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Original Article

Re-analysis of MRI Findings in Validated Havana Syndrome Cases (AHI1) in Peirpaoli *et al*. (2024) and Comparative Insights from Verma *et al*. (2019)

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Abstract

Anomalous Health Incidents (AHIs) of the Havana Syndrome involve sudden onset of neurological, sensory, and vestibular symptoms occurring simultaneously, followed by chronic deficits. A 2024 NIH neuroimaging study concluded "no significant MRI-detectable evidence of brain injury". We re- analyzed Pierpaoli *et al*. (2024) by extracting imaging results for 43 AHI1 patients (AHI cases validated by neuro-vestibular-otolithic criteria) and 48 matched controls. AHI1 cases exhibited a consistent pattern of subtle changes. Resting-state fMRI revealed significantly reduced functional connectivity in the salience network (insula and anterior cingulate hubs) in AHI1 compared to controls (adjusted p ~0.02 for network connectivity). DTI measures did not differ after correction, but nominal microstructural abnormalities (uncorrected $p < 0.05$) were noted in midline white-matter tracts. The corpus callosum (body and genu) of AHI1 patients showed ~2-3% lower return-to-axis probability, and the right cingulum and inferior cerebellar peduncle also showed slight diffusivity reductions (all trend-level). These new AHI1-specific findings were then compared to the previous neuroimaging studies in AHI1 group by Verma *et al*. (2019). Conclusion: Focused analysis of validated AHI1 patients in Pierpaoli *et al*. (2024) reveals reduced connectivity in salience network, while subtle white-matter microstructural alterations closely parallel those of the Verma *et al*. (2019) AHI1 cases. This strengthens the evidence that AHI1 phenotype represents a genuine diffuse brain injury syndrome. Advanced neuroimaging and qEEG techniques may be required to sensitively detect AHI1-related brain abnormalities. Public health efforts are warranted to issue diagnostic criteria and address cases in both governmental and civilian populations.

Keywords: ***Havana Syndrome; Anomalous Health Incidents; MRI, qEEG, diagnostic criteria, NIH, CDC.***

1. Introduction

Anomalous Health Incidents (AHIs) of Havana Syndrome refer to a cluster of acute neurological symptoms first reported in U.S. diplomatic personnel in 2016, and later reported in other geographical locations, including on US Soil, and in civilians [1,2]. The acute symptoms often include loud perceived noises, vibrating pressure sensations (aka buffeting), vertigo, ear pain, cognitive difficulties, and other neurological impairments occurring together, followed by chronic sequelae [3]. The leading hypothesis is exposure to directed, pulsed radiofrequency (RF) electromagnetic energy as the cause, as established by two consensus reports [4,5]. We suggested to recognize the syndrome as a novel form of non-kinetic (no impact) brain injury distinct from conventional concussion or acoustic trauma [6]. By 2025 it is widely accepted that directed energy exposure is the most probable cause [7].

Early in the investigation, Hoffer *et al*. (2018) established set of diagnostic criteria based on the first 25 confirmed cases; the original "Havana Cohort" [8]. Patients meeting Hoffer's criteria (here termed AHI1) displayed a distinctive pattern of objective neuro-vestibular deficits: ocular-motor abnormalities (e.g. convergence insufficiency, anti-saccades test) and vestibulo-ocular reflex impairments disproportionate to age, along with otolith organ dysfunction on testing in nearly 100% of cases. These objective neuro- vestibular findings effectively rule out purely psychogenic causality. Patients reporting AHI-like symptoms who do not meet Hoffer's objective criteria are classified as AHI2. This AHI1/AHI2 stratification aligns with the approach used in the recent NIH investigations and helps isolate those with verifiable injury from those with non-specific or functional disorders [9,10].

Given this clinical framework, researchers turned to neuroimaging to investigate potential brain changes in AHI1. Verma *et al*. (2019) (further referred to as Verma) reported modest but statistically significant neuroimaging differences in AHI1 patients compared to controls, including changes in white matter volume, microstructure, and functional connectivity [11].

