An Analysis of Data and Hypotheses Related to the Embassy Incidents

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Contents

1 EXECUTIVE SUMMARY

As of the time of our study, nearly two hundred federal employees and their family members assigned to posts in embassies and consulates around the world have reported adverse health symptoms perceived to have been precipitated by incidents experienced in their work places or residences. The reported incidents range from unfamiliar sounds to a sensation of head pressure, while the associated symptoms can include vertigo, nausea, headache, and tinnitus. In some cases, the severity and duration of the symptoms have been intense, leading to lasting cognitive and vestibular deficits. Identifying the causes of these incidents and providing mitigation and prevention strategies constitutes a high priority for the United States government. As part of this effort, JASON has been tasked by the Department of State to consider all available data and evaluate potential mechanisms with regard to their ability to produce effects that are consistent with those reported.

Making progress on identifying the causes of the health incidents (often referred to as Anomalous Health Incidents or AHIs) requires two parallel but related methodologies. The first one requires a well-defined medical case statement that is based on identifying a unique cluster of symptoms and establishing that such symptoms exceed their prevalence in a comparable background population. The second approach relies on identifying a cause or causes that can be physiologically linked to the reported symptoms and physically linked to the anecdotal[1] evidence in the incident reports, under the assumption that these symptoms are related to a single underlying cause and are not manifestations of a collection of medical conditions and functional disorders. We refer to these 'first' and 'second' approaches and discuss them in more detail below.

The Centers for Disease Control and Prevention (CDC) currently adopts a working case definition that requires the onset of symptoms in two phases: a first phase that includes at least one of head pressure, disorientation, nausea, headache, vestibular disturbances, auditory symptoms, or vision changes, followed by a second phase that includes either vestibular disturbances or cognitive deficits, with no readily recognizable alternate

[1]We use anecdotal in the following sense, in accordance with the Oxford English Dictionary: "consisting of, or based on, reports of individual cases rather than systematic research or analysis."

explanation. This case definition is based on the incidents self-reported by the affected personnel and on subsequent medical evaluations of the associated symptoms.

The Department of State provided to JASON incident data files that included, to varying degrees of detail, the location and narrative of a reported incident, the immediate sensations described by the individual, medical symptoms that developed over time, and a description of environmental factors that may have been pertinent to the incident or the associated symptoms. The accounts are highly varied in many regards: the number and specificity of the queries and responses naturally evolved substantially with time; the timing of the reports ranged from minutes to years after the incident, introducing different degrees of recall bias; the number of reported symptoms accompanying an incident varied from none to the full set included in CDC's criteria.

The narrative nature of the reports precluded a quantitative analysis. Upon performing a qualitative systematic review, JASON finds that the reported incidents themselves did not share any uniquely identifiable common set of characteristics. Further, JASON finds natural and credible explanations for these incidents in all but 20-30 out of the approximately 200 cases because either the incidents and symptoms fall within the realm of everyday common occurrences or the symptoms can be explained in ways that are not related to the perceived incidents.

With regard to the medical data, investigators at the National Institutes of Health (NIH) presented to JASON preliminary population-aggregated data of clinical test results for 65 individuals enrolled in an ongoing 5-year study on traumatic brain injury (TBI) of overseas U.S. personnel Shahim et al. (2021). The study began in May 2018 and was designed to evaluate whether signs of brain injury were present in any of the personnel in the days to years following the incidents. The data included outcomes from a multitude of diagnostic tests, such as MRI imaging, audiology exam, serum biomarkers, and assessment of vestibular function (inner ear and balance) tests including eye movement, gait, and Vestibular Evoked Myogenic Potential (VEMP). JASON finds no compelling evidence of TBI in the data obtained to date. We note that the NIH study has not been completed and that with additional data, the NIH may come to conclusions different from JASON's.

Having carefully evaluated all available incident and medical data, we find that statistical conclusions from the first, data-driven approach cannot be drawn with even a low degree of confidence because:

- Appropriate control groups for the personnel in question need to be lifestyle matched and would, therefore, ideally be limited to embassy personnel on active duty. Because this requirement poses significant logistical challenges, the control groups enrolled in current studies are small, heterogeneous, or not appropriately matched.
- The number of cases showing a substantial range of signs and symptoms is small, leading to significant statistical fluctuations in the sample and uncertainties in the results.
- Administering many successive independent tests to the affected individuals, without a clear hypothesis, increases the risk of encountering a spurious correlation.
- Self-reporting, varied initial evaluation criteria, and additional criteria for admission into the NIH clinical study each contribute to selection effects that are difficult to quantify and disentangle.

The net result of these complications is an inability to provide a rigorous case definition and, therefore, a corresponding lack of guidance toward an underlying cause based on this data-driven approach at this time.

Turning to the second parallel methodology, JASON evaluated multiple mechanisms as potential causes that could establish a physical and physiological connection between the reported symptoms and the information in the incident reports. In particular, the anecdotes provided in the incident data would require mechanisms of damage that can be directed covertly and over short periods of time at individuals who are indoors (as reported in more than 90% of the nearly 200 cases) and localized to a space of less than a few meters (as reported in approximately 75% of the cases). The proposed mechanisms would also have to induce recognizable and lasting health effects on humans through some identifiable physiological pathway. In our evaluation of potential mechanisms, we assessed the physics

of delivering directed energy to an individual consistent with the incident reports and we evaluated the potential of such mechanisms to cause sensations and symptoms consistent with the medical evaluations. We refer to these as our incident consistency criteria. In devising these criteria, we considered more heavily the smaller subset (approximately 15%) of cases that did not have immediate common or environmental explanations, rather than include the remainder that appeared to be more consistent with everyday occurrences of events and symptoms. The incident consistency criteria, discussed further in Section 2, contain the following: energy must be able to penetrate into buildings through walls and windows from a distance of several tens of meters, be localized to areas the size of a room or smaller, be generated with equipment that is mobile and covert, lead to audible and/or recordable sounds, cause immediate symptoms of pressure, vertigo, headache, or nausea, and cause long-term vestibular and/or cognitive dysfunction.

With regard to direct physical attacks, the scope of this JASON study was focused on mechanisms that can deliver energy from a distance, owing to the thorough work presented in previous investigations on mechanisms involving biological agents and toxins (such as the 2018 JASON study and the 2020 study by the National Academy of Sciences). We investigated electromagnetic energy delivery in the form of radio-frequency (RF) and ionizing radiation, and acoustic energy delivery in the form of infrasound, audible, and ultrasound waves. Present data allow us to rule out some of these mechanisms with a high degree of confidence. We propose methods for collecting data to assess the remaining possibilities more fully.

We also evaluated the potential role of functional disorders, i.e., conditions involving altered biological function without known structural change, in producing the reported cluster of symptoms (in contrast to physical attacks that can directly cause physiological damage). We considered functional disorders with different types of precipitating events, including purposeful attacks, inadvertent physical insults, events that can be considered ordinary but have an amplified effect due to prevailing circumstances (akin to post-traumatic stress disorder), medical conditions unrelated to any external insult, or unidentified events that precipitate a functional condition. We found that these disorders potentially explain at least a subset of the long-term symptoms reported by the affected personnel, even though

it is not possible to identify a common initial stimulus consistent with all the events and criteria.

Our analysis provides a way to focus future data collection on a hypothesis-based definition of Anomalous Health Incidents that combines both methodologies employed here. Having reviewed all incident data to select the cases that are potentially consistent with AHI and having explored a range of physically plausible mechanisms of attack or harassment, we identify the most relevant criteria on which a future incident questionnaire can be based. Furthermore, assigning numerical weights to the responses in the questionnaire can facilitate a rapid and uniform initial assessment of reported incidents across all embassies, allow for an initial triage, and help guide appropriate medical and monitoring response. We recommend that only incidents that meet a threshold be labeled as AHI and selected into a different database to increase the statistical robustness in subsequent hypothesis evaluations. Naturally, medical care and support of the affected individuals should be a high priority independent of the AHI-consistency decisions. In other words, we emphasize that judging a case to be inconsistent with an AHI carries no implications about its importance or severity from a medical standpoint.

Regarding adversarial intent, it is not possible to conclude at this time that the events reviewed by JASON are the result of intentional attacks that cause physical harm. However, it is not possible, either, to rule out mechanisms that do not cause any physical harm but which might constitute harassment and lead to health conditions and functional disorders, for example through unpleasant sounds or pressure sensations. Given this, and in the interest of protecting embassy personnel and their families, it would be prudent to be vigilant against tactics intended to produce anxiety and trauma, with an intent to either disrupt operations and/or cause long-term harm. The US government could minimize the effects of such tactics, if present, through open communication, education, and appropriate rapid medical response to any conditions that develop.

We provide our full findings and recommendations below.

Findings

F1. 85-90% of the reported incidents are consistent with common and known symptoms of pathophysiological or environmental origins; the remainder appear more complex and defy a straightforward explanation at present.

F2. The use of uniform, structured incident intake questionnaires with quantifiable responses can help homogenize collected data, be used for preliminary assessment (e.g., through assigning numerical values to responses) and appropriate rapid response, and may help illuminate patterns and potential causes.

F3. The medical data collected through the NIH study are not, on their own, sufficient to explain the reported abnormal signs and symptoms. Furthermore, the aggregated medical data are not sufficient to infer the existence of a novel clinical syndrome[2].

F4. Under the CDC's working definition of a case, the reported collection of symptoms could derive from a structural cause, functional disorder, psychogenic response, or some combination of the three. The government's response to the reported incidents may be causing additional stress to the affected individuals and may exacerbate normal psychological responses that are almost certainly present.

F5. On evaluation of specific energy delivery hypotheses on the basis of the combination of medical and situational data, and physical considerations, we find that:

- No single hypothesized mechanism can explain all of the incidents.

[2]Used here in accordance with the medical definition of a syndrome: a group of signs and symptoms that occur together and characterize a particular abnormality or condition, for which a direct cause may not be understood.

- Ionizing radiation, electromagnetic energy below 500 MHz and above 30 GHz, and sonic (infrasound/audible/ultrasound) energy delivered from a distance can, with high confidence, be eliminated as potential causes.

