FOIA p. 036]; it notes this effect "can be a subthermal effect" [1, FOIA p. 111]. It also rated strong fields and resonant disruption plausible (Section 5). Its declassified executive summary reached the same overall judgment [7].
3. The registry's January 2026 update treats Havana Syndrome as a distinct neurological syndrome with a clinically validated AHI1 phenotype, separable from attribution debates, and finds the leading mechanism "converging on directed, pulsed radiofrequency exposure" [5].
4. Computational modeling shows that single microsecond pulses that heat tissue by less than a thousandth of a degree can produce potentially injurious stress waves deep in the brain while remaining "permissible by current safety standards" as judged against time-averaged limits [9] (Section 5).

The Department of War's renaming of its AHI team to Directed Energy Bio-Effects (Section 2) points the same way. If that mechanism is involved in any case, the exposure is likely invisible to a time-averaged, heating-based limit. That is a gap in the standards HHS is asking about, whatever the source of any particular exposure. Two prior Intelligence Community Assessments on AHI (ICA 2023-02286 and ICA 2025-00008) were ordered rescinded in June 2026 for failing to meet IC analytic standards [33]; the 2022 IC Experts Panel assessment relied on throughout this comment was not (Section 8).

Section 4. Federal Public-Health Response to Diagnosed Civilians [Q10, Q17(c)]

[Q10, Q17(c)] The federal record on diagnosed civilians:

- **CDC's own record.** In December 2019 CDC completed its *Cuba Unexplained Events Investigation* final report for the Department of State. From the records of 95 evaluated persons it applied stratified case definitions (15 presumptive, 31 possible, 49 not likely cases); onset timing and a biphasic course were criteria; federal employment was not [26]. That report treated the condition as a public-health issue. In response to my November 2024 FOIA request (CDC/ATSDR No. 25-00184-FOIA), CDC released a 25 March 2022 e-mail from its Center for Global Health to overseas country directors and deputies, asking them to "share this with all CDC USDH [U.S. direct-hire] staff in your country" and transmitting "CDC Anomalous Health Incidents (AHI) Guidance" (not a draft) [34, FOIA pp. 4–5]. That guidance was addressed to the CDC workforce, not to the public or civilian clinicians. Separately, CDC withheld "a draft AHI guidance document" [34]. CDC also reported that "the CDC has not developed a registry for AHI"; the registry "is maintained by Department of Defense" [34, final letter p. 2].
- **Inspector General and state referrals.** In March 2025 I asked the Defense Health Agency Office of Inspector General Hotline to address the civilian registry report, which

urged CDC to track diagnosed civilian cases. It closed the matter on 12 March 2025, stating that "DHA OIG Hotline does not have purview over tracking of Havana Syndrome with any populace" [35]. According to the registry's January 2026 update, physicians for four diagnosed patients notified their state epidemiologists and asked for CDC guidance; no responses had been received at the time of that update [5]. Since that update, one has reported receiving a reply: a county health department, after consulting CDC, asked whether the patient had been in the military or a government employee, relayed that CDC's experience with AHI was "typically among individuals in the military or who are federal government employees," and advised only that clinicians "continue to treat the patient's symptoms" [36].

- **Petition to CDC.** On 29 September 2025 I petitioned CDC, by certified mail, to act on civilian Havana Syndrome: recognize it as a reportable condition among civilians; create or expand national surveillance to include civilian cases; publish diagnostic guidance for physicians; issue Health Alert Network bulletins and train public-health officials; and coordinate civilian access to programs equivalent to those for federal employees. The petition cites both the 2019 CDC case definition and the January 2025 Civilian Registry Report [14]. CDC-INFO answered that "this question is out of scope for CDC-INFO" [5]. On 15 July 2026 the CDC Executive Secretariat wrote that CDC had received the petition, that "the request falls outside the agency's purview to respond, so did not respond," and that "CDC did, however, forward your petition to the National Institutes of Health for their consideration" [37].
- **Petition to NIH.** On the same date I petitioned NIH for recognition that AHI affects civilians, inclusion of civilians in AHI research and a national civilian registry, retraction and re-issuance of the two 2024 JAMA studies, clinical evaluation guidelines for civilian providers, outreach and a referral pathway, and access for civilians to NIH clinical care and treatment protocols [38]. As of October 2026, NIH has not responded to either the petition I sent or the copy CDC forwarded.

[Q17(c)] Litigation. On 23 June 2026, I and other plaintiffs filed *Ber et al. v. Centers for Disease Control and Prevention et al.*, No. 8:26-cv-01822-KKM-LSG (M.D. Fla.), under the Administrative Procedure Act, 5 U.S.C. §§ 555(b) and 706, asking the court to compel CDC and NIH to recognize AHI/Havana Syndrome as a reportable condition for civilians, issue diagnostic guidance for physicians, establish civilian surveillance and reporting, provide civilians equal access to NIH research protocols and treatment programs, and issue Health Alert Network bulletins and training [39]. On 5 October 2026 the defendants moved to dismiss for lack of subject-matter jurisdiction under Rule 12(b)(1), arguing that the complaint identifies no discrete agency action the agencies were required to take and no final agency action [40]. The court has not ruled. I raise the case only as a fact relevant to Q10 and Q17: the agencies HHS asks about have not acted on the petitions or otherwise addressed diagnosed civilians.

Section 5. Standards and Evidence Below the Limits [Q5(p), Q6, Q7, Q8, Q12]

[Q5, Q5(p)] Current limits. The FCC limits, based on NCRP (1986) and ANSI/IEEE C95.1-1992 criteria [10], are set "well below the threshold for unacceptable rises in human tissue temperature" [12, para. 14]; ICNIRP 2020 restricts exposure "to limit temperature rise" [11, p. 487]. In 2021 the D.C. Circuit held the FCC's 2019 decision to retain these limits arbitrary and capricious for lack of a reasoned explanation that they protect against harms unrelated to cancer, a failure it said also undermined the FCC's conclusions on long-term exposure and "exposure to RF pulsation or modulation"; the court took "no position in the scientific debate," and the limits remain in effect [41]. The FCC maximum permissible exposure (MPE) limits in 47 CFR § 1.1310(e)(1), Table 1, cap time-averaged power density [10]. At 1 GHz:

- General population/uncontrolled: $f/1500 = 0.667 \text{ mW/cm}^2$, averaged over 30 minutes.
- Occupational/controlled: $f/300 = 3.33 \text{ mW/cm}^2$, averaged over 6 minutes.

Under § 1.1310(d)(4), "Exposure levels higher than the limits are permitted for shorter exposure times" if the average complies [10,42].

Current limits are anchored to heating. ICNIRP takes a whole-body specific absorption rate (SAR, the rate at which tissue absorbs RF energy) of 4 W/kg averaged over 30 minutes as the exposure that raises body-core temperature by 1 °C, and divides it by 10 for workers (0.4 W/kg) and by 50 for the public (0.08 W/kg) [11, pp. 488, 491–492]. The FCC adopts the same whole-body SAR limits, and its power-density limits above are "derived from whole-body SAR limits" [10, §1.1310(b), (c), (d)(2)], resting on "a whole-body averaged SAR threshold level of 4 watts per kilogram" [42, p. 8]. These limits apply to exposure at the person: the power-density limits apply to fields "spatially averaged over the body dimensions" [42, p. 10], and SAR is the rate at which the body absorbs that energy.

The pulsed parameters postulated for Havana Syndrome and described by the IC Experts Panel and Dagro are defined by peak power and pulse width, not average heating. Because these limits are averages over time, each one also sets an allowed amount of energy per unit area over its averaging window. Dagro et al. applied this to the IEEE C95.1-2019 exposure reference level (ERL; Dagro et al. write "exposure reference limit") at the same 1 GHz frequency, 5 W/m² averaged over 30 minutes, which they calculated equates to 9,000 J/m² [9]. They calculated that a single 5-μs pulse incident on the head carries 75 J/m² in their model, "significantly less than the ERL standard" [9]. As the IC Experts Panel observed, safety reviews have often considered "only average power densities, which can be low in many scenarios relevant to AHIs" [1, FOIA p. 038]. Limits averaged over minutes and keyed to a 1 °C rise do not directly constrain them.

[Q5(p), Q6, Q12] The Panel's pathway parameters. The IC Experts Panel summarized the signal parameters of the five electromagnetic pathways it considered; the table "shows parameters for pulsed signals, although continuous signals could also be used for some pathways" [1, FOIA p. 035].