Given the alarming outcome, NIH launched a comprehensive prospective study in 2019 –2020, ultimately evaluating 81 individuals reporting AHIs and 48 controls. Results from this NIH cohort were published in 2024 [9]. Pierpaoli *et al*. (2024) focused on advanced neuroimaging, and Chan *et al*. (2024) reported clinical and laboratory findings [9,10]. At the group level, the

NIH AHI cohort (which included both AHI1 and AHI2 cases) showed no significant brain differences from controls. This categorical conclusion prompted widespread debate and calls for re-analysis of clinically validated AHI1 group vs. control [12-15]. The concerns centered on methodological limitations, particularly the failure to stratify by clinical phenotype. Yet, until now, no formal re-analysis of the Pierpaoli *et al*. 2024 data by AHI1 subgroup had been formally undertaken.

In this work, we re-analyzed the 2024 NIH dataset, focusing specifically on the AHI1 subgroup versus controls to identify subtle brain alterations that may have been obscured in the mixed cohort. We then compared these AHI1-specific findings (further referred to as Pierpaoli AHI1) those of Verma to assess convergence and divergence from two independent studies (All Verma patients were AHI1-validated cases.) Additionally, we discuss neurophysiological evidence from quantitative EEG in a representative AHI1 case, to determine whether electrophysiological changes might parallel the MRI findings and could provide a potential complementary diagnostic tool, among others. AHI1 prevalence is discussed, with emphasis on the under-diagnosed, unacknowledged, and un-researched civilian cases.

2. Materials and Methods

We re-analyzed imaging data from the exploratory NIH study by Pierpaoli *et al*. (2024), specifically the subgroup of participants classified as AHI1 (acute-onset cases validated by Hoffer's 2018 neuro-vestibular criteria) versus healthy controls [9,8]. The NIH study enrolled 81 individuals with suspected AHIs (43 AHI1 and 38 AHI2) and 48 age- and sex-matched controls, with MRI acquisition at the NIH Clinical Center from 2018–2022. AHI2 cases were excluded from re-analysis because they didn't meet validation criteria of AHI1.

The NIH imaging protocol included high-resolution structural MRI, diffusion MRI (dMRI), and resting- state functional MRI. Within-network functional connectivity was computed for 13 standard resting-state networks by averaging Fisher-transformed correlation coefficients among regions of interest (ROIs) in each network. Diffusion metrics included fractional anisotropy (FA) and an advanced index, return-to-axis probability (RTAP), which is sensitive to restricted diffusion perpendicular to white-matter fibers. ROI- based analyses compared AHI1 vs. controls on volumetric and dMRI metrics for key brain regions (corpus callosum, major white-matter tracts, etc.) using nonparametric tests, with Benjamini–Hochberg false discovery rate correction as reported by Pierpaoli *et al*. We noted both the uncorrected p-values and whether they remained significant after correction.

For our re-analysis, we extracted the published AHI1 vs. control results as documented in Pierpaoli *et al*. (2024) and its Supplement (eFigures, eTables). Both resting-state functional connectivity measures and diffusion MRI metrics were considered. Further, the re-analysis was compared to corresponding findings in Verma *et al*. (2019) [11].

3. Results

3.1. Re-analysis of Pierparoli AHI1

Functional Connectivity

Within-network resting-state connectivity analysis revealed statistically significant reductions in salience network connectivity in AHI1 participants compared to controls. In the Pierpaoli AHI1 data, the anterior salience network showed a marked decrease in connectivity in AHI1 subjects (Mann–Whitney U test $p = 0.002$; Benjamini–Hochberg adjusted $p = 0.020$). The posterior salience network was similarly affected (unadjusted $p = 0.004$; adjusted $p = 0.030$). No other resting-state network reached significance after correction, although uncorrected p-values suggested trends toward lower connectivity in other sensory- integration networks (e.g., sensorimotor and dorsal attention networks). These results indicate a selective vulnerability of salience-processing hubs in AHI1, particularly the insula and anterior cingulate cortex, which filter salient sensory stimuli and mediate transitions between cognitive states.