- Short-pulse radio frequency (RF) radiation primarily in the range of 500 MHz to 30 GHz cannot be ruled out conclusively as a modality for a subset of the incidents at this time. However, it is unlikely given that there is no well-documented or broadly accepted mechanism to induce lasting neurological or other damage with focusable RF energy that would not produce a sensation of heating on the skin.

- Sounds associated with cranial acoustic sensations due to pulsed RF waves (the "Frey effect") cannot produce brain damage by mechanical means at tolerable sound levels. Further, such sensations cannot be recorded by electronic microphones and, therefore, cannot be associated with any recorded acoustic phenomena.

F6. The absence of observed disruption of electronics (e.g., cellphones, televisions, computers) reduces the allowed parameter space of any potential pulsed RF signals but does not rule them out, given that contemporary consumer electronics may not be affected by short pulses at peak flux levels below approximately 25 W/cm^2.

F7. Persistent monitoring with appropriate sensors at select locations is needed for further evaluation of RF hypotheses and could help exclude this remaining potential mechanism.

F8. It is JASON's judgement that, on the basis of available reports of events, related data, and health evaluations, it is not possible to conclude at this time that these events are the result of intentional attacks that cause physical harm.

Recommendations

R1. DOS should modify its questionnaire for incident reporting to enable a uniform, structured, and quantitative dataset, focusing on surveying specific physical sensations that would accompany remote energy attacks. Such a questionnaire can be used to minimize the variance and noise in data collection, to provide an initial triage, and to initiate rapid deployment of follow-up environmental monitoring. JASON provides an example of such a questionnaire in the report.

R2. DOS should categorize only the incidents that meet a threshold as AHI and select these into a different database. The threshold should be based on the intake questionnaire to provide clarity as quickly as possible to the affected personnel. DOS should guide individuals to appropriate medical care and support independent of the AHI-consistency decisions.

R3. DOS should develop criteria (e.g., linear classifiers) to rank the relevance of incident reports in the context of hypothesis testing. The focus should be on hypotheses that have not been ruled out by existing analyses.

R4. In conjunction with other agencies, DOS should consider using the uniform triage process to select affected individuals for inclusion in future medical studies, carrying out a specific series of tests all done in the same order and within a specified time frame, striving to make appropriately selected control groups larger, and providing incentives for individuals to participate in the control groups.

R5. Informed by related efforts by other agencies, DOS should analyze existing and future patient-level health data in conjunction with reported sensory symptoms to identify possible clusters, which may help define a potentially novel syndrome.

R6. DOS and collaborating agencies should adapt their baseline medical evaluation

program so that it is more comprehensive and responsive to a variety of hypothesized harm mechanisms, to be administered prior to deployment overseas. These evaluations should be used on an individual basis to provide context for any future health events and in an aggregated manner to provide an appropriate control group for the reported symptoms. It may be useful to consider offering incentives for individuals to participate in this program.

R7. DOS should deploy broadband RF sensors to locations of interest. The sensors should be capable of detecting RF waves in the 0.5–30 GHz range, from continuous waves to nanosecond pulses, sensitive to average flux levels down to 20 mW/cm^2 and peak flux levels down to 1 W/cm^2, and with sufficient sampling rates to detect a broad range of pulse repetition rates from 1 to 10,000 pulses per second. A two-tier monitoring program could be employed, where some sensors sensitive to a subset of the frequencies are deployed more broadly and sensors sensitive to the whole frequency range are deployed at select locations. Sensors for continuous data recording should be deployed in various, randomly selected locations. In addition, sensors should be deployed speedily at any location where a documented incident has occurred.

R8. DOS should carry out experiments using RF on relevant and commonly used electronics to establish the frequency, power, and pulse duration ranges that might disrupt their operation and track specific models of electronic devices that might be disrupted by future events. The data from such experiments, together with the absence of disruptions during reported events can be used to set parameter limits on directed RF power that might be of concern.

R9. Given that fear and stress can cause or exacerbate real harm to DOS employees, DOS should consult with relevant experts concerning messaging, training, guidance, and other measures to reduce anxiety associated with the reported incidents. These could include building awareness of the very limited mechanisms capable of producing physical harm via energy delivery at a distance. DOS should recognize psychological phenomena as real afflictions that cause pain and treat those cases the same way as other types of injuries.

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2 INTRODUCTION

The possibility that US personnel have been subjected to intentional attacks leading to lasting adverse health effects is, appropriately, an ongoing source of concern to the US government. This issue impacts the individuals involved as well as the federal agencies that employ them, and has serious implications for US foreign policy and for the conduct of our diplomatic operations. This document presents the results of the second JASON study of this issue. The previous JASON report, JSR-18-017 "Acoustic Signals and Physiological Effects on U.S. Diplomats in Cuba," contains deliberations, findings, and recommendations for the situation that obtained in 2018. In the intervening time, a number of other studies (by e.g., the National Institutes of Health, the National Academy of Sciences, etc.) were conducted. Other investigations are being undertaken in parallel with this 2021 JASON study.

We were charged by the Department of State to:

- examine all available studies and documentation related to the constellation of symptoms commonly referred to as "Havana Syndrome".
- examine data and attempt to identify any stimuli that may have caused the effects and quantify how those stimuli could have been produced.
- analyze current monitoring protocols and make recommendations to enhance preventative measures where possible.

Further, we were asked to include three components in the study:

- Analysis: JASON will define a credibility threshold for provided technical hypotheses based on retrospective analysis of data, and then list which theories exceed said threshold. In the process, JASON will suggest new tests for ranking hypotheses.
- Sensing: JASON will recommend what monitoring should be carried out in the mid-term and in the long-term to validate or eliminate probable causes.

- Mitigations: JASON will suggest measures to mitigate probable causes.

Reports of incidents continue to accumulate, particularly after personnel were asked to come forward and report incidents they deemed unusual or that elicited concern. The challenge at this stage is to assess and categorize the candidate incidents, on the basis of available data (medical and physical) from both the reporting population and appropriate "control" groups, to facilitate an evaluation of various hypotheses. Reports need to be assessed against a range of hypotheses, including:

- Actions undertaken by one or more adversaries with the intent of inflicting physical harm through the transmission of harmful amounts of energy,
- Actions undertaken by one or more adversaries where physical harm is an unintended consequence,
- Actions undertaken by one or more adversaries where real harm, for example via the causation of functional disorders, arises as a consequence of an attack which does not cause identifiable physiological damage,
- Instances of medical conditions that are of "normal" origin, consistent with the rates seen in an appropriate control group,
- Instances where individuals respond to an environment of high tension and awareness, where otherwise unremarkable sensations are interpreted as initiating events or symptoms (potentially in conjunction with functional disorders),
- Cases where genuine and lasting adverse health effects are brought on by a climate of heightened apprehension and anxiety (psychogenic illness),
- Environmental sources or naturally occurring pathogens (viral or bacterial),
- An increase in reported events due to reinforcement on social media or other interactions, whether spontaneous or incited.

Given the range of phenomena and circumstances described in the reports that were shared with us, we consider it unlikely that a single explanation applies to all of the candidate

incidents. In addition, several mechanisms may be at play at the same time. For example, an adversary may take advantage of heightened tension and awareness and attempt to inflict real harm using methods which do not transmit harmful amounts of energy but do trigger sensory responses. The findings of this report contain JASON's assessment of the information currently on hand for the candidate incidents. The recommendations contain JASON suggestions on how to improve the scope and utility of information gathered for future candidate events.

Our approach entailed (1) reviewing the information obtained from interviews with individuals, (2) reviewing the diagnostic health data that were shared with us, and prior reports and meta-analyses of this medical information, (3) reviewing the known mechanisms by which standoff devices could impart lasting adverse health effects, and (4) a systematic evaluation of possible means for transmitting effective amounts of energy in ways that are consistent with the reporting and that could produce lasting adverse health effects. We also took into consideration other proposed, but not well-accepted by the health science community, mechanisms for inflicting harm from afar (such as the "grey literature" discussed in Section 6.5.5).

Our study focused on evaluating scenarios where damaging amounts of energy could be broadcast from tens of meters of separation, at levels that could produce direct physical harm, as well as on assessing plausible scenarios where temporary physical stimuli triggered from a distance could lead to short-term and long-term symptoms (e.g., through precipitating a functional disorder). The conclusions from this study are intended to allow the government to rule out attack mechanisms that cause direct physical harm and provide guidance on how to sense for signals that can cause harm directly and indirectly. We do not address viral and bacterial infections, the possibility of environmental toxins or contaminants, or food or water-borne vectors, as these were studied in detail in the 2018 JASON report and in the 2020 report from the National Academies of Sciences, Engineering, Medicine (National Academies of Sciences & Medicine, 2020).

We think it is informative to briefly review the history and evolution of what started off as the "Havana Syndrome" or, more formally, as Anomalous Health Incidents. The initial

reports of anomalous experiences were tightly linked at the time to loud audible sounds, some of which were recorded. The previous JASON study provided an in-depth analysis of these sounds and (we believe) identified a natural and possibly benign source for the noises. The personnel involved in those initial candidate incidents were subjected to a variety of medical evaluations, including neurological functional tests and medical imaging. At that time the medical evidence for cerebral anomalies was presented as clear and compelling, a finding that was later called into doubt by subsequent analyses of those data (Muth & Lewis, 2018; Della Sala & Cubelli, 2018) as well as by the NIH Traumatic Brain Injury Study (Shahim et al., 2021). Less clear at the time was whether the incidence of anomalies exceeded that to be expected from an age, gender, and life-experience–matched control group. It was, and remains, a challenge to crisply identify a set of signs and symptoms that would comprise a clear case definition. The paucity of physical data associated with candidate events posed a challenge to making a definitive association with causal mechanisms.

In the time between the 2018 JASON study and this writing (Aug 2021), the situation has evolved. The audible-acoustic association with candidate events has declined dramatically. The interpretation of the earlier medical diagnostic information has been called into question, and the geographical distribution of candidate events is much broader. Given the range of experiences that appear in the reports on the various candidate incidents, it is not surprising that the medical signs and symptoms, when they exist, are not entirely consistent between cases and are complicated by confounding factors. The statistical interpretation of the medical information is impeded by a lack of data from appropriate control groups of sufficient population size and by the extreme nonuniformity of the data collection. We comment on physical data collection in the Appendices of this report.