Pathway	Peak Power (<i>Field Strength</i>)	Primary Frequency	Duration of Single Pulse (<i>Pulse Repetition Frequency</i>)	Biological Effects
Bulk heating	$> 10^3 \text{ mW/cm}^2$ (<i>> few kV/m</i>)	$\sim 200 \text{ MHz to } < 6 \text{ GHz}$	Any duration or repetition rate, including continuous wave signals	Heat and tissue damage. Higher frequencies penetrate and heat less deeply than lower frequencies.
Thermo-acoustic	$10^2 \text{ to } 10^5 \text{ mW/cm}^2$ (<i>50 to 200 kV/m</i>)	$\sim 200 \text{ MHz to } 3 \text{ GHz}$	$\leq 5 \mu\text{s}$ (<i>$\sim 1 \text{ Hz to } 1 \text{ kHz}$</i>)	Pressure wave and possible traumatic brain injury. May hear clicking. Lower frequencies penetrate more deeply. Higher frequencies produce sharper waveforms.
Microwave-auditory	$\geq 10 \text{ mW/cm}^2$ (<i>$\geq 1 \text{ kV/m}$</i>)	$200 \text{ MHz to } \sim 3 \text{ GHz}$	$\sim 1 \text{ ns to } 1 \text{ ms}$ (<i>$\sim 20 \text{ Hz to } 20 \text{ kHz}$</i>)	Generates internal sounds through Frey effect. Can create a sense of vibrational pressure or buffeting.
Strong fields	$\sim 10^2 \text{ mW/cm}^2$ (<i>> few kV/m</i>)	Impulse rise time likely more important than frequency	$\leq 1 \text{ ns}$ (<i>< a few MHz</i>)	Can fracture membranes and capillaries, damaging myelin sheaths around neurons and the blood-brain barrier, causing leakage of cerebrospinal fluid.
Resonant disruption	$\sim 10\text{s of } \mu\text{W/cm}^2$ (<i>$\sim 1 \text{ mV/m to } 1 \text{ V/m}$</i>)	$0 \text{ Hz to } 10 \text{ kHz}$, possibly as much as 10 MHz . Could be modulation of higher frequency carrier.	sub-ns to ms (<i>< 100 Hz?</i>)	Could interfere with neuronal function, induce currents, depolarize membranes, disrupt ion concentrations, or stimulate nerves.

Adapted from 2022 IC Experts Panel, Figure 8 [1, FOIA p. 035]. Values and italics as in the original.

Of the five pathways, the Panel judged bulk heating "an implausible pathway" and the microwave-auditory effect "an implausible pathway on its own" [1, FOIA pp. 036–037]. It rated three plausible: thermo-acoustic effects ("a plausible pathway, with information gaps") [1, FOIA p. 036], strong fields ("a plausible pathway, with uncertainties") [1, FOIA p. 038], and resonant disruption ("a plausible pathway, with significant research gaps") [1, FOIA p. 039]. Overall, it found that "Electromagnetic energy, particularly pulsed signals in the radiofrequency range, plausibly explains the core characteristics, although information gaps exist" [1, FOIA p. 008].

[Q6, Q12] One mechanism on a continuum: the thermoacoustic effect. A short RF pulse absorbed in tissue produces a rapid, minute temperature rise; the tissue expands thermoelastically and launches a pressure wave, which the Panel described as pulses "converted to an acoustic pressure wave that travels through the brain" [1, FOIA p. 036]. A thermoelastic mechanism is not a thermal (heating) effect: the temperature rise only drives the expansion and is far too small and brief to injure by heat [9; 1, FOIA p. 036]. I use three terms in distinct senses. *Acoustic* refers to the pressure wave in tissue. *Auditory* refers to its perception: the wave "travels by bone conduction to the inner ear. There, it activates the cochlear receptors via the same process involved for normal hearing" [43, p. 621]. *Mechanical* refers to the displacement and strain the wave imposes on tissue and on sensory hair cells [44]. What the wave does depends mainly on peak power and pulse width, not on average power. The values below are given in each author's own units.

- *Low peak power: often described as the microwave auditory (Frey) effect, but not limited to sound perception.* At the low end, the pressure wave reaches the inner ear and is heard as a click, buzz, or tone. The effect depends "upon the energy in a single pulse and not on average power density" [45]. For pulses of 10 to 32 μ s, Lin found laboratory "threshold power densities of 2.25 to 40 kW/m² or an average quantity of 13.86 kW/m²" to be "a realistic threshold peak power density" for the effect [46, p. 19], and he concluded that, assuming the accounts are consistent, the effect "provides a scientific explanation" for the reported sounds [47]; he had written in 2021 that, "assuming reported accounts are reliable," the effect "provides a scientific explanation" [48]. By Lin's estimate, a 10- μ s pulse at the hearing threshold raises temperature only "on the order of 10⁻⁶ °C," yet launches a pressure wave [46, p. 24]. Elder and Chou concluded that hearing RF-induced sounds, even "at exposure levels many orders of magnitude greater than the hearing threshold," "is considered to be a biological effect without an accompanying health effect" [45]. This view is now contested. Lin wrote in 2025 that time-averaged SAR "is woefully inadequate to account for the unique characteristics of pulse modulations (with nano to micro-second pulse widths) or to capture the effects of pulse modulated exposures including the microwave auditory effect, which occurs for microsecond pulses without any measurable temperature rise and at low levels of SAR" [49]. ICNIRP, finding no evidence that the effect affects health in realistic exposures, does "not provide a restriction to specifically account for microwave hearing" [11, p. 520]. The Panel called the microwave-auditory pathway "a weak thermo-acoustic effect" [1, FOIA p. 039], insufficient on its own for AHIs because at those powers "the pressure and shear forces are insufficient to disrupt cellular membranes or cause other biological effects" [1, FOIA p. 037]. However, the same pulsed signal could also engage the resonant-disruption pathway (see below), which depends not on peak power but on PRF or modulation frequency matched to the body's own rhythms.
- *Higher peak power, short pulses: pressure waves that can disrupt brain tissue.* The Panel warned that "Brain tissue is fragile and vulnerable to mechanical disruption on scales not

easily observed by medical imaging" [1, FOIA p. 037]. Dagro et al. (2021; U.S. Army Research Laboratory, with Air Force Research Laboratory and academic coauthors) simulated single 1-GHz pulses in an anatomical human head; the study is computational, with no tissue or animal data [9]. Temperature rises below 0.0005 °C within microseconds caused thermoelastic stress waves focusing tensile pressure deep in the brain. With 5-μs pulses:

- Above 1.5×10^6 mW/cm²: tensile pressures comparable to football head impacts (20 to 120 kPa).
- 1×10^7 mW/cm²: tensile pressures exceeding what Dagro et al. call the "commonly cited thresholds for brain tissue cavitation," negative pressures of "roughly -100 to -150 kPa," at which bubbles can form and collapse in tissue, although its "behavior in brain tissue is far from fully characterized" [9].

The authors called these exposures "permissible by current safety standards," judged against time-averaged IEEE limits. They cautioned that these levels are "orders of magnitude larger than most real-world exposure conditions" and that the findings "should not be considered as clinically conclusive evidence" [9]. The Panel likewise notes that high-power short pulses (<5 μs) can produce stress waves while "the temperature rise might be only 0.001 degree Celsius" [1, FOIA p. 073]. Baker et al. (2026) modeled thermoacoustic pressure waves from incident pulses in a layered head model and predicted that continuous pulsing builds up pressure in the head [50]. Yaghmazadeh wrote in 2024 that high-power sub-millisecond RF pulses can induce brain changes "while being compliant with current regulatory guidelines' limits, highlighting the necessity of revising them" [51].

[Q6, Q12] Pulsed RF is not limited to thermoacoustic effects. The Panel rated two further pathways plausible that work by different mechanisms.

- **Strong fields.** Nanosecond, high-field pulses "exert considerable forces on charged particles and cellular membrane potentials" [1, FOIA p. 038], possibly including "piezo-electric-like pressure sensors in the skin and body" [1, FOIA p. 039]. The Panel noted that such fields "have been shown to disrupt ion transport, cell membranes, the blood-brain barrier, and other aspects of living tissues," but that "Much of the work in this area has been performed in vitro or in vivo by using direct contact probes," so "there is little direct evidence of whether nonlocalized cellular effects would cause the core symptoms" [1, FOIA p. 038]. Cell studies illustrate the mechanism: nanosecond pulses induce phosphatidylserine externalization and calcium redistribution (in Jurkat T-lymphoblasts, in vitro) [52], and a single 600-ns pulse of 0.84–1.76 kV/cm opened calcium-permeable nanopores in neural cells [53]. Both used contact electrodes at fields far above anything an RF limit permits; because neither is below FCC limits, I cite each only as mechanism support.
- **Resonant disruption.** This pathway is distinct from the thermoacoustic one: it depends on PRF or modulation frequency rather than on energy delivered per pulse, and it is in