Diffusion MRI

At the ROI level, no diffusion metric differences between AHI1 and controls survived FDR correction. However, several white-matter structures showed nominal differences in uncorrected analyses ($p < 0.05$) with small effect sizes. In particular, the corpus callosum exhibited lower return-to-axis probability (RTAP) in AHI1 patients: the callosal body showed a median ~2.8% reduction (unadjusted $p \approx 0.04$, n.s. after correction) and the genu a ~3.4% reduction ($p \approx 0.03$, n.s.). The right cingulum (cingulate gyrus bundle) also had a ~2.1% RTAP decrease ($p \approx 0.047$, n.s.), and the splenium of the corpus callosum and right superior longitudinal fasciculus showed small (~2%) RTAP decreases that did not reach significance. Although none of these survived correction, the consistent pattern suggests mild microstructural changes particularly in midline interhemispheric fibers (corpus callosum) and limbic tracts (cingulum).

Summary of Pierpaoli AHI1 Re-analysis

The above Pierpaoli AHI1 subgroup findings stand in contrast to Pierpaoli *et al*.'s overall analysis of the entire AHI cohort (AHI1 + AHI2 combined), which reported essentially null results at the group level. The original mixed-cohort analysis likely diluted any neurobiological signal by including clinically heterogeneous participants, many of whom had no objective neurological findings. In our focused re- analysis of the vestibular-confirmed AHI1 subgroup, a consistent pattern of brain dysfunction clearly emerges. Moreover, participants classified as AHI2 (those who failed the vestibular validation) were frequently diagnosed with Persistent Postural-Perceptual Dizziness (PPPD) or other functional neurological disorders in the companion clinical study by Chan *et al*. (2024) [10]. This contrast reinforces the neurobiological specificity of the AHI1 group and suggests that the salience network connectivity deficits observed in AHI1 are unlikely to reflect mere stress or psychological comorbidity.

3.2. Comparison of Pierpaoli AHI1 with Verma AHI1

Verma, *et al* (2019) was the previous neuroimaging study conducted at the University of Pennsylvania which included 40 government personnel AHI1 patients verified by Hoffer (2018) diagnostic validation criteria, and 48 demographically similar healthy controls [11,8]. The study found significant neuroimaging differences in the AHI1 patient group. White matter abnormalities were a prominent feature: patients had approximately 5% lower total white matter volume than controls ($P < 0.001$), with significantly reduced white matter volume in multiple regions including the frontal, parietal, and occipital lobes. Verma also identified microstructural diffusion changes in midline brain structures. For example, patients showed lower mean diffusivity in the inferior cerebellar vermis, indicating subtle alterations in white matter integrity. Here, Verma findings are compared with the re-analysis of Pierparoli AHI1 (Table 1).

Functional Connectivity

Functional MRI connectivity findings overlapped only partially between Pierpaoli AHI1 and Verma results. Verma observed significantly reduced resting-state connectivity in the auditory and visuospatial networks patients compared to controls (FDR-corrected P ≈ .003 in both networks), with no significant change in an executive control network. By contrast, Pierpaoli AHI1 analysis detected its largest connectivity deficit in the salience network. AHI1 patients showed markedly lower within-network connectivity in both anterior and posterior salience network hubs (insula and anterior cingulate) relative to controls (Mann–Whitney P ≈ .002–.004; adjusted P ~ .02–.03). In the Pierpaoli AHI1 data, auditory network connectivity was not significantly reduced (and only a trend-level decrease was noted in a dorsal visuospatial attention network). Thus, Pierpaoli' AHI1's functional connectivity results only partially replicated the specific network deficits reported in Verma. The divergence in network findings (auditory/visuospatial vs. salience) may stem from differing analytic approaches and cohort characteristics: Verma's analysis focused on networks related to the acute sensory symptoms (e.g. hearing and spatial orientation) with salience networks, being outside of the scope, whereas Pierpaoli AHI1 surveyed a broad set of canonical networks and found salience processing to be most affected.