Another consideration to which we draw attention is the time that has elapsed since the initial reports, and the substantial press coverage that they continue to receive. Even if no intentional adversarial actions occurred early on, at this point we need to anticipate adversaries exploiting the current situation and perhaps taking actions to confuse, confound, and impede our resolving the issue. We have further comments on this in the Appendices as well.

This report begins with our assessment of the case reports that were provided to us, for the candidate incidents. We highlight the benefits of implementing the collection of structured data that is amendable to automated analysis, and the merits of taking prompt action (including the installation of in situ RF monitoring systems) when certain conditions are satisfied. We provide a summary appraisal of the existing medical data. We then evaluate all available data in the context of known mechanisms that can produce lasting adverse health effects, including through pathways that may trigger functional disorders, including ionizing radiation, sound (ranging from low frequency infrasound to inaudible ultrasound), and electromagnetic waves. We assess these possible intrusion mechanisms in the context of stand-off means of producing long-lasting health effects that are consistent with the medical data presented to date.

We are grateful to the many individuals who provided briefings, data, and background material for this study. A list of briefings delivered to JASON can be found in Appendix H. We also take this opportunity to extend our gratitude to the many US personnel who undertake the nation's business overseas.

2.1 Some Definitions and Terminology

We provide the following terms and their definitions as used throughout this report.

- **Organic Disease:** Any health condition in which there is an observable and measurable disease process, such as inflammation or tissue damage. An organic disease is one that can be validated and quantified through the standardized biological measures known as biomarkers.
- **Non-organic Disease:** Any health condition which manifests with symptoms but whose disease process is either unknown or unable to be measured by current scientific means.
- **Syndrome:** A recognizable complex of symptoms and physical findings that occur together and characterize a particular abnormality or condition, for which a direct cause may not be understood.

- **Anecdotal:** Consisting of, or based on, reports of individual cases rather than systematic research or analysis.
- **Traumatic Brain Injury:** An injury that affects how the brain works.
- **Functional Disorder:** A medical condition involving altered biological function without known structural change.
- **Psychogenic Illness:** A medical condition involving loss or alteration of function that does not have a precipitating physical cause.
- **Situational:** Relating to location and surroundings of a place.
- **Nociceptor:** A sensory receptor for painful stimuli. Nociception is the perception of a painful or injurious stimulus.
- **Etiology:** The cause, set of causes, or manner of causation of a disease or condition.
- **Afferent:** Conducting or conducted inward or toward something (for nerves, the central nervous system; for blood vessels, the organ supplied).

3 ANALYSIS OF INCIDENT AND SIGNALS DATA

3.1 Assessment of Case Data

JASON was provided with a set of incident data from personnel assigned to posts in US embassies and consulates who reported experiencing sensory and medical incidents over the course of the last four years. The dataset spanned multiple countries and was complete up to May 2021. We provide a general assessment of these data here and include some additional details in an appendix.

The data provided to us are in the form of spreadsheets that included various details about the incidents in multiple columns. They are all in narrative form, providing an account of the perceived incident, sensations experienced by the affected personnel, symptoms that developed immediately or after a period of time, the location of the incident, and other environmental details that may be pertinent to the experience (e.g., if it took place indoors or outdoors, whether other people were present and if they also experienced any sensations, whether the affected individual changed locations and if this alleviated the sensations). Each incident was self-reported by the affected personnel. A small subset (10-15%) of the reports were prompted by embassy-wide meetings or agency-wide requests that asked all personnel to report "anything unusual" experienced during assignments.

The level of detail and the content of the incident reports is highly heterogeneous. The files show that the incident reports have evolved significantly over time. Early data from Havana include fewer pieces of information, can be captured in a handful of columns, and do not appear to be responses to a uniform set of questions. Later reports include many more questions and responses and fill several tens of columns. We took this evident evolution in the incident files as a testament to the significant efforts by the Department of State and other government agencies towards gathering relevant data, identifying a cause, and providing a solution to these incidents. The level of detail captured also varies by location. In addition, because the reports contain answers in narrative form, the details provided are not consistent across reports, even when reports are guided by a uniform set of questions. Finally, the delay between the time of the incident and the time of the report varies from

hours (typically for more recent reports) to years (for some of the earlier reports.) In general, reports prompted by requests occurred after longer delays than unprompted ones. In our opinion, this range of delays introduces different levels of recall bias to different reports.

Analyzing the incidents as a whole, we find that the data show a multitude of differences across cases.

- The incidents described do not follow one single or clear pattern but show significant variation across events, sensations, and symptoms. The severity likewise spans a wide range: some affected individuals only report an unfamiliar sound, others communicate a multitude of sensations and near-term symptoms, including sensations of pressure and sound accompanied by vertigo, nausea, tinnitus, and/or headache. Less than 20% of cases appear to report all of the sensations and symptoms in CDC's current case definition.

- The nature of the incidents show significant evolution over time, from reports of loud, external sounds in earlier cases in Havana to a sensation of pressure or headache at the onset for later ones. Overall, they do not appear to describe the same phenomena.

- In later incidents, the majority of the affected personnel express awareness of the prior incidents in their reports and appear to verbalize a stress response to the possibility that they have been targeted.

- The reports are divided between descriptions of one-time, sudden events and those that continue for hours, days, or weeks, or occur repeatedly.

We emphasize that even among the subset of incidents with similarities (e.g., a report of a sound), the specifics described vary a great deal. In the case of sound, descriptions range from high pitched sounds of short duration to low-frequency bass beats to constant noises that resemble running water or rolling marbles. In some cases, the sounds last for days or more; in others, they represent a singular incident. These descriptions do not immediately point to a single origin for the sounds or a single underlying cause for the events.

All of these characteristics make it difficult to provide a quantitative assessment of the incidents in order to look for patterns and correlations. With that caveat in mind, we nevertheless attempt to quantify a few key features that will be important for connecting the events to an underlying cause (or causes).

In 85-90% of the incidents (all but 20-30 cases out of nearly 200), it was our judgement that the reported sensations could be explained by normal, benign, environmental or natural causes. These include sounds that seemed to be originating from appliances, noisy hallways or natural sounds; ear or head pressure accompanying reported colds; and pressure sensations that accompany the onset of headaches. Eliminating such incidents left 10-15% of incidents that evaded an immediate alternative explanation. **(Finding 1)**

The accounts of the remaining cases have similarities with one another and likely serve as the basis for the current case definition. This pattern of sensations and symptoms includes a sudden onset of a sound or a sensation of pressure, followed by a subset of the following symptoms: headache, vertigo, nausea, tinnitus, gait problems, confusion, or other cognitive problems. The large overlap between these non-specific symptoms and other common medical conditions makes it difficult to identify a link between these reports and a physical cause. Nevertheless, we identified a set of frequently reported characteristics and distilled these into "incident consistency criteria" we will use to evaluate various hypotheses. Below, we highlight these criteria and our reasoning for inclusion in our assessment.

Incident Consistency Criteria:

More than 90% of the cases were reported to take place indoors. At least 75% of the cases took place in personnel residences or hotels. The remainder took place either within embassy buildings or occurred in other indoor or outdoor public locations (the latter making up less than a total of 10 cases). This gives rise to the first evaluation criterion: in the majority of the cases, an external source must be able to **penetrate through the walls or windows of a building** and still be able to have adverse health effects on people.

In the majority of cases, personnel report being a significant distance away from other people or residences. This includes being inside an embassy building with controlled outside access, the interior room of a house, or in an outside facing room that is not adjacent to a neighbor. We assess from these descriptions that if harm is inflicted by an external energy source, that the source should be able to **operate from a distance of tens of meters from its target**, at least in a significant fraction of the cases.

More than 50% of cases explicitly refer to localization and/or directionality. Examples include affected personnel reporting that the sensations subside after changing positions in a room, other individual(s) in close proximity not reporting signs or symptoms, and the sound being described as coming from a particular direction or location. This leads us to our criterion that if an external source of energy is responsible for the events, it should be **focusable to an area of a few square meters**, at least in a subset of the incidents.

There were no reports of disruptions in the functionality of personal electronic devices. Moreover, any reported interruptions around the time of an incident could be traced to normal events such as electrical maintenance. As a result, we require that an external energy source used to cause harm to people **should not cause discernible interruption in electronic devices or communications**.

Future Data Collection:

Going forward, incident assessments could be facilitated with a questionnaire with multiple choice answers that aim to gather only pertinent data. The questions should be structured around factors described in Sections 5 and 6 and address specific physical sensations that are expected to accompany remote energy attacks. A quantifiable questionnaire can help reduce the level of heterogeneity present in the data and potentially illuminate patterns, if present. JASON provides an example of such a questionnaire in Appendix A. **(Recommendation 1)**

This questionnaire could be used for a uniform initial assessment and triage process both to guide rapid medical care as well as to trigger physical/environmental monitoring for any signals of interest (we discuss this further in Section 6). It would be useful to

assign numerical scores to responses to answers in the questionnaire, based on severity and relevance. A total numerical score can form the basis of the initial assessment and triage: certain sets of medical and surveillance actions can be taken when threshold conditions are met. **(Recommendation 2)**

Finally, it would be useful to develop classifiers to rank the relevance of incident reports in the context of hypothesis testing. For example, linear classifiers sort data into categories based on a linear combination of input features. Different hypotheses would form different categories based on the different expected set of sensations, symptoms, and situational information. **(Recommendation 3)**

3.2 Signals Data

JASON has been provided with documentation and briefings on a variety of environmental data that has been collected at incident locations. We discuss our assessment of these data in an appendix.

We briefly note here that

- Some recordings of the sounds have been made by personnel during the incidents they reported.
- Other sensors that are sensitive to various signals and hazards have been deployed to some locations.
- No significant detections have been reported from such sensors.
- Some sensors were active during incidents while others were used for post facto inspections.

  We factor in the recorded sounds and the negative result from sensors in our assessment of potential energy delivery mechanisms that may cause adverse health effects (Chapters 5 and 6.)