principle tunable. The Panel wrote that "the human body has a variety of naturally occurring rhythmic or repetitive phenomena, ranging from neural activity to cellular functions and mechanical vibrations," and that "these resonances are typically at low frequencies, below 100 Hz" [1, FOIA p. 039]; that human systems are vulnerable to fields that resonate with these patterns "including at low powers" [1, FOIA p. 039]; and that "Some work suggests that resonant coupling into biological frequencies decreases the power required and increases the biological effects," though "such coupling requires further study for biological processes" [1, FOIA p. 040]. A low-frequency modulation on a higher-frequency carrier would propagate like the carrier "while causing biological effects similar to those produced by the lower-frequency component" [1, FOIA p. 040]. The Panel's listed peak power, " ~ 10 s of $\mu\text{W}/\text{cm}^2$ " [1, FOIA p. 035], is far below the FCC general-population MPEs in the range those limits cover (for example, $100 \text{ mW}/\text{cm}^2$ at 0.3–1.34 MHz and $0.2 \text{ mW}/\text{cm}^2$ at 30–300 MHz) [10]. In a controlled human study, RF pulsed at 14 Hz, within the sleep-spindle band, increased spindle-band EEG power, and the authors concluded that "modulation frequency components within a physiological range may be sufficient to induce these effects" [54]; the peak spatial SAR was $2 \text{ W}/\text{kg}$, at the ICNIRP head limit and above the FCC $1.6 \text{ W}/\text{kg}$ localized limit if read as a 1-g average; because it is not shown to be below FCC limits, I cite it only as mechanism support, not as evidence of harm. Resonant disruption is contested, especially among regulators: ICNIRP 2020 states that "there is no evidence that continuous (e.g., sinusoidal) and discontinuous (e.g., pulsed) EMFs result in different biological effects" [11, p. 487], while acknowledging that EEG alpha and sleep-spindle bands "are affected by radiofrequency EMF exposure" at SARs below $2 \text{ W}/\text{kg}$, "but there is no evidence that these relate to adverse health effects" [11, p. 518]. The Panel nonetheless rated the pathway plausible [1, FOIA p. 039], which calls for research, not dismissal. In my view, resonance may operate at two scales: the Panel's resonant-disruption pathway addresses the body's naturally occurring rhythms, which it lists as including "mechanical vibrations" and places "typically at low frequencies, below 100 Hz" [1, FOIA p. 039], while pulsed exposures also excite an acoustic resonance of the head itself, predicted at about 8 kHz for the human head [46, p. 25]; whether either can be driven to injurious amplitude has not been studied.

The plausible pulsed-RF pathways are thus thermoacoustic (from the auditory end to the tissue-disrupting end), strong-field, and resonant. Each depends on peak or pulse parameters that time-averaged limits do not capture.

[Q6, Q12] Experimental evidence. Laboratory studies have reported biological effects from pulsed exposures whose time-averaged dose meets current limits (whole-body average SAR of $0.08 \text{ W}/\text{kg}$ for the general public; $0.4 \text{ W}/\text{kg}$ occupational):

- **Raslear et al. (Walter Reed Army Institute of Research, 1993).** Rats exposed to 3-GHz, 80-nanosecond pulses at peak powers in excess of 700 megawatts received "a

maximal, whole-body-averaged, specific-absorption rate of 0.072 W/kg." The authors found dose-response impairment in discriminating time intervals, indicating "that the microwave exposures were affecting cognitive function in the rats, particularly the decision-making process" [55]. The comparison of that SAR with the 0.08 W/kg public limit is mine; compliance with power-density limits was not assessed.

- **Lu et al. (1999).** Rats exposed for 6 minutes to ultra-wideband pulses at 0.5 kHz PRF (1.00-ns pulses, 93 kV/m peak, whole-body SAR 70 mW/kg) developed a drop in arterial blood pressure lasting from 45 minutes to 4 weeks, "a robust, consistent, and persistent effect" [56]. A second group at 1-kHz PRF (SAR 121 mW/kg) exceeded the 0.08 W/kg public limit; I rely on the 0.5-kHz group. The comparison of these SARs with that limit is mine; compliance with power-density limits was not assessed. These pulses had no microwave carrier, and the effect was cardiovascular, not a brain or vestibular injury.
- **Gao et al. (2020).** Rats given a single exposure to 20,000 ultra-wideband pulses (0.9 ns, 10 Hz, peak field 400 kV/m) at a reported average SAR of 7.01×10^{-3} W/kg showed transient blood-brain barrier opening and a rise in serum S100 β [57] (Section 6(c)). The comparison of that SAR with the 0.08 W/kg public limit is mine; compliance with power-density limits was not assessed.

[Q5, Q5(o), Q5(p)] Other national and international standards. Newer frameworks cover only part of the gap and do not protect against the exposures the Panel postulated:

- **ICNIRP 2020.** For brief exposures at 400 MHz–6 GHz, local specific absorption (SA; Table 3) and incident energy density (Table 7) limits apply to "any pulse, group of pulses, or subgroup of pulses in a train"; peak field limits (Table 8) cover only 100 kHz–10 MHz [11]. The per-pulse limit is itself thermal.
- **IEEE C95.1.** IEEE C95.1-1999 and C95.1-2005 capped the peak electric field for pulsed exposure at 100 kV/m. C95.1-2019 defines peak limits and pulse-fluence limits for pulsed RF exposure [58,59], but I could not verify whether the 100 kV/m cap was retained. The FCC has not adopted these peak limits [10]; OET 65 notes that "the FCC did not explicitly adopt limits for peak power density" [42, p. 10].

No framework I could verify limits pulse width, PRF, modulation, or cumulative pulse count on the basis of thermoelastic pressure or resonant coupling.

[Q7, Q8] Measurement. Under current compliance practice, exposure is judged against time-averaged limits. For pulsed sources, OET 65 asks surveyors to determine "the pulse width and repetition rate" [42, p. 49], but no limit applies to either [10], and it cautions that "many commercially-available survey instruments cannot measure high peak-power pulsed fields accurately" [42, p. 47]. An exposure can therefore be judged compliant without its pulse structure being evaluated against any limit. Because it is not yet known which features of pulse structure are responsible for the postulated injury, assessment should capture them rather than average them away: sub-microsecond to microsecond-resolved instruments that report peak power density, pulse width, PRF, duty cycle, per-pulse energy, and modulation along with

averages.

Section 6. From AHI1 Findings to Exposure Pathways [Q6, Q12, Q16]

[Q6, Q12, Q16] This section maps each AHI1 phenotype feature (Section 2, same letters) to the pathways the IC Experts Panel considered (Section 5). Each link is labeled established or inference. Plausibility is graded as moderate (the mechanism has been shown in models or animals and is consistent with the finding, but has not been tested in people) or low (a candidate route with limited or indirect support). None establishes a cause, and none depends on any particular source.

Spatial distribution of mechanical stress. Dagro et al. found that short pulses produce stress waves that "converge near the deep regions of the brain," with large negative pressures in "the periventricular, diencephalon, and midbrain regions (encompassing the inferior colliculus and other areas important to eye movement and hearing response)" [9]. Baker et al. predicted the greatest effects for short pulses at the center of the intracranial region, and pressure build-up under continuous pulsing close to the location of the incident stimulus [50]. These locations are consistent with the deep and midline white-matter findings in the imaging cohorts, including higher fractional anisotropy in the splenium of the corpus callosum at an uncorrected threshold [18] and reduced fibre density in the fornix and splenium [22], and with the Panel's reasoning that one-sided ear symptoms point to the inner ear and brainstem pathways [1, FOIA p. 017]. Neither model includes the inner ear or otolith organs, the site of the defining vestibular findings [20]; otolith mechanics have been studied outside these RF models (see (d)). While both simulations are at very high peak power, Dagro et al. calculated that such a pulse can still fall far below the time-averaged energy limit (Section 5). Both speak to the mechanism, not to evidence of injury in people. *Pathway: thermoacoustic. Status: inference. Plausibility: moderate, because two models place peak stress in deep or central regions, in close anatomical proximity to the deep and midline imaging findings, although neither model resolves the structures involved; limited by high-peak-power simulations and the models' omission of the inner ear.*

(a) Symptoms occurring simultaneously and the audible marker.

- **Simultaneous onset.** The core characteristics are defined by audio-vestibular phenomena and "other nearly simultaneous signs and symptoms" beginning together (Section 2(a)) [1, FOIA p. 007]. The Panel found that "A disturbance of the auditory or vestibular components of the ear could explain the core characteristics and could be triggered by some of the potential causal mechanisms examined" [1, FOIA p. 017]. In my view, their occurrence together is better explained by one exposure acting through one mechanism, a pulsed pressure wave reaching the inner ear and brain at the same moment, than by separate causes. *Pathway: thermoacoustic. Status: inference. Plausibility: moderate, because the Panel postulated that an ear disturbance from the examined mechanisms could explain the core characteristics; a single exposure producing them together has not been tested.*

- **The audible marker.** The audible marker occurs in some cases but not all; the Panel noted that the microwave-auditory effect "could appear as a side effect of any of the other four pathways, resulting in the perception of sounds in some individuals" [1, FOIA p. 037]. The auditory mechanism is established: the pressure wave "travels by bone conduction to the inner ear" [43, p. 621]. Because in AHI1 cases the perceived sound, where present, was accompanied by other symptoms, and because the auditory and higher-power effects arise from the same thermoelastic mechanism, in my view this is more consistent with higher- than with low-power thermoacoustic exposure. *Pathway: thermoacoustic, auditory end. Status: the mechanism is established; its use as a marker of higher-power exposure is inference. Plausibility: moderate, because the auditory mechanism is established in people; its use as a marker of higher-power exposure has not been tested.*

(b) Buffeting and vibrational pressure. The onset sensation of pressure or vibration reported in the original Havana cases (Section 2) [6,17] is the effect the Panel attributed to the microwave-auditory pathway, which "Can create a sense of vibrational pressure or buffeting" [1, FOIA p. 035]. Frey reported: "With somewhat different transmitter parameters, we can induce the perception of severe buffeting of the head" [60]; which parameters produce it is not established. As a further candidate, in my inference, resonant disruption may contribute: the Panel placed the body's natural rhythms "typically at low frequencies, below 100 Hz" [1, FOIA p. 039]. *Pathways: thermoacoustic (auditory end), per the Panel's table; resonant disruption as a candidate. Status: the Panel's attribution is a judgment, not a measurement; the resonant link is my inference. Plausibility: moderate for the thermoacoustic attribution, which rests on the Panel's judgment and Frey's qualitative report; low for resonant disruption, owing to the lack of data on RF-driven tissue resonance.* No clinical study has measured the pulse parameters accompanying buffeting.