Diffusion MRI

Structural and diffusion MRI metrics showed further convergences and divergences between the two studies. In Verma, widespread white matter volume reductions was reported: in addition to a ~27 cm^3 lower total white matter volume, patients had significantly smaller white matter volumes in frontal, parietal, and occipital regions. Notably, Verma's diffusion tensor imaging also revealed altered microstructure in midline pathways: patients had lower mean diffusivity in the cerebellar vermis and higher fractional anisotropy in the corpus callosum splenium, indicating changes in these long-range tracts. In Pierpaoli AHI1, no MRI volume differences reached significance after correction, but subtle white matter alterations were evident at trend level. AHI1 patients showed a non-significant trend toward larger corpus callosum volume than controls, and exhibited small (~2%) decreases in diffusion integrity indices within major white matter tracts. Specifically, Pierpaoli AHI1 data showed reduced diffusion metrics (e.g. lower return-to-axis probability) in the corpus callosum (body and genu), as well as nominally lower indices in the right cingulum and right superior longitudinal fasciculus, compared to controls (uncorrected P ~ 0.03–0.05 in these regions). None of these tract differences survived false-discovery rate correction. Despite the modest magnitude of Pierparoli AHI1's diffusion findings, the affected pathways align with those highlighted in Verma. Both studies independently implicate midline interhemispheric fibers and cerebellar-vestibular circuits as sites of abnormality in AHI1. For instance, Verma documented increased anisotropy in the splenium of the corpus callosum, and the Pierpaoli AHI1 analysis likewise pointed to callosal involvement (through a different diffusion measure showing reduced microstructural coherence in the callosal body). In addition, Pierpaoli AHI1 observed an uncorrected diffusion difference in the inferior cerebellar peduncle, mirroring Verma's finding of altered diffusion in the cerebellar vermis and providing convergent evidence of cerebello-vestibular tract involvement.

Although Pierpaoli *AHI1* results were more subtle and mostly trend-level, both studies converge on a consistent anatomical pattern: key midline and long-range white matter tracts (notably the corpus callosum, cerebellar connections, and cingulum) emerge as common loci of change in individuals with AHI1.

3.3 Pierparoli AHI1 and Verma Comparison Conclusions

Pierpaoli AHI1 re-analysis and Verma converge on a consistent neuroanatomical pattern: both identify midline and long-range white matter tracts, - notably the corpus callosum, cerebellar connections, and cingulum as loci of change in AHI1. At the same time, they diverge in certain network-level details and in the magnitude of observed differences, reflecting both a core common pathology and variability likely related to cohort characteristics and analytic approaches. Verma's scope excluded silence network that Pierparoli AHI1. identified as locus of hypoconnectivity, even after adjustment. Taken together, these independent MRI studies strengthen the evidence that AHI1 is associated with subtle but reproducible brain abnormalities (**see Table 1**).

Table 1: Comparative Summary of AHI1 Neuroimaging Findings in Pierpaoli (2024) and Verma (2019)

Functional Network/ROI	Pierpaoli AHI1 (NIH re-analysis)	Verma AHI1 (2019)	Convergence / Divergence
Salience network	Significantly reduced connectivity in anterior/posterior salience network. (p = 0.002–0.004 unadj; p = 0.02–0.03 adj)	Not investigated, not reported	**Divergence*** – Different networks implicated. Salience identified only in Pierpaoli.
Auditory network	No significant difference.	Significantly reduced connectivity ($p \approx 0.003$, adj).	**Divergence*** – Auditory network deficit only in Verma.
Visuospatial attention network	Trend-level decrease; not significant after correction.	Significantly reduced connectivity ($p \approx 0.003$, adj).	**Partial convergence** – Both studies show effect; only Verma reached significance.
Corpus callosum	~2–3% RTAP reduction in genu/body ($p \approx$ 0.03–0.04 unadj; n.s. after adj); slight volume increase (trend-level).	Reduced white-matter volume (~5%); altered FA in splenium (significant).	**Convergence** – Both find structural abnormalities in CC, with stronger effects in Verma.
Cingulum	~2.1% RTAP decrease in right cingulum ($p \approx$ 0.047 unadj; n.s. after adj).	Implicated in general white-matter change; no standalone stat reported.	**Convergence** – Both indicate limbic tract involvement.
Cerebellar-vestibular pathways	Nominal diffusivity reduction in inferior cerebellar peduncle (uncorrected $p < 0.05$); not significant after correction.	Lower mean diffusivity in cerebellar vermis (significant).	**Convergence** – Both report cerebellar microstructural changes in AHI1.