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4 MEDICAL DATA AND THEIR INTERPRETATION

The investigators at the National Institutes of Health (NIH) presented to JASON preliminary population-aggregated data of clinical test results for 65 individuals enrolled in an ongoing 5-year study on traumatic brain injury (TBI) of overseas U.S. personnel (Shahim et al., 2021). We base our analysis of the medical findings in this report on these data, noting that NIH may arrive at different conclusions at the end of their study with additional data. However, we first briefly review earlier medical studies performed on the affected personnel to identify underlying causes of their symptoms in order to provide some context for the variety and evolution of the medical tests and evaluations that were performed.

Because the initial incident reports described loud sounds accompanying the symptoms, the first evaluations for the early cohort from Havana were performed at the University of Miami, with a focus on audiological and otolaryngological tests (i.e., related to hearing, ear, nose, and throat). Between February and April 2017, the medical team in Miami evaluated 80 embassy staff and family members, of which 16 were judged to have clinical symptoms consistent with mild traumatic brain injury or concussion, although other causes including stress and underlying medical conditions were not ruled out (Baloh & Bartholomew, 2020). These 16 individuals, along with 8 others identified in the intervening time, were then evaluated at the University of Pennsylvania Center for Brain Injury and Repair. Out of the 24, 21 individuals completed the set of evaluations that included cognitive, vestibular, oculomotor, audiometric, and imaging tests. This study, published in the Journal of the American Medical Association (JAMA; Swanson et al. 2018), concluded that all 21 individuals exhibited acute and persistent symptoms consistent with concussions.

The JAMA study was later criticized for making claims that were not well supported by the data and for attempting to interpret the data without appropriate comparisons to control groups (see, e.g., Muth & Lewis 2018; Della Sala & Cubelli 2018). Subsequent analyses of these data concluded that most medical findings were within normal ranges and that there were no structural changes in the brains of the individuals. For example, follow-up studies argued that imaging revealed “nonspecific white matter changes” in only three individuals that could have resulted from a wide range of conditions, including aging

and depression. In addition, these critiques pointed out that many of the symptoms overlap with numerous other medical conditions and did not provide clear evidence for brain injury.

In our view, these examples serve to illustrate some obvious but important aspects of these medical evaluations. First, it is important to enroll appropriate control groups. Because the non-specific symptoms reported by the affected personnel can result from a range of normal situations and medical conditions, defining appropriate age-, sex-, and lifestyle-matched control groups and enrolling statistically relevant numbers of individuals in the studies is crucial for obtaining robust conclusions. Second, administering many successive independent tests to the affected individuals, without a clear hypothesis, increases the risk of encountering spurious correlations (we revisit this in section 4.1.3). Therefore, JASON advises formulating specific hypotheses – such as expected physical damage associated with particular attack mechanisms – and performing tests specifically designed to look for those effects (as opposed to a broad range of more general medical tests).

4.1 NIH Traumatic Brain Injury Study

JASON reviewed preliminary aggregated medical data from the NIH's ongoing 5-year study on TBI of overseas U.S. personnel, which began in May 2018. At the time of this review (August 2021), the NIH investigators presented JASON with population-aggregated data of clinical test results for 65 cases: 26 from Cuba, 6 from China, 9 from Vienna, and 24 from other locations. As of June 2021, this sample represents about 0.6% of an estimated 11,000 overseas non-military personnel who are candidates for conjectured harm.[1]

The NIH study evaluated patients at different stages in their clinical progression, ranging from days to years after the initial onset of symptoms. The selection of patients for inclusion in the study was subject to several filters, including self-reporting of a perceived anomalous health incident, triage based on credible medical findings that led to an NIH referral, a willingness to participate in the study, and a determination by NIH investigators that there was no obvious alternative cause for those findings. Each of these selection criteria

[1]There are ˜9,000 State Department employees overseas. The study sponsor estimates that ˜20% of non-military overseas employees are from agencies other than the State Department, giving a combined total of ˜11,000 non-military overseas U.S. personnel.

has a subjective component and shaped the selected cohort in ways that may influence the prevalence of specific medical findings, potentially making it more difficult to identify a causal mechanism underlying the reported incidents.

NIH enlisted a variety of control groups in an attempt to reach different comparison populations. Foreign service members posted in Cuba during the first set of events (n=12) were the most lifestyle-matched, followed by foreign service members who did not serve in Cuba (n=15). Because enrolling active-duty embassy personnel in a long-term medical study poses significant logistical challenges, these control groups are post-duty and thus understandably small. NIH also enrolled other, larger control populations in their study to increase the size of the control groups, albeit not lifestyle-matched, consisting of local NIH volunteers (n=40) and TBI patients in a different longitudinal study (n=109). These choices necessarily resulted in heterogeneous control groups. NIH readily acknowledges this and other limitations in their study, such as the prior treatment some affected personnel received, recall bias, enrollment delays, and confounding illnesses. Going forward, it would be beneficial to use the uniform triage process to select affected individuals for inclusion in future medical studies, to carry out a specific series of tests all done in the same order and within a specified time frame, and to put even more effort into assembling larger and appropriately selected control groups. **(Recommendation 4)**

Based on the aggregated medical data provided, JASON does not find any evidence of a novel medical syndrome that would go beyond the expectation of intercurrent illnesses in the general population. **(Finding 3)** In the future, the researchers may be able to take advantage of the individual incident reports and patient medical records to design improved selection criteria that provide evidence for such a novel syndrome, for example, based on the detection of rare clusters of symptoms not normally found to occur in the general population. **(Recommendation 5)**

Reviewing the entire cohort of affected individuals, JASON finds no strong evidence of TBI in the data obtained. JASON was not given access to the results of the patients' MRI studies, either conventional or diffusion tensor, but was told that up to that date, the studies showed no abnormal findings that would support the hypothesis of TBI. Data on

two serum biomarkers, neurofilament light chain (NF-L) and glial fibrillary acidic protein (GFAP), showed mean elevated levels in the affected individuals, but these values were not significantly different from those of the tested control individuals. There are (at least) two credible explanations: either there was no adverse exposure causing TBI, or the signal of TBI is drowned out by a significant background of unharmed individuals.

This situation emphasizes the importance of how the selection criteria are applied in defining the "treatment" group. We further note that the measured serum biomarkers are not unique to TBI. In particular, elevated NF-L could result from damage to peripheral nerve axons as a consequence of various causes, including inflammatory or demyelinating neuropathies, autoimmune axonopathies, chemotherapy-induced toxicity, and crush injury (Mariotto et al., 2018; Meregalli et al., 2020). Because peripheral nerve damage is a component of some of the hypothesized mechanisms of harm described in this report, further analysis should be done to differentiate between central and peripheral sources of elevated serum NF-L. We raise this concern because the clinical focus on TBI may be misdirected. As explained in section 6.5, it is very difficult to deliver harmful quantities of energy to the central nervous system at a distance, making TBI implausible.

Just before completion of the JASON study, the NIH provided us with aggregated medical data for a subgroup of 6 individuals from their study, together with 12 control cases selected by the Department of State that were matched for age and gender. The 6 members of the subgroup were selected from a larger group of 29 individuals that JASON identified as having had similar anomalous experiences, based on our review of the nearly 200 case reports and without reference to the medical data. The medical data were provided for only 6 of the 29 requested cases because only those individuals had given prior consent for data sharing. JASON has asked that efforts be made to obtain consent from the other 23 identified cases.

Given the small number of cases in the subgroup, it is difficult to draw firm conclusions. In summary, however, there were no significant differences for any of the measures of vestibular function in the subgroup compared to the control group (Shahim et al., 2021). These studies included both cervical and optical vestibular myogenic evoked potentials,

which are objective measures of neuronal function of the saccule and utricle, respectively (the two otolith organs of the inner ear). There also were no significant differences between the two groups with regard to rotational measures (gain, phase, and symmetry), and to more subjective tests of oculomotor function, including: optokinetic nystagmus response, smooth pursuit, vergence pursuit, fixation and spontaneous square waves jerks, horizontal saccades, anti-saccades, self-paced saccades, predictive saccades, motor reaction saccades, audio-visual reaction times, and pupillary reflex.

There were statistically significant differences between the subgroup and controls with regard to some aspects of their general neurological exam, with the selected subgroup of 6 individuals exhibiting higher depression scores and higher scores on both an inventory of medical symptoms and a general health questionnaire. With regard to serum biomarkers, the mean level of both NF-L and GFAP was higher in the subgroup compared to the controls, although not reaching overall statistical significance. This is because there was much more scatter in the subgroup, including one outlier who scored markedly above the mean on both measures.

4.1.1 Hypothesis-testing on combined datasets

By combining the medical data with the incident reports pertaining to circumstances at the time of onset of symptoms, there is the potential to improve significantly the assessment of alternative causal hypotheses. A selection criterion recommended by the Centers for Disease Control and Prevention, which is already in use by some agencies (although perhaps not applied uniformly) requires a biphasic onset of symptoms. Specifically, the CDC's criteria are:

1. An initial phase that includes at least one of the following:
    - Head pressure
    - Disorientation
    - Nausea
    - Headache

- Vestibular disturbances
- Auditory symptoms
- Vision changes

2. A secondary phase that includes at least one of the following, with no alternative explanation:
   - Vestibular disturbances
   - Cognitive deficits

These criteria, subsets of these criteria, and extrapolations involving additional situational data could be used to test alternative hypotheses. The NIH study found evidence of vestibular dysfunction among a significant fraction of the study cohort.[2] However, incidents of vestibular dysfunction are very common in the general population, affecting 15–20% of adults on an annual basis, with vestibular vertigo accounting for about a quarter of these cases (Neuhauser, 2016). Thus the finding of vestibular dysfunction alone could result in individuals meeting the study selection criteria who have a condition that might be better attributed to other causes, such as benign paroxysmal positional vertigo, vestibular neuritis, or vestibular migraine, thus diluting the ability to identify a novel syndrome. However, if paired with other selection criteria, for example, auditory symptoms or a sensation of ear pressure, the selection process might identify a cohort of individuals who have a similar underlying cause for their symptoms. **(Recommendation 5)**

4.1.2 Example of vestibular migraine with auditory symptoms

To illustrate the usefulness of applying more refined study selection criteria, consider vestibular migraine as a potential explanation for the symptoms experienced by the affected individuals. Vestibular migraine is a recurring type of migraine that produces dizziness, nausea, and vestibular disturbances (Lempert et al., 2012). Clinical studies have shown that the risk of cognitive dysfunction is increased in vestibular migraine (Rizk et al., 2020). It is notable

[2] Based on abnormal test scores for self-paced saccades, motor reaction saccades, and audio-visual reaction times, and an attenuated amplitude of vestibular evoked myogenic potentials.

that about half of vestibular migraine patients report abnormal sounds and/or a sensation of ear pressure. This condition would be a potential explanation for the biphasic pattern of sound or pressure, followed by vestibular disturbances, in the reported health incidents. One can then ask, what is the probability that the selection criteria have simply picked out naturally-occurring cases of vestibular migraine with auditory symptoms within the population of overseas U.S. personnel? Could this affliction account for the approximately 25 affected personnel who, in our estimation, are showing the broader and more severe range of symptoms that evade an immediate alternate explanation based on the incidents?