(c) Early blood biomarkers (GFAP, NfL, UCHL1). The Panel reported "early, transient elevations in biomarkers suggestive of cellular injury to the nervous system" in the few individuals tested in time [1, FOIA p. 008]. Their diagnostic value, it explained, pertains to exposures "sufficient to cause leakage of these proteins from brain cells or injury to the blood-brain barrier"; if "only the ear or peripheral nerves were stimulated, current scientific knowledge would not predict that the biomarkers would be elevated" [1, FOIA p. 016]. Two pathways offer a physical route to such leakage: strong fields, which "Can fracture membranes and capillaries" and damage "the blood-brain barrier" [1, FOIA p. 035], and which in vitro alter or porate cell membranes [52,53]; and thermoacoustic stress in the injurious range. In my reading, the auditory end and resonant disruption do not. One animal study gives the strong-fields route mechanism support: in rats, a single exposure to 20,000 ultra-wideband pulses (0.9 ns, 10 Hz, peak field 400 kV/m; average SAR reported as 7.01×10^{-3} W/kg) transiently opened the blood-brain barrier and raised serum S100 β at six hours [57]. That SAR is below the 0.08 W/kg public whole-body limit [10]. Microsecond pulses at 2.45 GHz (10 μ s, 100 pps, 10 mW/cm² average, SAR about 2 W/kg) increased tracer uptake by cortical capillary endothelium [61]; this exceeds the public limits, so I

cite it only as mechanism support for barrier opening. Earlier low-level pulsed barrier findings were not reproduced [62], and ICNIRP, reviewing reports of albumin leakage in rats, finds that "there remains no evidence of an effect" [11, p. 519]. Human data are thin. No published AHI study I found reports UCHL1, which the Panel noted "was not included in assays performed to date"; the Panel also wrote that "There is no published work on the plausible causal mechanisms... and the type of brain injury that would result from such exposure" [1, FOIA p. 114]. *Pathways: strong fields; injurious-range thermoacoustic as a candidate. Status: mechanism support in animals, including one study within the whole-body average limit; I found no RF study that has measured GFAP, NfL, or UCHL1 in blood. Plausibility: moderate, because one pulsed animal study within the whole-body average limit reports blood-brain barrier opening and a blood biomarker rise (S100 β) [57]; limited by the absence of RF studies measuring GFAP, NfL, or UCHL1 and by earlier low-level barrier findings that did not replicate.*

(d) Otolithic injury. The defining AHI1 finding is otolithic: "100% of individuals had an otolithic abnormality" in the acute Miami series [20]. Pulsed microwaves do drive inner-ear hair cells mechanically: in guinea pigs exposed to 918-MHz pulsed microwaves, Chou et al. recorded "a mechanical disturbance of the hair cells of the cochlea" [63]; the exposure levels were not verified as within FCC limits, and the study is cited only as mechanism support. The utricle and saccule share the cochlea's fluid-filled labyrinth and bone, and utricular afferents are "exquisitely sensitive to auditory-frequency air conducted sound (ACS) and bone conducted vibration (BCV)" [44]. *Pathway: thermoacoustic. Status: cochlear hair-cell motion is established in animals; extending it to the otolith organs, and to injury, is inference. Plausibility: moderate, because pulsed microwaves move cochlear hair cells in animals and the otolith organs respond to bone-conducted vibration [44]; the grade rests on that documented otolith response, since no RF model or study includes the otolith organs.* No RF model includes the labyrinth (Section 7).

(e) Oculomotor findings. As set out under Spatial distribution of mechanical stress above, the models concentrate stress in deep and central regions [9,50]; the clinical findings are set out in Section 2(e). One link is named by the source: Dagro et al. describe the midbrain region where they predict large negative pressures as containing "other areas important to eye movement" [9, p. 11]. A second is adjacent, by my anatomical reading: in Verma et al.'s 40 patients, worse near point of convergence correlated with greater volume of the ventral diencephalon [18, pp. 344–345], overlapping Dagro et al.'s diencephalic stress focus [9, p. 11]. Dagro et al. tie this deep focusing to pulse duration: a 5- μ s pulse, shorter than the intrinsic time of "approximately $\tau_c = 59$ μ s," made stress waves "converge near the deep regions of the brain to create high tensile stresses" [9, pp. 6–7]; the foci come from a single 1-GHz, 5- μ s pulse at 1×10^6 mW/cm² [9, pp. 7, 11]. The oculomotor findings thus map onto the midbrain and diencephalic regions where short-pulse stress is modeled to concentrate. *Pathway: thermoacoustic. Status: the midbrain link to eye movement is named by Dagro et al.; the ventral-diencephalon link is adjacent, by my anatomical reading; no RF study has measured eye movements or pupil responses. Plausibility: low, because the only source-named link is a modeled stress focus for a postulated single 5- μ s*

pulse at 1×10^6 mW/cm², the imaging correlation is exploratory, and the model does not resolve oculomotor structures [9, p. 15].

(f) Brain imaging and network-level disruptions. As set out under Spatial distribution of mechanical stress above, Dagro et al. place large negative pressures in "midbrain regions (encompassing the inferior colliculus...)" and in periventricular and diencephalic regions [9, pp. 7, 11]. In Verma et al.'s cohort, diffusivity was lower in the inferior colliculi, identical by name with Dagro et al.'s, and fractional anisotropy higher in the splenium of the corpus callosum, both uncorrected [18, pp. 342–343]. In Canadian diplomats and family members with symptoms similar to those of U.S. personnel, fixel-based fibre density was lower in the fornix and splenium [22, pp. 1, 2, 5]. In AHI1, return-to-axis probability in the splenium was about 2% lower, not significant (my analysis) [24, p. 9]. By my anatomical reading, the splenium and fornix are adjacent to the modeled foci: both border the lateral ventricles, around which Dagro et al. place "periventricular" stress [9, pp. 7, 14]. Uncorrected cingulum and inferior cerebellar peduncle changes and salience-network hypoconnectivity (adjusted $p = 0.020$ and 0.030) (my analysis) [24, pp. 8–10] match no model region. Modeling and imaging in separate cohorts thus converge, identically at the inferior colliculus and adjacently at the splenium and fornix, on the deep midline structures where short-pulse thermoacoustic stress is modeled to concentrate. *Pathways: thermoacoustic, with strong fields ("damaging myelin sheaths" [1, FOIA p. 035]) as a white-matter candidate. Status: identical by name at the inferior colliculus; adjacent, by my anatomical reading, at the splenium and fornix. Plausibility: moderate for white matter, given this convergence; limited by uncorrected or non-significant findings, directions of change that differ across studies, structures no model resolves [9, p. 15], and very high peak power; low for network-level findings, since salience hypoconnectivity maps onto no model region.*

(g) Cognitive deficits. The clinical findings are set out in Section 2(g); here I trace the pathway. In rats, 80-ns pulses at 3,000 MHz, with peak power in excess of 700 MW and "a maximal, whole-body-averaged, specific-absorption rate of 0.072 W/kg," were "affecting cognitive function in the rats, particularly the decision-making process" [55, pp. 459, 465]. That SAR is below the 0.08 W/kg public limit; the comparison is mine, and power-density compliance was not assessed (Section 5). The authors speculated that the "thermoelastic pressure wave in brain tissue" might produce effects that "resemble those following a traumatic head injury," but measured no brain pressure [55, p. 476]. Dagro et al. cite this unreplicated study [9, p. 2] and frame their model as "a testable hypothesis between potential neurocognitive effects and the thermoelastic mechanism" [9, p. 13]; the Panel likewise lists "Pressure wave and possible traumatic brain injury" for thermoacoustic exposure [1, FOIA p. 035]. In Canadian diplomats and family members, fibre density was lower along the fornix, a tract with "an important role in memory and cognition" [22, pp. 6, 10]. The fornix and the splenium of the corpus callosum border the lateral ventricles, adjacent to Dagro et al.'s periventricular foci [9, p. 11] (see (f)). In AHI1, salience hubs "which filter salient sensory stimuli and mediate transitions between cognitive states" were hypoconnected (my analysis) [24, p. 9]. Animal behavior, modeling, and imaging thus map onto one postulated route: short high-peak pulses, thermoelastic stress around

the ventricles, and injury to the adjacent memory and attention pathways. *Pathway: thermoacoustic; strong fields, through the blood-brain barrier (c), as a candidate. Status: one pulsed rat study within the whole-body average SAR limit reports a cognitive effect [55]; the fornix and splenium adjacency is my anatomical reading; the salience link is my inference. Plausibility: moderate; limited by one unreplicated study, very high peak power, a fornix change that tracked headaches and tinnitus rather than memory [22, p. 10], and no executive-control network difference in Verma et al.'s cohort [18, p. 345].*

Taken together, items (a)–(g) show that each core feature of the AHI1 phenotype can be matched to a postulated pathway of pulsed RF exposure, and that for some of them the reported effects occur at exposures within current public limits [10]. This is the reasoning of differential diagnosis: asking which single cause could produce these signs together. The IC Experts Panel reached a similar conclusion, finding that electromagnetic energy, "particularly pulsed signals in the radiofrequency range, plausibly explains the core characteristics" [1, FOIA p. 008].