** Divergence between Pierpaoli and Verma in functional networks (e.g., salience vs. auditory) may reflect differences in analytic focus. Verma emphasized networks linked to acute sensory symptoms and excluded salience networks from the scope of the study, while the NIH study examined a broader set of canonical networks. These differences suggest AHI1 affects multiple systems, with the most salient disruption detected varying by method and sample.*

4. Discussion

Our re-analysis of the NIH AHI1 subgroup reveals subtle but consistent brain changes that align closely with findings from Verma *et al*. (2019) in cases validated by objective neuro-vestibular-otolithic abnormalities [9,11]. Both studies independently implicate midline and long-range white matter tracts (corpus callosum, cingulum, and cerebellar-vestibular pathways) as common sites of injury. The convergence across cohorts studied years apart by independent teams supports the interpretation of AHI1 as a genuine neurobiological entity: a diffuse, non-kinetic brain injury with a repeatable white-matter signature.

Although the anatomical findings overlap, the studies differ in which functional networks were most affected. Verma reported significant reductions in auditory and visuospatial connectivity, while our re- analysis of Pierpaoli AHI1 found the largest disruption in the salience network. This divergence likely reflects differences in analytic focus and cohort characteristics: Verma explored networks linked to acute sensory symptoms and excluded salience networks from the scope of the study, whereas the NIH study evaluated a broader set of canonical networks. Both are plausible, and together suggest that AHI1 disrupts multiple interconnected systems, with variable expression depending on methodology and clinical presentation.

Importantly, these abnormalities are not merely artifacts of group-level noise or statistical chance. While many findings are near the threshold of statistical significance, their anatomical and functional coherence across studies lends them credibility. The repeated involvement of structures like the corpus callosum, cerebellar tracts, and limbic fibers is unlikely to be coincidental. These regions form an integrated circuitry for multisensory integration, spatial orientation, and attentional filtering. These are the domains plausibly impaired in AHI1 patients who report vertigo, cognitive fog, and disorientation. The injury pattern appears diffuse and non-acute, consistent with a non-kinetic mechanism. As Pierpaoli *et al*. observed, the absence of overt MRI lesions does not imply the absence of injury, but may reflect microstructural or network-level changes too subtle for standard 3T MRI to capture [9]. These findings should not be grounds for dismissal, but rather a "green light" for applying more sensitive detection methods.

The specificity of this pattern to AHI1 further strengthens the case for its biological reality. AHI2 patients, (those reporting symptoms without objective vestibular findings, showed no corresponding MRI abnormalities). In the NIH clinical companion study, many were diagnosed with functional disorders such as Persistent Postural-Perceptual Dizziness (PPPD), conditions associated with stress rather than structural neurotrauma [10]. Notably, Pierpaoli's original group-wide analysis (combining AHI1 and AHI2) yielded null results, almost certainly due to signal dilution from heterogenous, unvalidated AHI2 cases [9].