The lifetime prevalence of all types of vestibular migraine is estimated to be 0.01 (95% CI, 0.0070–0.0137) and of those, the fraction producing auditory symptoms or ear pressure is estimated to be 0.45 ± 0.16 (Neff et al., 2012). The onset of vestibular migraine can occur at any age, with a mean age of onset around 40 years (37 years for females and 42 years for males; Hilton & Shermetaro Hilton & Shermetaro). Migraines typically predate the onset of vertiginous symptoms by an average of 8 years and are more common among women by a ratio of approximately 5:1.

One might suspect that a vestibular migraine subject, having not yet experienced such a migraine, would not understand what is happening to them and report their first episode as a health incident. Age-stratified data for first onset are not available in the literature, but we can still make a useful estimate of the probability of this scenario under the assumption that the onset is uniformly distributed between ages 20 and 50. If the lifetime prevalence of any vestibular migraine is 0.01, then the probability of developing the migraine at any given year between the ages of 20 and 50 would be 0.01/30=0.00032. About half of those migraines will have an auditory component, roughly consistent with the prevalence of sound in the reports. For the entire population of $n = 11,000$ non-military overseas U.S. personnel, we then expect 3.5 such events per year. While this appears insufficient to explain all of the approximately 200 reported events, it could be plausible for the selected group of approximately 25 cases since 2016, for which the rate would be $25/5 = 5$/year.

This is a significant number that, on the surface, could make up a large fraction of the health incidents. However, the above calculation does not account for the clustering of events in geographical location (several appearing in one post) and in time (several appearing within a year instead of over several years). For a 100-person post (such as Havana), the expected number of first-time vestibular migraines would instead be $0.00032 \times 100 = 0.03$/year, which could not explain the appearance of several similar health incidents within one year.

We can ask a similar question using different assumptions about human behavior. Instead of requiring that vestibular migraine be a first-time event, we can relax that requirement and consider that the fear of an attack by an adversary may cause any migraine subject to report such a migraine as a health incident, even if they had experienced one before. In this case, we need to know that the 1-year prevalence of vestibular migraine is 0.009 (95% CI, 0.0062–0.0127), because those having vestibular migraines tend to experience episodes about once a year (Neuhauser, 2016). The expected number of vestibular migraines for the total embassy population is then $0.009 \times 11,000 = 99$/year, whereas for a single 100-person post would be $0.009 \times 100 \approx 1$/year.

From the foregoing, we can conclude that the occurrence of vestibular migraine in the general population does not, by itself, explain the cluster of symptoms reported by U.S. overseas personnel. One must carry out a similar analysis for other potential explanations for various constellations of signs and symptoms among the affected individuals to determine if their occurrence deviates significantly from what might be expected for the age-matched general population.

4.1.3 The look-elsewhere effect

It is important to keep in mind that there may be several different clinical conditions that underlie the reported symptoms, each of which is far too uncommon to provide a unifying explanation, but that collectively account for a significant fraction of the cases. For any given uncommon condition, one might conclude from the small probability of occurrence that it cannot possibly explain the observed cluster of reported symptoms. However, such

conclusions fail to take into account what is known as the "look-elsewhere effect" (Lyons, 2008), also known in statistics as the multiple comparisons problem or the multiple hypothesis testing problem. The look-elsewhere effect refers to the overestimation of significance of an observation due to failure to search the entire relevant space of parameters. Even if the probability of occurrence of a particular medical condition among the U.S. overseas population is very small, the probability of a few people simultaneously experiencing symptoms due to *some* type of unusual condition may not be rare at all.

As discussed in section 4.1.1, most of the symptoms experienced by the affected personnel are very common among the general population across many illnesses. However, the notion that these symptoms may have a more sinister origin can be socially propagated, especially in a stressful environment. Hence, a very small cluster of individuals who experience these symptoms due to an uncommon medical condition could form the seed of a socially-amplified cluster of patients who report similar symptoms. Thus, to determine the probability of a true syndromic cluster occurring at a particular post, one must integrate over random coincidences of all possible conditions, however rare, that might provide the seed for social propagation of similar reported symptoms.

With regard to the many reported anomalous health incidents, it is difficult to determine the appropriate search space that must be considered when attempting to carry out a quantitative evaluation of the look-elsewhere effect. It should nevertheless be understood that the probability of occurrence of an odd symptom cluster of *some* kind greatly exceeds the probability computed for the incidence of a cluster of any specific illness that has been identified after the fact. An alternative possibility is that there are one or more common conditions that explain the majority of cases, together with a collection of uncommon conditions that account for the remainder.

4.2 Differentiating Organic, Functional, and Psychogenic Conditions

It is important to consider and evaluate multiple types and causes of illness with regard to the anomalous health incidents. In our evaluation, JASON considered the potential role of the full range of illnesses including organic disease, functional disorders, and psychogenic

illness. Organic diseases involve structural, biological changes. Functional disorders have physical origins, but involve altered biological function without known structural changes. Psychogenic conditions do not have a precipitating physical cause. It is helpful to identify the contributions of these different components in an attempt to differentiate between the following possibilities:

- Direct physical and physiological harm caused by energy delivered as a result of a physical attack;
- Functional disorders that arise as a consequence of physical attack;
- Functional disorders that have other precipitating events, including inadvertent physical insults, medical conditions unrelated to any external insult, and events that can be considered ordinary but have an amplified effect due to prevailing circumstances;
- Psychogenic disorders that do not have a precipitating physical cause.

All medical conditions have a real, psychological component. Despite this, functional disorders and psychogenic illness have different etiologies. In persistent postural-perceptual dizziness (PPPD) and other functional disorders, there is a strong psychological component, but this is not meant to imply that these individuals have psychopathological abnormalities. Rather, the psychological (and accompanying psychosocial) factors should be regarded as amplifiers that cause an acute precipitating event to evolve into a functional disorder. Such a dynamic process is thought to underlie PPPD (Figure 4.1). Individuals who by nature or circumstances are in a state of anxiety and heightened body vigilance are more likely to convert an acute precipitating event to a functional condition (Staab & Ruckenstein, 2007). This is because such individuals have heightened sympathetic activation, which provides a physiologic basis for their stress and anxiety. We note that such a condition could be triggered by adversarial action without the use of attacks that produce direct physical harm.

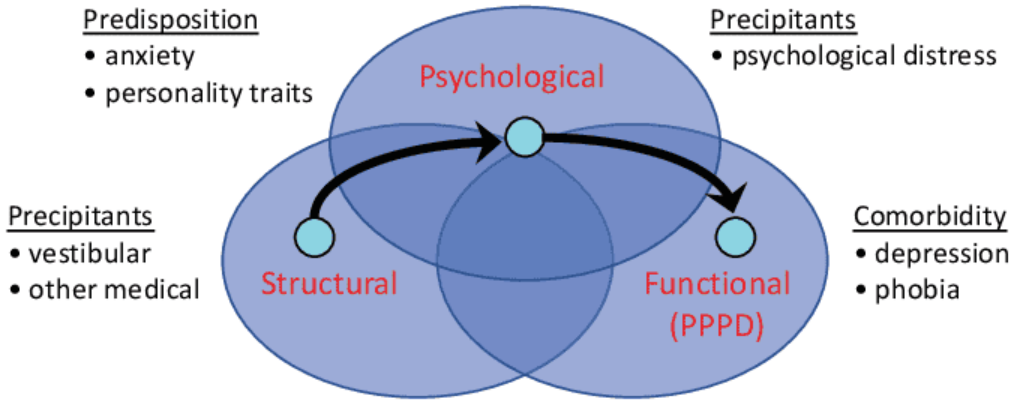

Figure 4.1: Putative mechanism of PPPD, involving a dynamic process that begins with a precipitating structural condition that is amplified by psychological factors to result in a functional disorder (based on Dieterich et al. (2016).)

4.2.1 Functional Disorders: Example of persistent postural-perceptual dizziness (PPPD)

One of the most common vestibular disorders is persistent postural-perceptual dizziness (PPPD), which occurs in 15–20% of patients who present for evaluation of vestibular symptoms (Dieterich et al., 2016; Staab et al., 2017). It is difficult to estimate the prevalence of PPPD in the general population, but the annual incidence of dizziness (including vertigo) in the U.S. adult population is 15–20%, with vestibular vertigo accounting for more than one-third of these cases (Neuhauser, 2016). This disorder is characterized by symptoms of dizziness, unsteadiness, and non-spinning vertigo that are exacerbated by motion or exposure to complex or moving visual patterns (Staab et al., 2017). Patients often describe a feeling of fuzziness or lightness in the head, disorientation, or lack of visual focus. PPPD is triggered by a variety of conditions that cause dizziness, unsteadiness, or problems with balance, include acute or episodic vestibular syndromes, especially when coupled with psychological distress. The symptoms of PPPD may fluctuate, but are present on most days and can persist for months or longer.

Several features of PPPD match the secondary phase of the CDC criteria for Anomalous Health Incidents, but this disorder does not explain the triggering condition(s) that would have given rise to symptoms in the initial phase of the syndrome. One of the precip-

itating causes of PPPD is vestibular migraine, but PPPD occurs more commonly following other acute conditions, such as benign paroxysmal positional vertigo or vestibular neuritis, and in some patients the precipitating cause is never identified. Given the common occurrence of these precipitating conditions and the high prevalence of PPPD in the general population, it would be difficult to rule out this condition as an explanation for the secondary phase of symptoms among at least some of the affected U.S. overseas personnel.