Section 7. Research Gaps: Repeated Sub-Threshold Pulses and the Inner Ear [Q5(o), Q16, Q17(b), Q17(c)]

I ask NIH and CDC to address the research gaps identified in Sections 5 and 6:

1. **Repeated sub-threshold pulses (Section 5).** I identified no peer-reviewed microwave study designed to test whether trains of pulses, each below a single-pulse injury threshold, cause cumulative mechanical injury in brain or vestibular/otolith tissue. Published evidence on nanosecond and microsecond pulses within current time-averaged limits is thin, and few studies report the peak field, pulse energy, and SAR needed to compare them with those limits. Analogous rodent studies of repeated sub-concussive impact show cumulative brain effects; none examined vestibular tissue [64–66]. Dagro et al. wrote that cyclic loading "could potentially induce different injury thresholds that are unknown at this time" [9]. Which features of pulse structure are responsible for the postulated injury is not known (Section 5, Measurement). The Panel wrote: "Little research in the West has systematically explored configurations of electromagnetic energy that could cause nonthermal clinical effects" [1, FOIA p. 031].
2. **Gaps in the Section 6 connections.**
 - (Opening of Section 6) Neither head model includes the inner ear or otolith organs [9,50].
 - (a) Whether a single pulsed exposure can produce simultaneous auditory and vestibular effects, and whether the sound, where present, marks higher-power exposure, is untested.
 - (b) Which pulse parameters produce buffeting is not established [60], and no clinical study has measured them; the resonant link is my inference, resting on coupling that the Panel said "requires further study for biological processes" [1, FOIA p. 040].
 - (c) I found no RF study that has measured GFAP, NfL, or UCHL1 in blood, and

no published AHI study I found reports UCHL1 [1, FOIA p. 114].

- (d) No RF model includes the labyrinth, so extending cochlear hair-cell motion [63] to the otolith organs, and to injury, remains inference.
- (e) No RF study, in animals or people, has measured eye movements or pupil responses after pulsed exposure; the link between the modeled midbrain stress foci [9] and oculomotor deficits is untested, and whether the vergence and pupil pattern reported in acute Havana cases [21] replicates is unknown.
- (f) The imaging studies converge on the splenium of the corpus callosum [18,22,24], and the closest model match is the midbrain region encompassing the inferior colliculus [9,18]; neither head model resolves the corpus callosum or individual brainstem nuclei [9,50], so whether the modeled deep and central stress foci correspond to these structures is untested. Because the direction of change at the splenium differs across the imaging studies and the models predict where stress concentrates, not how tissue responds [9], a test would need a head model that resolves these structures under multiple-pulse loading, which both groups left for later work [9,50], together with tissue-response data. No study has tested a link between the modeled foci and salience-network hypoconnectivity in AHI1 [24].
- (g) Evidence that pulsed exposure within the whole-body average limit affects cognition rests on a single rat study [55], of which I found no replication, and no study has tested which pathway produces it.

For NIH.

3. Priority work: dosimetry that reports peak power density, pulse width, PRF, and SA per pulse, at the person and at the tissue (item 1; 6(b); see also Recommended HHS Action 6); head models that add the petrous bone, cochlea, and otolith organs to report pressure and hair-bundle strain at the maculae by pulse width and angle of incidence (opening of Section 6; 6(d)); and in-vitro and animal studies of single and repeated sub-threshold pulses in brain tissue, axons, and vestibular end organs, varying pulse count, PRF, and peak power, with vestibular physiology, otolith histology, and blood GFAP, NfL, and UCHL1 (item 1; 6(a), 6(c), 6(d)). This work must begin in vitro and in animals: deliberate pulsed exposure of human subjects would risk injury and would not be approved by an institutional review board.
4. An exploratory observational clinical study of civilians diagnosed with the Havana Syndrome AHI1 phenotype, using the same vestibular/otolith testing, oculomotor and cognitive testing, advanced neuroimaging, MEG, and timely biomarker battery (6(c)–(g)). This would study people already diagnosed, not deliberate exposure.

For CDC.

5. Standardized vestibular/otolith testing, including cervical and ocular vestibular evoked myogenic potentials (cVEMP, oVEMP) [67], audiometry, oculomotor testing that includes quantitative video-oculography of binocular disparity vergence and pupillary

light responses, to test whether the pattern reported in acute Havana cases [21] replicates, and brain injury biomarkers in cohorts reporting symptoms after suspected pulsed exposure, each using one stated case definition, with no pooling across definitions.

Section 8. Surveillance, AHI1, and Civilians [Q4(l), Q4(m), Q10, Q11(g), Q17(c)]

[Q11(g)] Civilians who report symptoms similar to those described in AHI cases may be one of the "other populations" that Q11(g) describes. If pulsed RF is involved in any of these cases, the relevant exposure would differ from ordinary everyday and occupational exposure in the pulse parameters described in Section 5. If members of the public have had such an exposure, it has not been measured, and its parameters are unknown.

[Q10] Existing data sources serve only U.S. government personnel who report AHIs and, in some programs, their families, and exclude civilians:

- The Defense Health Agency's April 2025 AHI clinical recommendation for Military Health System primary care managers provides a standard evaluation, referral to the National Intrepid Center of Excellence (NICoE), and a voluntary AHI Patient Registry [30].
- From 2018 to 2021, the NIH Clinical Center enrolled U.S. government employees and their adult family members in an intramural AHI study; enrollment required referral from a U.S. government entity [2,3].
- Federal law requires DoD to provide treatment in military treatment facilities to current and former U.S. government employees and their family members diagnosed with AHI-related conditions; the NICoE at Walter Reed coordinates this care [13,30].
- The HAVANA Act of 2021 authorizes the CIA and the State Department (or another agency head) to pay covered employees, individuals, and dependents for qualifying brain injuries, subject to appropriations [16].
- Federal employees may file workers' compensation claims for AHIs under FECA [15].

The exploratory NIH studies, which tested participants a median of 76 days (clinical and biomarker testing [2]) and 80 days (imaging [3]) after their most recent incident, found no significant differences from matched controls on most clinical, biomarker, and imaging measures, apart from imbalance and self-reported symptoms [2,3]. My peer-reviewed re-analysis of the 2024 NIH neuroimaging study [24] separated the AHI1 cases (the core-characteristics subset with otolithic findings; Section 2) from the pooled AHI group and compared the 43 AHI1 participants with the 48 matched controls. Resting-state functional MRI showed significantly reduced connectivity in the salience network, centered on the insula and anterior cingulate (adjusted $p \approx 0.02\text{--}0.03$). Diffusion measures did not differ after correction, but nominal changes (uncorrected $p < 0.05$) appeared in midline white-matter tracts: about 2–3% lower return-to-axis probability in the body and genu of the corpus callosum, and slightly reduced diffusivity in the right cingulum and inferior cerebellar peduncle. My re-analysis reports that these changes parallel the findings of Verma et al. (2019) [24; 18], though the matched structures and measures

differ. Pooling all participants under one label can dilute these signals; analyzed separately, the AHI1 phenotype is consistent with a diffuse brain injury syndrome that more sensitive methods, such as advanced neuroimaging, MEG (reported in a preprint [23]), and quantitative EEG, may be needed to detect.

The NIH study I re-analyzed was halted in August 2024 after an NIH internal review found that informed-consent requirements "were not met due to coercion, although not on the part of NIH researchers"; NIH stated that the findings "do not impact the conclusions of the study" [68].

On 11 June 2026, the Office of the Director of National Intelligence notified Congress that the Director of National Intelligence had directed the National Intelligence Council to rescind two Intelligence Community Assessments on AHI, ICA 2023-02286 and ICA 2025-00008, for "failing to meet the established Intelligence Community (IC) Analytic Standards," including "Reliance on an ethically flawed medical study without noting methodological critiques" [33]. Although the notice does not name the study, the 2025 assessment based its medical judgment on "research published in 2024 from the National Institutes of Health (NIH)" [69]. The rescission did not cover the 2022 IC Experts Panel assessment on which this comment relies [1]. I believe the rescission in effect undercut the NIH studies' conclusion that there was no MRI-detectable evidence of brain injury, a conclusion on which those assessments relied. The NIH Director responded publicly that the assessments "mischaracterized the NIH study," that "[o]ur research was not complete at the time of this report" (true of the 2023 assessment, but not of the 2025 assessment, which relied on the published 2024 studies [69]), and that the study was not "designed to determine whether a foreign adversary or external mechanism caused these health effects" [70]. That response did not address the published methodological criticism of the NIH studies. In a JAMA editorial accompanying them, Relman wrote that the investigators "aggregated cases, potentially diluting important 'signals' from what might be unrecognized discrete clusters of individuals"; that, for the blood biomarkers (plasma GFAP and NfL), "only 2 participants with AHIs had baseline (pre-AHI) measurements and only 16 had post-AHI measurements within 3 days—and hence, their study lacked relevant statistical power" (the 16 were participants with AHIs, 12 drawn within 24 hours and 4 within 72 hours; their AHI 1 and AHI 2 breakdown was not reported [2]); and that "current MRI technology may be insensitive to, or improperly timed for, the kinds of potentially transient cellular and local physiological disruption that characterizes AHIs" [71]. Civilians who report similar symptoms have no comparable data source; one should be developed (Recommended HHS Actions 2 and 3).