This contrast highlights a critical point: imaging abnormalities in AHI1 are not attributable to stress or psychogenic illness. When patients meet stringent AHI1 criteria, they consistently show a pattern of objective brain changes consistent with a mild acquired brain injury. In contrast, AHI2 more closely resembles functional dizziness with no evidence of neuroanatomical disruption. This distinction has clear implications. Clinically, it affirms that properly defined Havana Syndrome cases involve real neurological injury. Methodologically, it underscores the need for precise case definitions in future studies. Inclusion of unverified or psychogenic cases risks masking subtle effects, and by stratifying AHI1 from AHI2 we can yields valid results.

qEEG swLoreta findings in a validated AHI1 case

To date, no formal reports of electric neuroimaging such as quantitative EEG with swLoreta (standardized weighted Low-Resolution Electromagnetic Tomography) has have been available for the AHI1 cohort. While it is not a stand-alone diagnostic method, qEEG with swLORETA allows to map EEG signals to specific brain regions, making it a valuable tool for detecting network-level dysfunction in conditions like mild brain injury alongside MRI [16].

Here, we discuss qEEG with swLoreta findings in a verified AHI1 patient (male, 56 years old) who met the stringent neuro-vestibular criteria for the syndrome, including documented severe unilateral otolith organ damage on vestibular testing [17]. Patient's qEEG revealed aberrant brain network activity that converges with the MRI-defined pattern described above. Notably, this patient's qEEG demonstrated disrupted functional connectivity involving fronto-limbic regions and interhemispheric pathways, paralleling the salience network and midline tract abnormalities seen on MRI. For example, source analysis of resting- state EEG showed relative hypoactivity in the anterior cingulate cortex and insular regions (key salience network nodes) alongside disturbances in temporoparietal areas involved in auditory and spatial processing. This is an overlap with both the Pierpaoli AHI1 and Verma network findings. Moreover, the qEEG identified additional features not captured by MRI: an abnormal prominence of low-frequency (delta-band) activity in frontal-central regions. Such delta-band abnormalities are often observed in patients with diffuse brain injury or concussion, reflecting cortical deafferentation or slowed network oscillations [18]. The presence of focal delta irregularities in this AHI1 patient dovetails with the notion of a mild traumatic encephalopathy, similar to patterns reported in mild TBI, where decreased alpha and increased slow-wave power signify network-level dysregulation [19]. Additionally, a Canadian study in AHI patients revealed pronounced delta-band abnormalities on MEG [20].

Although drawn from a single-case observation, these qEEG results reinforce the reality of AHI1-related brain dysfunction. They echo the MRI changes in midline and salience-related circuits while also highlighting electrophysiological perturbations that may serve as sensitive indicators of brain injury in AHI1. This illustrates the potential of advanced neurodiagnostic modalities, such as EEG-based network mapping, to detect and monitor AHI1 neural changes in ways that complement conventional MRI. We understand the limitations of analyzing just one clinical case, but it wouldn't be fair to exclude this case of validated AHI1 from discussion amidst scarcity of formalized qEEG assessment in this distinct clinical group.

Advanced Modalities for Detecting Subtle Neurophysiological Injury in AHI1

To better detect and objectify these mild injuries, future research should leverage advanced neuroimaging and neurophysiological modalities. High-field MRI (7 Tesla and above) could improve the signal-to-noise ratio for microstructural damage in white matter tracts and perhaps reveal small foci of gliosis or micro- hemorrhage that 3T scans might miss [21]. Advanced diffusion techniques (e.g., neurite orientation dispersion and density imaging or free-water mapping) might more finely characterize the integrity of midline fibers in AHI1 patients [22]. Likewise, magnetoencephalography (MEG) and high-density EEG with source modeling should be employed to capture the functional disturbances in real time [23]. As indicated by our qEEG case, electrophysiological recordings are capable of detecting abnormal network oscillations (such as excessive delta-band synchrony) that correlate with brain injury even when structural MRI is equivocal [24]. Notably, MEG studies in

mild TBI have shown that slow-wave (delta–theta) abnormalities in frontotemporal regions can serve as biomarkers of diffuse axonal injury when MRI is normal, – a parallel that likely extends to AHI1 [24]. Finally, multimodal approaches integrating vestibular testing with neuroimaging could prove especially powerful. Given that virtually all verified AHI1 patients exhibit peripheral otolith organ damage and oculomotor deficits [25,7], combining objective vestibular diagnostics with functional brain imaging (for example, task-based fMRI during vestibular stimulation, or vestibular- evoked potentials) might pinpoint how labyrinthine injury and central integration deficits are linked in this syndrome. Thus, the available evidence (subtle but coherent) validates that AHI1 involves real brain changes, and it compels the medical community to "listen to the data" by deploying cutting-edge tools to fully characterize this novel form of mild brain injury. What eludes standard MRI may declare itself with more refined techniques.