PPPD is regarded as a chronic functional disorder, which refers to altered biological function in the absence of known structural changes at the level of cells or tissues. "Functional" is not a synonym for "psychogenic" or "psychosomatic", but rather indicates a pathophysiological state that has no discernable molecular or anatomical correlate. With the progression of medical knowledge, some disorders that previously were described as functional have come to be recognized as having a structural basis (e.g., fibromyalgia). Other disorders may have a strong functional component that overlays an organic component (e.g., chronic fatigue syndrome). PPPD is considered to be a pure functional disorder. It is not a diagnosis that is made following the exclusion of all others, but it does require one to rule out other disorders that might better account for the symptoms, and does not preclude the existence of an underlying trigger that has a more clearly defined pathophysiological basis (Staab et al., 2017).

There is a subset of affected U.S. overseas personnel who might be described as having PPPD, a condition that would be expected to occur in a few percent of the overall population, irrespective of any link to some postulated "attack". Clearly, not all of the affected individuals meet the diagnostic criteria for PPPD as being the cause of the secondary phase of their condition. Yet, it is important to recognize that individuals who are under stressful conditions are especially sensitive to environmental sensory triggers, which can help to elicit PPPD and other functional disorders. For these individuals, an initial noxious stimulus may instigate a feedback loop that involves activation of the stress response pathways, which in turn heighten anxiety and sensitivity to external stimuli. In addition, persons with certain underlying medical conditions, especially those that might give rise to the initial phase of the reported symptoms, are more susceptible to exacerbation of those conditions during times of stress. (Finding 4)

4.2.2 Psychogenic illness

Distinct from a functional disorder, there also are psychogenic conditions in which the medical symptoms do not derive from a precipitating structural event. Psychogenic illness may occur at the level of an individual, a local cluster of individuals, or within a broader population. It has been suggested, based solely on publicly accessible information, that the embassy health incidents are the result of a mass psychogenic illness (Baloh & Bartholomew, 2020). JASON reviewed the literature on psychogenic illnesses, which have been reported to occur in diverse settings and with diverse associated symptoms. It is difficult to make quantitative estimates of the prevalence of psychogenic illness, but attempts to do so suggest that the phenomenon is not rare (Page et al., 2010). One study, for example, estimated the prevalence of psychogenic, non-epileptic seizures in the general population to be between 1/50,000 and 1/3,000 (Benbadis & Hauser, 2000).

Psychogenic illness occurs when a patient's psychological concern about a perceived physical threat or condition produces actual bodily symptoms. There is broad agreement in the medical literature on the following:

(i) The experience of physical symptoms in patients with psychogenic illness is real, not faked. The illness can entail objectively observable physiological changes (Jones, 2000; Bartholomew, 2000).

(ii) Psychogenic illness can occur in any healthy population, without underlying psychiatric conditions (Broderick et al., 2011; Sapkota et al., 2020).

(iii) Some stress situations increase the risk of psychogenic illness (Weir, 2005), and the symptoms may be specific to fears and preoccupations of a given culture (Bartholomew, 2000).

Statements (i) and (ii) are not well understood by the general public or even by many medical professionals, resulting in a stigma being associated with psychogenic illness. In the face of such stigma, a somatogenic explanation may seem preferable over a psychogenic one, even if the latter is better supported by the available data.

A psychogenic etiology is a hypothesis to either confirm or rule out based on empirical data, whether in an individual or a group experiencing similar symptoms. The diagnosis of psychogenic illness is obtained by exclusion, when no satisfactory alternative structural or functional explanation can be found, especially when there is a pre-existing social connection among the affected individuals and there are more cases than would be expected for the corresponding population in the same period of time. Indicators of a psychogenic illness include:

- Attribution of symptoms by the affected individuals to a threatening external agent;
- Symptoms that are incompatible with what the individuals believe to be the causal event;
- Lack of a plausible connection between what is believed to be the causal agent and the circumstances under which exposure to such an agent might reasonably occur (Page et al., 2010).

The medical literature tends to focus on local, temporal clusters of *mass* psychogenic illness. However, there is also the possibility of more isolated or sporadic cases of psychogenic illness that are distributed across a broader population, especially if there is some form of shared knowledge among the affected individuals. In particular, social media has become a vehicle for amplifying the connection between an individual experience and a collective syndrome of psychogenic illness (Bartholomew et al., 2012). Furthermore, somatic and psychogenic illness are not mutually exclusive, and could give rise to the same constellation of symptoms, amplified at the level of the individual through psychological factors and at the level of a group of individuals through both psychological and psychosocial factors. One implication of a hybrid model is that a plausible physical hypothesis need not account for every symptom experienced across the set of affected individuals. Rather, competing explanations for the reported symptoms may include physical causes, functional syndromes derived from physical causes, and purely psychogenic effects.

Given the complexity and diversity of symptoms experienced by the affected U.S. personnel over the past 5 years, as well as the diverse circumstances associated with the

onset of those symptoms, it would be unreasonable to expect all cases to have a common etiology. The CDC's diagnostic criteria, which emphasize the biphasic nature of symptoms, may help to stratify patients who have experienced an acute adverse event from those who developed a chronic disorder as a consequence of such events. In some cases, the chronic symptoms of vestibular disturbances and cognitive deficits may derive from the acute event, in other cases these late symptoms may reflect the conversion of a structural disorder to a functional disorder, and in still other cases the syndrome may be psychogenic without any adverse triggering event. For the latter group, it is important to emphasize that psychogenic illness is common and does not imply that the affected individuals have a psychiatric disorder. These individuals should be reassured that their symptoms are real, even though they may derive from acute stress response or misinformation. It also is important to remind these individuals that information they may have heard regarding "suspected" causes is not equivalent to a validated explanation for the reported cases (Jones, 2000).

We end this section by noting that in *all* conditions, whether organic, functional, or psychogenic disorder, anxiety can worsen and prolong the symptoms in affected individuals. Some level of stress and anxiety is evident from the incident reports that describe sensations and symptoms when the affected personnel experience an event. Inconsistent, uncommunicative, or delayed responses in the aftermath of the incident can significantly worsen the situation. The U.S. Government could aim to reduce such stress by a variety of means including messaging, education, and rapid follow-up health and environmental monitoring. It could also help ease anxiety to use the rapid questionnaire-based triage process to let the affected personnel know if and when their incidents or health symptoms are not consistent with AHI, but still refer them as quickly as possible to appropriate medical care for whatever other condition they do have. It would be valuable to build awareness that mechanisms that can deliver energy at a distance and cause brain injury, without accompanying damage and sensations on other parts of the body, are extremely limited and to reassure overseas personnel that there is ongoing monitoring for signals associated with any such mechanism. **(Finding 4, Recommendation 9)**

4.3 Potential Underlying Causes

Whether both phases of symptoms derive from a structural cause or only the initial phase does and then gives rise to a functional disorder, it is important to consider potential mechanisms that might underlie the initial phase. There are several medical conditions that can give rise to a syndrome of vestibular disturbances with auditory symptoms, variably accompanied by headache, head pressure, nausea, visual disturbances, or disorientation. These conditions include: benign paroxysmal positional vertigo, vestibular neuritis, labyrinthitis, vestibular migraine, mal de débarquement syndrome, and Méniére's disease.

There also are conditions of "central vertigo" that derive from pathological brain processes, such as stroke, cerebral tumor, or multiple sclerosis (Vanni et al., 2017). In addition, there are many conditions that can cause "dizziness", which lacks the sensation of self-motion of true vertigo, but may also be associated with headache, nausea, lightheadedness, and disorientation, symptoms that have commonly been reported by the affected U.S. personnel. Dizziness can result from many causes, including anemia, hypoglycemia, electrolyte imbalance, cardiac arrythmia, and orthostatic hypotension, all of which are common medical conditions. Collectively, the conditions that give rise to peripheral vertigo, central vertigo, and dizziness could explain the initial phase of symptoms in many, if not most, of the affected personnel. Clearly, however, no single condition can explain all of the reported cases. Conversely, there may be a subset of cases that cannot be explained by any of the currently recognized medical conditions.

In principle, there are several afferent neural pathways that could result in acute vestibular disturbances that may be accompanied by disruption of auditory, oculomotor, cerebellar, or cognitive function. In considering the possibility of extrinsic causes, we will focus on pathways that might be activated by external stimuli, excluding injury due to trauma or direct physical contact. We also exclude obvious external stimuli that do not match the case reports, such as damaging sounds, intense IR, visible, or UV light, or exposure to ionizing radiation. Within the electromagnetic spectrum, frequencies above 10–20 GHz cannot penetrate human skin by more than 1 mm (see section 6.3.1), and thus could not affect afferent nerves that lie below the dermal layer, which has a thickness of 1–4

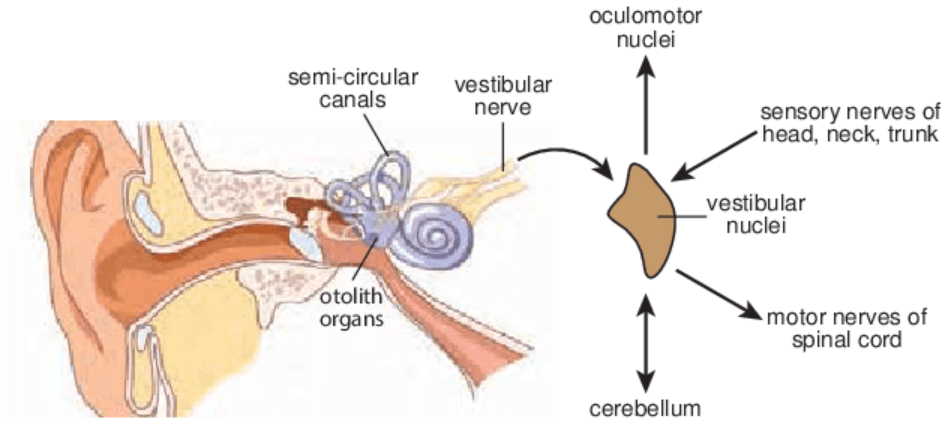

Figure 4.2: Neural pathways of the vestibular system. Sensory input from the otolith organs (saccule and utricle) and the semi-circular canals is carried by the vestibular nerve to the vestibular nuclei, where it is integrated with positional sensory input from the body, coordinated by the cerebellum. Output from the vestibular nuclei to the cerebellum, motor nerves of the spinal cord, and oculomotor nuclei of the brainstem help to control balance, posture, and eye movement, respectively.

mm. Frequencies below 1–2 GHz would require a very large emitter and could not be tightly focused to provide the type of localized effects that have been reported (see section 6.4). However, frequencies in the range of 1–20 GHz might conceivably stimulate or modulate the firing of peripheral nerves, which in turn might excite central pathways that result in acute vestibular disturbances with auditory symptoms.