[Q4(l), Q4(m)] Outside the Military Health System, there is no standard evaluation protocol for these patients. In my experience, clinicians assess them differently, and the results are difficult to compare. Even the Defense Health Agency's April 2025 clinical recommendation [30] does not include otolithic tests, such as cVEMP and oVEMP, or brain injury blood biomarkers, such as GFAP, NfL, and UCHL1. It states that AHI "currently lack[s] evidence-based, objective diagnostic criteria" and that "the medical community relies on expert opinion from providers who care for most reported AHI cases" [30, p. 3]. The guidance essentially defers the diagnosis

to the experts, and the resulting "AHI" designation is an administrative label, not a diagnosis. This model is not appropriate for public medicine, where civilian clinicians need defined tests and criteria they can apply themselves.

[Q17(c)] The most detailed technical analysis, the IC Experts Panel assessment, was released only in part, through FOIA [1]. HHS, CDC, and NIH have issued no civilian case definition, reporting pathway, or clinician guidance that I am aware of.

Recommended HHS Actions

1. **[Q4(l), Q10(a), Q11(g), Q17(b), Q17(c)]** Formally recognize the Havana Syndrome AHI1 clinical phenotype (core characteristics plus objective otolithic, oculomotor, cognitive, and imaging findings, with early blood biomarkers where timely testing is available) as diagnosed by expert physicians, analyzed separately from AHI2 (Section 2). Treat AHI1 as a placeholder until a precise phenotype-based clinical name that does not presuppose a cause is adopted, unless the cause is resolved; the Department of War's July 2026 renaming to Directed Energy Bio-Effects names a suspected mechanism, not the clinical condition (see Section 2) [32].
2. **[Q10(a), Q10(f), Q17(c)]** Establish or designate one civilian case definition, applied consistently and not pooled with other definitions, and a reporting pathway and registry so that physicians can report diagnosed civilian cases without requiring federal employment.
3. **[Q4(l), Q4(m), Q17(b), Q17(c)]** Issue clinician guidance for evaluating these patients, including standardized vestibular/otolith (cVEMP/oVEMP) [67], audiological, oculomotor (clinical screening with referral for formal neuro-optometric evaluation [17], and quantitative video-oculography including binocular disparity vergence and pupillary light reflex tests [21]), and early brain-injury biomarker testing, and open NIH research protocols to civilians referred through clinical pathways that do not depend on federal employment.
4. **[Q17]** Have CDC and NIH act on the September 2025 petitions [14,38] for civilian recognition, surveillance, and guidance, and designate HHS and CDC to lead the civilian public-health response in the open, alongside existing DoD work, not in place of it.
5. **[Q5(n), Q5(p), Q18]** Update exposure standards for pulsed exposures:
 - (a) Ensure RF exposure standards and assessment methods capture pulse parameters (peak power density, pulse width, PRF, energy per pulse, modulation) so that sub-thermal pulsed effects, including thermoacoustic, resonant, and strong-field pathways, and cumulative effects can be studied and, if warranted, regulated (Sections 5 and 7).
 - (b) Require any HHS or FDA exposure assessment or classification system to report peak and pulse metrics (peak power density, pulse width, PRF, energy or specific absorption per pulse, and modulation) alongside time-averaged values, and advise the FCC on whether its limits need pulse-specific terms such as the

peak limits in IEEE C95.1-2019 [58,59].

- (c) Keep this work distinct from general RF-exposure concerns. The exposure limits are time-averaged (over 6 minutes for occupational and 30 minutes for general-population exposure), with no per-pulse limit [10]. This comment makes no claim about everyday RF exposure; its purpose is to ensure that a pulsed signal whose peak parameters could be harmful cannot be judged acceptable simply because its average is low.
6. **[Q7, Q8]** Have FDA develop and validate sub-microsecond to microsecond-resolved measurement protocols that record pulse structure, not only averages (Section 5).
 7. **[Q16, Q17(b)]** Fund an NIH research program on single and repeated sub-threshold pulsed exposure in brain, axonal, and vestibular/otolith tissue, with pulse-resolved dosimetry and head models that include the inner ear, and an exploratory NIH clinical study of civilians diagnosed with the AHI1 phenotype (Section 7).
 8. **[Q17, Q18]** Establish interagency data sharing with DoD/DHA and ODNI, including declassification review of health-relevant technical findings.
 9. **[Q17(c)]** Appoint an HHS Havana Syndrome (AHI) coordinator.

Respectfully submitted,

Len Ber, MD

Medical Director, Civilian Registry for Diagnosed Havana Syndrome Patients

References

1. Office of the Director of National Intelligence, IC Experts Panel on AHI. Anomalous Health Incidents: Analysis of Potential Causal Mechanisms (full assessment, September 2022). Released in part under FOIA in *James Madison Project v. ODNI*, No. 22-cv-00674, ODNI FOIA Case DF-2022-00138, March 28, 2023. 147 pp. Page pins ("FOIA p.") give the last three digits of the release's six-digit Bates stamps (000001–000147).
2. Chan L, Hallett M, Zalewski CK, et al. Clinical, biomarker, and research tests among US government personnel and their family members involved in anomalous health incidents. *JAMA*. 2024;331(13):1109–1121. [doi:10.1001/jama.2024.2413](https://doi.org/10.1001/jama.2024.2413).
3. Pierpaoli C, Nayak A, Hafiz R, et al. Neuroimaging findings in US government personnel and their family members involved in anomalous health incidents. *JAMA*. 2024;331(13):1122–1134. [doi:10.1001/jama.2024.2424](https://doi.org/10.1001/jama.2024.2424).
4. Ber L. Collecting Information on Diagnosed Cases of "Havana Syndrome" and its Anomalous Health Incidents (AHI) among Civilians Occurring on US Soil: Interim Report. Civilian Registry for Diagnosed Havana Syndrome Patients (CRHS). January 2025. [doi:10.13140/RG.2.2.30841.38243](https://doi.org/10.13140/RG.2.2.30841.38243).
<https://www.researchgate.net/publication/387759199>
5. Ber L. Civilian Registry for Diagnosed Havana Syndrome Patients and Anomalous Health Incidents (AHI) among Civilians Occurring on US Soil: January 2026 Update. January 2026. [doi:10.13140/RG.2.2.19090.93122](https://doi.org/10.13140/RG.2.2.19090.93122).

- <https://www.researchgate.net/publication/398931431>
6. National Academies of Sciences, Engineering, and Medicine. An Assessment of Illness in U.S. Government Employees and Their Families at Overseas Embassies. Relman DA, Pavlin JA, eds. Washington, DC: The National Academies Press; 2020.
[doi:10.17226/25889](https://doi.org/10.17226/25889).
 7. Intelligence Community Experts Panel on Anomalous Health Incidents. Executive Summary. Office of the Director of National Intelligence; declassified February 1, 2022.
https://www.dni.gov/files/ODNI/documents/assessments/2022_02_01_AHI_Executive_Summary_FINAL_Redacted.pdf
 8. Giordano J, DiEuliis D, Fitzgerald E, et al. Assessing anomalous health incidents of "Havana Syndrome": potential utility - and issues - of using modular integrated artificial intelligence. *EC Neurology*. 2025;17(4):01–08. Published March 13, 2025.
<https://ecronicon.net/ecne/assessing-anomalous-health-incidents-of-havana-syndrome>
 9. Dagro AM, Wilkerson JW, Thomas TP, et al. Computational modeling investigation of pulsed high peak power microwaves and the potential for traumatic brain injury. *Sci Adv*. 2021;7(44):eabd8405. [doi:10.1126/sciadv.abd8405](https://doi.org/10.1126/sciadv.abd8405).
 10. Radiofrequency radiation exposure limits. 47 CFR § 1.1310 (as amended at 85 FR 18145, Apr. 1, 2020). Electronic Code of Federal Regulations.
<https://www.ecfr.gov/current/title-47/chapter-I/subchapter-A/part-1/subpart-I/section-1.1310>
 11. International Commission on Non-Ionizing Radiation Protection (ICNIRP). Guidelines for limiting exposure to electromagnetic fields (100 kHz to 300 GHz). *Health Phys*. 2020;118(5):483–524. [doi:10.1097/HP.0000000000001210](https://doi.org/10.1097/HP.0000000000001210).
 12. Federal Communications Commission. Resolution of Notice of Inquiry, Second Report and Order, et al. FCC 19-126, ET Docket No. 19-226. Released December 4, 2019. <https://docs.fcc.gov/public/attachments/FCC-19-126A1.pdf>
 13. U.S. Government Accountability Office. Havana Syndrome: Better Patient Communication and Monitoring of Key DOD Tasks Needed to Better Ensure Timely Treatment. GAO-24-106593. July 29, 2024. <https://www.gao.gov/products/gao-24-106593>
 14. Ber L. Petition for Recognition, Surveillance, and Guidance Regarding Civilian "Havana Syndrome" (Anomalous Health Incidents) Cases [petition to CDC], restating the registry's January 2025 report. September 29, 2025.
<https://lenbermd.substack.com/p/formal-petition-to-the-cdc>
 15. U.S. Department of Labor, Office of Workers' Compensation Programs. FECA Bulletin No. 22-03, Processing Claims for Anomalous Health Incidents (AHI) under the Federal Employees Compensation Act (FECA).
<https://www.dol.gov/agencies/owcp/FECA/regs/compliance/DFECfolio/FECABulletins>
 16. Helping American Victims Afflicted by Neurological Attacks Act of 2021 (HAVANA Act of 2021). Pub. L. No. 117-46, 135 Stat. 391 (Oct. 8, 2021).
<https://www.govinfo.gov/content/pkg/PLAW-117publ46/html/PLAW-117publ46.htm>