Prevalence of AHI1 cases

While 2024 NIH studies involved government personnel and their dependents only, growing evidence indicates civilians have also been affected. An estimate based on the 2024 U.S. Government Accountability Office (GAO) report, combined with other sources, indicates over 400 AHI1 cases, including at least a dozen confirmed civilian patients [1,2]. Unlike federal employees, civilian sufferers have received no official support: the NIH has not studied civilian cases, and the Centers for Disease Control and Prevention (CDC) has issued no clinical guidance [26,27]. As a result, civilian cases are vastly under-diagnosed and under- reported.

The lack of action is not only a scientific lapse but also an ethical one, given that civilians are continuing to suffer progressive neurocognitive debilitation with no official acknowledgment [2]. Going forward, a robust public health response is urgently needed, including CDC guidance, physician alerts and education, and transparent tracking of existing and new cases in order to ensure that AHI1 is recognized and addressed in all affected populations, not just within government circles.

Our analysis and the broader literature affirm that AHI1 is a real neuropathological condition. Progress, however, has been stalled by investigative setbacks. The NIH's prospective studies, once a key source of data, were abruptly halted in 2024 after an internal review found that some participants had been coerced into enrolling, undermining informed consent [28]. This coercion reportedly stemmed not from NIH investigators but from external institutional pressures, specifically, Intelligence Agencies. Out of ethical concern, NIH leadership terminated the study. This resulted in a Congressional Committee sending criminal referrals to the Department of Justice into NIH's potentially illegal action and mishandling of Havana Syndrome [29]. As of 2025, no new NIH-sponsored investigations are underway.

Going forward, a multidisciplinary, ethically sound research program is urgently needed. Standardized diagnostic criteria for AHI1 are essential. Currently, only Hoffer's neuro-vestibular syndrome criteria have been applied [8]. The present article makes a case for additional diagnostic criteria. CDC must issue case definition and diagnostic criteria to ensure civilians cases are recognized and accounted for. Also, much needed AI-based diagnostic tools have also been proposed [7]. Future studies must concentrate on the validated AHI1 cases, including diagnosed civilian cases, ensure voluntary participation, independent oversight, and full transparency to rebuild trust. They must integrate diverse fields and methods, including comprehensive neurological and vestibular testing, high-resolution magnetic and electric neuroimaging, and cognitive tests, among others.

Finally, we should not be dissuaded by the Intelligence Community's inability to assign attribution in Havana Syndrome and AHI1 ("who's done it") while the scientific consensus on the most plausible mechanism ("how's it happening") exists and a clinically distinct phenotype is present (AHI1). For example, medical profession can identify asbestos injury without naming the construction company that put it in the walls. Let science lead the way of discovery unburdened by politically motivated narrative.

5. Conclusions

The emerging picture of AHI1 is that of a novel diffuse form of acquired non-kinetic (no impact) brain injury that can be characterized by subtle neural network disruptions, particularly in pathways subserving multisensory integration and salience, with a repeatable white-matter signature. To fully elucidate this condition, the research community must resume research using improved methodology and ethics, developing better diagnostic tools (potentially leveraging high-field MRI, MEG, and qEEG). CDC and NIH must establish standardized diagnostic and reporting practices. Equally important, public health authorities must recognize the reality of AHI1 in all patient groups including civilian, and mount a coordinated response. The estimated few hundred affected individuals may seem small, but the impact on each patient's life is profound, and there is potential for more cases to emerge. The medical community must be equipped to diagnose and manage Havana Syndrome/AHI1 appropriately.

Declarations

Ethical approval

Not Applicable

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Consent of Publication

Not Applicable

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