The vestibulocochlear nerve (cranial nerve VIII), mediates sensations of movement (vestibular portion) and sound (cochlear portion). The vestibular nerve carries sensory information to the vestibular nuclei of the brainstem, with most of that input coming from the otolith organs and semicircular canals of the inner ear, which sense linear and angular acceleration, respectively (Figure 4.2). The vestibular nuclei also receive input from the cerebellum, which helps to coordinate movement and maintain balance, and from sensory pathways in the head, neck, and trunk that provide information about spatial positioning. The primary outputs from the vestibular nuclei are to the cerebellum (feedback circuits for movement and balance), motor pathways of the spinal cord (control circuits for postural equilibrium), and oculomotor nuclei of the brainstem (control circuits for eye movement).

There are pathways that link stimuli near the skin directly or indirectly to the vestibular circuits. For example, the trigeminal nerve (cranial nerve V) has sensory branches originating in the upper (ophthalmic) portion of the face that project to the vestibular organs. Stimulation of these trigeminal branches can result in increased blood flow to the vestibular organs, with both short-term effects on vestibular function and potential longer-term effects of vestibular hypersensitivity. Vestibular migraine, for example, is thought to be mediated through these pathways (Baloh, 1997; Vass et al., 2001).

Direct electrical stimulation of the skin of the forehead has been shown to elicit spontaneous nystagmus (repetitive involuntary eye movements), especially in patients who have a history of migraine (Marano et al., 2005). In contrast, no nystagmus is seen following stimulation of other nerves, such as the medial nerve at the wrist. The character of the nystagmus indicates that the effect of trigeminal nerve stimulation is stimulatory to the vestibular organs on the same side of the body. Of course, direct electrical stimulation of the trigeminal nerve is not being considered with regard to the affected U.S. personnel. However, it has also been shown that nystagmus can be elicited by gentle vibratory stimulation to the forehead (Sheth et al., 1995), suggesting that there may be more indirect means of stimulating the trigeminal nerve that give rise to vestibular disturbances.

Another afferent pathway that links to the vestibular circuits involves sensory inputs carried by the vagus nerve (cranial nerve X), with some of those inputs originating within a centimeter of the skin surface. Apropos of its name, which means “wandering”, the vagus carries sensory input from many locations of the body, including the skin, heart, respiratory tract, digestive tract, and other internal organs. It also controls motor function of the mouth and throat, as well as involuntary (parasympathetic autonomic) function of the heart and digestive tract. Of particular interest here is the input the vagus receives from the skin behind the ear, the external portion of the ear canal, and the meningeal surface of the brain. The vagus can also be stimulated indirectly by massaging the carotid sinus (located just below the jawline), splashing cold water on the face (the dive reflex), or applying modest pressure to the orbits of the eyes (the oculocardiac reflex) (Ceylan et al., 2019). These maneuvers are sometimes used to slow abnormal heart rhythms, and non-invasive stimulation of the vagus nerve has been used to treat both PPPD and vestibular

migraine (Eren et al., 2018; Beh & Friedman, 2019). As with the trigeminal system, surficial stimulation of the vagus nerve might alter vestibular function, either increasing or decreasing the symptoms of vertigo. For the vagus, the situation is more complicated because its stimulation also results in reduced heart rate and blood pressure, which can cause symptoms of dizziness and lightheadedness that are often reported as vertigo.

The above discussion notwithstanding, it remains difficult to link a plausible mode of external nerve stimulation to the constellation of symptoms seen in the initial phase of the syndrome experienced by affected U.S. overseas personnel. Stimulation by electromagnetic radiation sufficient to cause structural damage would require delivering such a large amount of energy that it would be accompanied by a sensation of heat and/or pain. It is possible, as discussed above, that many of the affected individuals are experiencing dizziness rather than true vertigo during the initial phase of symptoms. It would be easier to bring about dizziness, rather than vertigo, through external stimuli. Finally, there may be some unknown neural pathway that links external stimuli to the reported symptoms that lies beyond the current body of medical knowledge. Human neurophysiology is remarkably complicated and is intertwined with other physiological, cognitive, and emotional factors, compounded by the variability of the individual human experience.

4.4 Clinical Context of the Affected Individuals

The variability of human experience may provide an opportunity to identify causal relationships between an individual's reported symptoms and an underlying medical condition. As noted in the earlier subsections, there are many potential causes of symptoms of headache, nausea, dizziness/vertigo, lightheadedness, disorientation, strange sounds, and visual disturbances. An individual who reports these symptoms may be given medication to try to alleviate them, or may already be taking medication for a different condition. In either case, the response to those medications could shed light on the underlying cause of symptoms that were associated with an anomalous health incident.

For example, individuals who suffer migraines, which have a prevalence of approximately 12% in the general population (Yeh et al., 2018), may be treated with analgesics,

ergotamines, triptans, beta blockers, tricyclic antidepressants, or calcitonin gene-related peptide (CGRP) antagonists. In addition to their potential benefit for migraine, these medications have relevance to some of the causal hypotheses discussed above. A beta blocker decreases sympathetic tone and may reduce the anxiety that contributes to a functional disorder such as PPPD. Conversely, ergotamine increases sympathetic tone and can increase anxiety, at least in the short term. A CGRP antagonist directly affects the physiological pathways that underlie vestibular migraine.

A similar case can be made for many other medications, including: gabapentin, which is used to treat neuropathic pain, but can precipitate depression and other mood disorders; beta adrenergic agonists, which are used to treat asthma, but increase sympathetic tone and can increase anxiety; benzodiazepines, which reduce anxiety and therefore may benefit someone with a functional disorder; and carbamazepine, an anti-convulsant that also modulates conduction in the trigeminal nerve. This list goes on.

Every individual has a story to tell and in effect constitutes an "n-of-1" clinical trial that relates their medication history to their past and present medical condition. It appears highly unlikely that all of the nearly 200 reported cases of anomalous health incidents derive from a single, or even a small number, of medical conditions. By individuating rather than aggregating the data associated with those cases, it may be possible to identify subgroups that meet the diagnostic criteria for a known medical condition. Collectively, these subgroups may account for the majority of cases, without the need to invoke a new syndrome of unknown etiology.

To aid with future efforts in this direction, JASON recommends adapting the baseline medical program to capture the full health history of each individual prior to their assignments to posts around the globe. Such evaluations may be beneficial in uncovering the particular medical conditions that either already affect the individual or that may play a role in determining their medical response to environmental factors. It would also provide a more rigorous basis for evaluating whether particular medical conditions can account for some or all of the reported health incidents. **(Recommendation 6)**

4.5 Understanding and Managing the Role of Social Interactions

The role of social interaction in reinforcing and exacerbating some of the conditions described above cannot be overstated, especially in an age of social media and ubiquitous connectivity. Society's understanding of social dynamics in the context of social networks continues to evolve. This topic has a voluminous literature that is well outside the scope of this report. However, the study of the social dynamics and best leadership practices of groups of people operating with a common mission in stressful situations has a history that long predates social media, especially in the U.S. Navy (Allingham, 1964; McGlynn, 2005; Roos, 1943). We cite two illustrative examples:

- A 1956 study mapped and examined the social networks aboard a Navy ship and their role in the spread of rumors aboard ship. Examining the failure of artificially seeded rumors to take hold during the experiments, the author concluded: "Here, it now seems, lies the only suitable explanation of the test rumor's limited 'success'... something in the makeup of the community basically prevented its taking hold... Of major importance would seem the active and consistent program of information and education pursued by the commanding officer. The captain meticulously informs the crew of all matters, which affect their personal planning, as they arise." (Allingham, 1964)
- A more recent dissertation in 2005 looked at best practices when decommissioning Navy ships and found the existence of rumors and the effect of their spread to be one of the major themes presented as leadership challenges and discussed at length, where "...extraordinary efforts to communicate also appear to be important in naval contexts where rumors can become rampant." (McGlynn, 2005)

A thorough study of the best practices concerning messaging, training, guidance, and other measures, with an eye towards reducing anxiety and building resilience in an open and transparent way, appears warranted, especially given that adversarial attacks via social networks are well documented (Prier, 2017; West, 2016; Singer & Brooking, 2018). **(Recommendation 9)**

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5 ACOUSTIC ATTACK HYPOTHESES

In this section, we consider acoustic-energy projection as a possible covert mechanism for causing harm to individuals. Acoustic-energy projection includes high-energy directed infrasound, audible sound waves, and ultrasound, which are sound waves in different frequency ranges and, as a result, have different interactions with the human body and varying penetration properties into buildings.

In our assessment of sound waves of these different frequency ranges, we follow the criteria we devised from the incident and health reports, as discussed in Section 2: energy must be able to penetrate into buildings through walls and windows from a distance of several tens of meters, be localized to areas that the size of a room or smaller, be generated with equipment that is mobile and covert, lead to audible and/or recordable sounds, cause immediate symptoms of pressure, vertigo, headache, or nausea, and cause long-term vestibular and/or cognitive dysfunction. To that end, we identify the intensity levels, in decibels (dB), that are reported to be linked to health effects and compare these intensities with what may be achieved with delivery mechanisms in realistic operational settings.