17. Swanson RL, Hampton S, Green-McKenzie J, et al. Neurological manifestations among US government personnel reporting directional audible and sensory phenomena in Havana, Cuba. *JAMA*. 2018;319(11):1125–1133. [doi:10.1001/jama.2018.1742](https://doi.org/10.1001/jama.2018.1742)
18. Verma R, Swanson RL, Parker D, et al. Neuroimaging findings in US government personnel with possible exposure to directional phenomena in Havana, Cuba. *JAMA*. 2019;322(4):336–347. [doi:10.1001/jama.2019.9269](https://doi.org/10.1001/jama.2019.9269)
19. Ber L. Helping physicians to understand "Havana Syndrome" and a novel method of managing AHIs. *Advanced Medical Sciences: An International Journal (AMS)*. 2025;12(1):1–11. [doi:10.5121/ams.2025.12101](https://doi.org/10.5121/ams.2025.12101).
<https://airccse.com/ams/papers/12125ams01.pdf>
20. Hoffer ME, Levin BE, Snapp H, Buskirk J, Balaban C. Acute findings in an acquired neurosensory dysfunction. *Laryngoscope Investig Otolaryngol*. 2019;4(1):124–131. Published online December 12, 2018. [doi:10.1002/lio2.231](https://doi.org/10.1002/lio2.231)
21. Balaban CD, Szczupak M, Kiderman A, Levin BE, Hoffer ME. Distinctive convergence eye movements in an acquired neurosensory dysfunction. *Front Neurol*. 2020;11:469. [doi:10.3389/fneur.2020.00469](https://doi.org/10.3389/fneur.2020.00469)
22. Aristi G, Kamintsky L, Ross M, et al. Symptoms reported by Canadians posted in Havana are linked with reduced white matter fibre density. *Brain Commun*. 2022;4(2):fcac053. [doi:10.1093/braincomms/fcac053](https://doi.org/10.1093/braincomms/fcac053)
23. Friedman A, Calkin C, Adams A, et al. Havana Syndrome among Canadian diplomats: brain imaging reveals acquired neurotoxicity. *medRxiv* [preprint, not peer reviewed]. Posted September 29, 2019. [doi:10.1101/19007096](https://doi.org/10.1101/19007096)
24. Ber L. Re-analysis of MRI Findings in Validated Havana Syndrome Cases (AHI1) in Peirpaoli [sic] et al. (2024) and Comparative Insights from Verma et al. (2019). *Int J Innov Res Med Sci*. 2026;11(01):08–14. Published January 2, 2026. [doi:10.23958/ijirms/vol11-i01/2152](https://doi.org/10.23958/ijirms/vol11-i01/2152)
25. Giordano J. Evaluating anomalous health incidents of the Havana Syndrome: the case for a structured qualitative and quantitative symptom assessment instrument. *EC Neurology*. 2025;17(9):01–09. Published August 22, 2025. <https://ecronicon.net/ecne/evaluating-anomalous-health-incidents-of-the-havana-syndrome-the-case-for-a-structured-qualitative-and-quantitative-symptom-assessment-instrument>
26. Centers for Disease Control and Prevention. Cuba Unexplained Events Investigation: Final Report (Havana, Cuba, August 2016 to March 2019). December 3, 2019. Released under FOIA; posted by the National Security Archive, February 2, 2021. <https://nsarchive.gwu.edu/briefing-book/cuba/2021-02-02/cdc-report-havana-syndrome-medical-mystery-remains-unresolved>
27. Marshall K, Sullivan K, French LM. Clinical presentation and engagement with a cognitive rehabilitation clinic in individuals reporting anomalous health incidents. *Front Neurol*. 2026. Published August 26, 2026. [doi:10.3389/fneur.2026.1858023](https://doi.org/10.3389/fneur.2026.1858023)
28. Tripathi A, Llorin J, Brody DL. Self-reported effects of incobotulinumtoxinA on

- headache with migraine-like characteristics in participants with traumatic brain injury vs. anomalous health incidents treated at a single specialty center. medRxiv [preprint, not peer reviewed]. Posted August 19, 2026. [doi:10.64898/2026.08.18.26360627](https://doi.org/10.64898/2026.08.18.26360627)
29. Biggs AT, Henry SM, Johnston SL, Whittaker DR, Littlejohn LF. Unconventionally acquired brain injury: guidance and instruction about an emerging challenge to warfighter brain health. *J Spec Oper Med*. 2021;21(2):44–48.
 30. Defense Health Agency. Assessment and Management of Reported Anomalous Health Incidents: Clinical Recommendation for Primary Care Managers. April 2025. <https://www.health.mil/Reference-Center/Publications/2025/04/16/AHI-Clinical-Recommendation>
 31. U.S. Department of Justice. Implementation of HAVANA Act of 2021. Interim final rule. 89 Fed. Reg. 28633–28638. April 19, 2024. FR Doc. 2024-08336. <https://www.federalregister.gov/documents/2024/04/19/2024-08336/implementation-of-havana-act-of-2021>
 32. U.S. Department of War. Department of War Establishes the Directed Energy Bio-Effects Cross-Functional Team, Delivering First HAVANA Act Payments [news release]. July 10, 2026. <https://www.war.gov/News/Releases/Release/Article/4540375/departments-of-war-establishes-the-directed-energy-bio-effects-cross-functional/>
 33. Office of the Director of National Intelligence. (U) Congressional Notification: Rescission of Intelligence Community Assessments on Anomalous Health Incidents (AHI) and Directed Review. Memorandum signed by Sara Lynn Pickett, Assistant Director of National Intelligence, Office of Legislative Affairs; dated 6/11/26. <https://intelligence.house.gov/wp-content/uploads/2026/06/ICA-Recall-Notification.pdf>
 34. Centers for Disease Control and Prevention / ATSDR FOIA Office. Final response to FOIA request 25-00184-FOIA (records on tracking AHI and Havana Syndrome among the U.S. population, 2018–2024), with released records, including (FOIA pp. 4–5 of the released records) the e-mail from Ted Pestorius, Deputy Director, Management and Overseas Operations, CDC Center for Global Health, to the CDC CGH Overseas Country Deputy Directors and Overseas Country Directors lists, "A Message from NSC on Anomalous Health Incidents," March 25, 2022, asking them to "share this with all CDC USDH staff in your country" [U.S. direct-hire] and transmitting "CDC Anomalous Health Incidents (AHI) Guidance." March 3, 2025.
 35. Defense Health Agency Office of Inspector General, Hotline. Letter closing request for assistance, FRNO 2025-04317. March 12, 2025.
 36. County health department e-mail to a treating physician, September 2025. Available from the author on request.
 37. CDC Executive Secretariat. "Response to Petition Regarding Havana Syndrome" [e-mail to Len Ber, MD]. July 15, 2026. Available from the author on request.
 38. Ber L. Petition for NIH Action on Civilian "Havana Syndrome"/Anomalous Health Incidents: Recognition, Research Inclusion, and Clinical Protocols [petition to NIH]. September 29, 2025. <https://lenbermd.substack.com/p/formal-petition-to-the-nih>

39. Ber et al. v. Centers for Disease Control and Prevention et al., No. 8:26-cv-01822-KKM-LSG (M.D. Fla. filed June 23, 2026), Complaint, ECF No. 1.
40. Ber et al. v. Centers for Disease Control and Prevention et al., No. 8:26-cv-01822-KKM-LSG (M.D. Fla.), Defendants' Motion to Dismiss, ECF No. 13 (filed October 5, 2026).
41. Environmental Health Trust v. Federal Communications Commission, 9 F.4th 893 (D.C. Cir. 2021), Nos. 20-1025, 20-1138; slip op. at 10, 21, 30–31.
<https://media.cadc.uscourts.gov/opinions/docs/2021/08/20-1025-1910111.pdf>
42. Federal Communications Commission, Office of Engineering and Technology. Evaluating Compliance with FCC Guidelines for Human Exposure to Radiofrequency Electromagnetic Fields. OET Bulletin 65, Edition 97-01. Washington, DC: FCC; August 1997. <https://www.fcc.gov/general/oet-bulletins-line>
43. Lin JC, Wang Z. Hearing of microwave pulses by humans and animals: effects, mechanism, and thresholds. *Health Phys.* 2007;92(6):621–628.
[doi:10.1097/01.HP.0000250644.84530.e2](https://doi.org/10.1097/01.HP.0000250644.84530.e2)
44. Pastras CJ, Gholami N, Jennings S, et al. A mathematical model for mechanical activation and compound action potential generation by the utricle in response to sound and vibration. *Front Neurol.* 2023;14:1109506. [doi:10.3389/fneur.2023.1109506](https://doi.org/10.3389/fneur.2023.1109506)
45. Elder JA, Chou CK. Auditory response to pulsed radiofrequency energy. *Bioelectromagnetics.* 2003;24(Suppl 6):S162–S173. [doi:10.1002/bem.10163](https://doi.org/10.1002/bem.10163).
46. Lin JC. The microwave auditory effect. *IEEE J Electromagn RF Microw Med Biol.* 2022;6(1):16–28. [doi:10.1109/JERM.2021.3062826](https://doi.org/10.1109/JERM.2021.3062826)
47. Lin JC. Microwave auditory effects among U.S. government personnel reporting directional audible and sensory phenomena in Havana. *IEEE Access.* 2022;10:44577–44582. [doi:10.1109/ACCESS.2022.3168656](https://doi.org/10.1109/ACCESS.2022.3168656).
48. Lin JC. The Havana syndrome and microwave weapons [Health Matters]. *IEEE Microw Mag.* 2021;22(11):13–14. [doi:10.1109/MMM.2021.3102201](https://doi.org/10.1109/MMM.2021.3102201).
49. Lin JC. Health and safety practices and policies concerning human exposure to RF/microwave radiation. *Front Public Health.* 2025;13:1619781.
[doi:10.3389/fpubh.2025.1619781](https://doi.org/10.3389/fpubh.2025.1619781)
50. Baker CD, Baker M, Idesman A, Joshi RP. Modeling electromagnetically driven thermoacoustic pressure wave buildup in the brain. *Biomed Phys Eng Express.* Published online October 6, 2026. [doi:10.1088/2057-1976/aeb0b2](https://doi.org/10.1088/2057-1976/aeb0b2). PMID: 42838080.
51. Yaghmazadeh O. Pulsed high-power radio frequency energy can cause non-thermal harmful effects on the brain. *IEEE Open J Eng Med Biol.* 2024;5:50–53.
[doi:10.1109/OJEMB.2024.3355301](https://doi.org/10.1109/OJEMB.2024.3355301)
52. Vernier PT, Sun Y, Marcu L, Craft CM, Gundersen MA. Nanoelectropulse-induced phosphatidylserine translocation. *Biophys J.* 2004;86(6):4040–4048.
[doi:10.1529/biophysj.103.037945](https://doi.org/10.1529/biophysj.103.037945)
53. Roth CC, Tolstykh GP, Payne JA, et al. Nanosecond pulsed electric field thresholds for nanopore formation in neural cells. *J Biomed Opt.* 2013;18(3):035005.

- [doi:10.1117/1.JBO.18.3.035005](https://doi.org/10.1117/1.JBO.18.3.035005)
54. Schmid MR, Loughran SP, Regel SJ, et al. Sleep EEG alterations: effects of different pulse-modulated radio frequency electromagnetic fields. *J Sleep Res.* 2012;21(1):50–58. [doi:10.1111/j.1365-2869.2011.00918.x](https://doi.org/10.1111/j.1365-2869.2011.00918.x)
 55. Raslear TG, Akyel Y, Bates F, Belt M, Lu ST. Temporal bisection in rats: the effects of high-peak-power pulsed microwave irradiation. *Bioelectromagnetics.* 1993;14(5):459–478. [doi:10.1002/bem.2250140507](https://doi.org/10.1002/bem.2250140507). PMID: 8285916.
 56. Lu ST, Mathur SP, Akyel Y, Lee JC. Ultrawide-band electromagnetic pulses induced hypotension in rats. *Physiol Behav.* 1999;67(3):753–761 (corrected and republished from *Physiol Behav.* 1999;65(4–5):753–761, [doi:10.1016/s0031-9384\(98\)00214-5](https://doi.org/10.1016/s0031-9384(98)00214-5)). PMID: 10497968.
 57. Gao P, Chen Q, Hu J, et al. Effect of ultra-wide-band electromagnetic pulses on blood-brain barrier permeability in rats. *Mol Med Rep.* 2020;22(4):2775–2782. [doi:10.3892/mmr.2020.11382](https://doi.org/10.3892/mmr.2020.11382)
 58. IEEE. IEEE Standard for Safety Levels with Respect to Human Exposure to Electric, Magnetic, and Electromagnetic Fields, 0 Hz to 300 GHz. IEEE Std C95.1-2019. New York: IEEE; 2019. [doi:10.1109/IEEESTD.2019.8859679](https://doi.org/10.1109/IEEESTD.2019.8859679).
 59. Bailey WH, Harrington T, Hirata A, et al. Synopsis of IEEE Std C95.1-2019 "IEEE Standard for Safety Levels With Respect to Human Exposure to Electric, Magnetic, and Electromagnetic Fields, 0 Hz to 300 GHz." IEEE Access. 2019;7:171346–171356. [doi:10.1109/ACCESS.2019.2954823](https://doi.org/10.1109/ACCESS.2019.2954823).
 60. Frey AH. Human auditory system response to modulated electromagnetic energy. *J Appl Physiol.* 1962;17(4):689–692. [doi:10.1152/jappl.1962.17.4.689](https://doi.org/10.1152/jappl.1962.17.4.689)
 61. Neubauer C, Phelan AM, Kues H, Lange DG. Microwave irradiation of rats at 2.45 GHz activates pinocytotic-like uptake of tracer by capillary endothelial cells of cerebral cortex. *Bioelectromagnetics.* 1990;11(4):261–268. [doi:10.1002/bem.2250110402](https://doi.org/10.1002/bem.2250110402)
 62. Merritt JH, Chamness AF, Allen SJ. Studies on blood-brain barrier permeability after microwave-radiation. *Radiat Environ Biophys.* 1978;15(4):367–377. [doi:10.1007/BF01323461](https://doi.org/10.1007/BF01323461)
 63. Chou CK, Galambos R, Guy AW, Lovely RH. Cochlear microphonics generated by microwave pulses. *J Microw Power.* 1975;10(4):361–367. [doi:10.1080/00222739.1975.11688973](https://doi.org/10.1080/00222739.1975.11688973)
 64. Honig MG, Dorian CC, Worthen JD, et al. Progressive long-term spatial memory loss following repeat concussive and subconcussive brain injury in mice, associated with dorsal hippocampal neuron loss, microglial phenotype shift, and vascular abnormalities. *Eur J Neurosci.* 2021;54(5):5844–5879. [doi:10.1111/ejn.14711](https://doi.org/10.1111/ejn.14711)
 65. Gangolli M, Benetatos J, Esparza TJ, et al. Repetitive concussive and subconcussive injury in a human tau mouse model results in chronic cognitive dysfunction and disruption of white matter tracts, but not tau pathology. *J Neurotrauma.* 2019;36(5):735–755. [doi:10.1089/neu.2018.5700](https://doi.org/10.1089/neu.2018.5700)
 66. Xu X, Cowan M, Beraldo F, et al. Repetitive mild traumatic brain injury in mice

- triggers a slowly developing cascade of long-term and persistent behavioral deficits and pathological changes. *Acta Neuropathol Commun.* 2021;9(1):60. [doi:10.1186/s40478-021-01161-2](https://doi.org/10.1186/s40478-021-01161-2)
67. Rosengren SM, Colebatch JG, Young AS, et al. Vestibular evoked myogenic potentials in practice: methods, pitfalls and clinical applications. *Clin Neurophysiol Pract.* 2019;4:47–68. [doi:10.1016/j.cnp.2019.01.005](https://doi.org/10.1016/j.cnp.2019.01.005).
68. Griffin J, Friden L. Havana Syndrome study halted as review finds some patients were coerced. Fox News. September 13, 2024 (quoting an NIH statement). <https://www.foxnews.com/world/havana-syndrome-study-halted-review-finds-some-patients-coerced>
69. National Intelligence Council. Updated Assessment of Anomalous Health Incidents, as of December 2024. ICA 2025-00008-B. Approved for public release January 9, 2025. <https://www.dni.gov/files/ODNI/documents/assessments/NIC-Unclassified-ICA-Updated-Assessment-AHI-December2024.pdf>
70. Havana Syndrome probe upended after reports' retraction. *Miami Herald*. June 2026 (quoting NIH Director Jay Bhattacharya). <https://www.miamiherald.com/news/nation-world/world/americas/cuba/article316239390.html>
71. Relman DA. Neurological illness and national security: lessons to be learned. *JAMA.* 2024;331(13):1093–1095. [doi:10.1001/jama.2023.26818](https://doi.org/10.1001/jama.2023.26818)