5.1 Introduction and Background

Infrasound (IS), is generally described as acoustic waves in the frequency range

$$5\,\mathrm{Hz} \lesssim \nu_{\mathrm{is}} \lesssim 20\,\mathrm{Hz}\,, \tag{5-1}$$

sometimes regarded as extending somewhat below 5 Hz or somewhat above 20 Hz, i.e., a range of acoustic frequencies below the audio spectrum typically perceived by the human ear. In particular and in the present context, infrasound is reported to display, "a special capacity to affect human health and adaptation because its frequencies converge with those generated by the human body" (Persinger, 2014).

Acoustic excitation in the audio range, i.e.,

$$20\,\mathrm{Hz} \lesssim \nu_{\mathrm{au}} \lesssim 20\,\mathrm{kHz}\,, \tag{5-2}$$

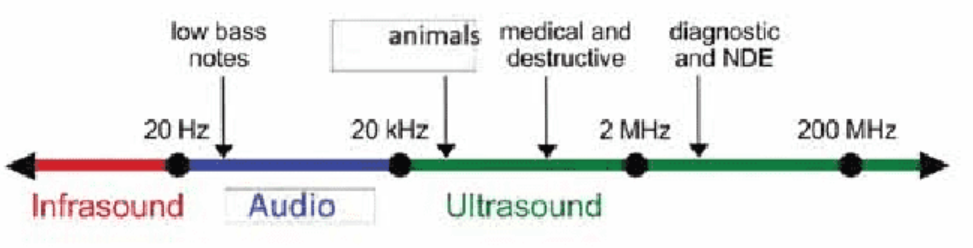

Figure 5.1: Sound-frequency regimes.

brackets human audio perception, with actual low- and high-frequency limits and minimum intensity levels (dB) that can be consciously perceived that vary across individuals and, for any one individual, will also depend on age.

Ultrasound typically refers to acoustic excitation overlapping and extending beyond the high-frequency end of the audible spectrum. Very high frequency sound (VHFS) conventionally refers to the range of

$$11.2\,\mathrm{kHz} \leq \nu_{\mathrm{vhfs}} \leq 17.8\,\mathrm{kHz}\,, \tag{5-3a}$$

and ultrasound (US) to

$$\nu_{\mathrm{us}} > 17.8\,\mathrm{kHz}\,, \tag{5-3b}$$

as noted by Fletcher et al. (2018), even though "ultrasound" sometimes refers to the two concatenated frequency regimes in equations (5-3).

Even higher sound frequencies are used for diagnostic and other imaging applications, as indicated in Fig. 5.1.

We emphasize that humans may be aware of exposure to sound, i.e., acoustic excitation, both below and above audio/audible frequencies, especially of intense sound. People may sense the presence of infrasound even though they may not be able to attribute such a sensation to sound, sometimes perceived and described as a whole-body experience. They may also be able to perceive the presence of ultrasound, as employed as an insect or rodent repellent, for example, even though they may not be able to hear those frequencies, as we'll discuss below.

Land animals may be able to hear both infrasound, especially large animals such as elephants, or ultrasound, such as rodents, dogs, and cats. Dogs are reported to be sensitive to frequencies as high as 45 kHz, while cats are sensitive to frequencies as high as 65 kHz, and can serve as sentinels alerting humans to the presence of such frequencies. Ultrasonic dog whistles (23–45 kHz) that humans may perceive as quiet hissing, exploit dogs' sensitivity to such high-frequency sounds.

Appendix B provides a review of basic concepts as well as estimates of sound attenuation and the limits of focusing sound waves. These calculations support the discussion below.

5.2 Infrasound and Reported Effects

Sound waves interact with the human body in several ways. We first note that there is a large mismatch of acoustic-impedance, $Z_{\rm ac}$, between air and water, and hence between air and the human tissue/body as a whole. Nevertheless, special transduction by human ears allow very low pressure oscillations to be detected. Further, the air-body interface cannot be considered to be rigid at lower frequencies, *e.g.*, for $\nu < 1\,$kHz, where tissue elasticity and viscosity becomes important (*e.g.*, Fry, 1952) and leads to a non-negligible absorption of acoustic energy by the human body. In very intense sound fields, wide-spread stimulation of somatic mechanoreceptors can occur (*e.g.*, Stephens & Bate, 1966). Below 100 Hz, corresponding to sound wavelengths in air, $\lambda_{\rm air} > 3.4\,$m, the body responds as a whole (*e.g.*, Jauchem & Cook, 2007).

Humans routinely interact with a variety of infrasound sources in their environment. These waves are generated by means ranging from natural sources, such as wind turbulence, to man-made sources, such as helicopters and ceiling fans at their blade-passing frequency, road noise, buffeting in cars with only one window (partially) open, Helmholtz resonances in building HVAC-systems, and more recently, from wind turbines.

Owing to the long wavelengths of infrasound waves, it is not possible to focus infrasonic acoustic energy (and the resultant pressure excitations) in free space at length scales relevant to this report (i.e., meters or tens of meters). However, human sensation, perception, and detection are not entirely linear processes. For example, it is possible to generate a localized sensation at a beat frequency ν between two frequencies ν_1 and $\nu_2 = \nu_1 + \nu$, where the frequency ν_1 might be short-wavelength ultrasound (which can be localized) and frequency ν may be in the infrasound range (which cannot be localized). Similarly, non-linear transduction of an ultrasonic acoustic wave at a carrier frequency of ν_1, modulated at an infrasound frequency ν, will generate harmonics at $\nu_1 \pm \nu$, with beating that will generate excitation and a possible sensation at both ν and 2ν. In both cases, the sensation of the infrasound frequency at a frequency ν, can be localized as dictated by the generation and transmission of sound at an ultrasonic frequency, ν_1. Infrasound can also be delivered in interior spaces from/via HVAC furnace combustion chambers, ducts, and plena, localizing excitation, to some extent, creating standing-wave resonance modes in sufficiently large rooms.

Below audible frequencies, the sensation of sound from vibratory stimulation within the ears or by the whole body can appear similar. There is a large variance in sensation, however, attributable to individual differences in perception by humans in response to the "same" physical stimulus (Persinger, 2014).

A number of studies report variable and mostly mild effects of infrasound waves on human health.

Bolin et al. (2011) report a statistically significant association between noise levels and self-reported sleep disturbance in two of three studies. They note that low-frequency noise (LFN) from wind turbines may cause other health problems, but acknowledge that empirical support for these claims is lacking.

Kasprzak (2013) reports on influences by infrasound on a cohort of 17 men and 18 women, aged 19-23, who declared they had no prior medical conditions and were not under the influence of medicines. In the research, the cohort was exposed to infrasound at a frequency of $\nu = 13\,\mathrm{Hz}$ and a sound pressure level of $\mathrm{SPL} = 109.1\,\mathrm{dB(LIN)}$, $62.1\,\mathrm{dB(A)}$,

112.9 dB(G), [1] for an experiment duration of 35 min, with an exposure lasting 20 min, while recording EEG signals. This study reported statistically significant ($p < 0.01$) effects on the sensory motor rhythm (SMR) brain wave rhythm in the $12 - 15$ Hz frequency range, but statistically insignificant effects on other brain-wave rhythms.

Modern wind turbines can generate significant infrasound intensities and harmonics. A three blade wind turbine at 4 rpm will generate a frequency $\nu = 0.2$ Hz and a relative pressure level at about 100 m distance about 50 dB outside a brick house and only slightly less inside the house. The frequency range between 1 and 4 Hz, the delta range for brain waves (EEG measures), is the one involved with slow wave (deep sleep). Disruptions to this sleep can affect the synchronized release of hormones and proteins that facilitate tissue repair and normal homeostasis (Persinger, 2014).

Considering wind-turbine infrasound noise, Berger et al. (2015) find no evidence that exposure to such G-weighted infrasound levels can directly impact human health, attributing self-reporting effects to subjective variables. Baliatsas et al. (2016) in their review paper on low-frequency noise related to wind farms, reported "some associations ... between exposure to LFN and annoyance, sleep-related problems, concentration difficulties and headache in the adult population living in the vicinity of a range of LFN sources. However, evidence, especially in relation to chronic medical conditions, was very limited. The estimated pooled prevalence of high subjective annoyance attributed to LFN was about 10%."

Tonin et al. (2016) also considered low-frequency noise related to wind farms and report that, "at least for the short-term exposure times conducted here-in, that the simulated infrasound has no statistically significant effect on the symptoms reported by volunteers, but the prior concern volunteers had about the effect of infrasound has a statistically significant influence on the symptoms reported. This supports the nocebo effect hypothesis."

A longitudinal, randomized experimental pilot study by Ascone et al. (2021) to investigate the effects of infrasound on "human mental health, cognition, and brain structure" concluded that infrasound (6 Hz, 80–90 dB) applied in the bedroom for 28 consecutive

[1] Units of dB(A)/dBA, dBB, dBG, *etc.*, refer to different spectral-weight filters applied to acoustic spectra to approximate human ears' response to sound. See discussion, for example, in `https://en.wikipedia.org/wiki/Decibel`.

nights produced no effect on healthy individuals. They proposed further, fine-grained studies focused on bodily sensations and perception.

Summarizing the review of infrasound and its effects on humans, available reports acknowledge complaints of discomfort with some affecting sleep patterns, but do not document significant evidence of medical/physiological consequences, especially persistent ones, often ascribing causes and complaints to subjective annoyance and the like.

5.3 Ultrasound and Reported Effects

As discussed earlier in this section, ultrasound (US) acoustic excitation is at frequencies above human normal auditory sensory means. Nevertheless, in that frequency regime, nonlinear response of biological tissues can provide a sensation of perception unrelated to hearing and alert to intense ultrasound exposure. In addition, with respect to the interactions with the human body, inertia by human tissues that cannot react elastically dominates, and acoustic impedance mismatch considerations apply.

Humans exposed to very-high-frequency sound (VHFS) or US acoustic excitation report a number of effects, including pain in the ear, discomfort, and irritation (Ueda et al., 2014). There are similar reports by other researchers, with typically insufficient experimental controls. In a more controlled study on the effects of ultrasound on humans, Fletcher et al. (2018) selected frequencies where individual participants in their cohort had a SPL = 63 dB hearing threshold level and then set the VHFS/US tone level 25 dB higher to avoid exceeding the maximum allowable SPL = 92 dB, set to meet allowable daily noise exposure. The test frequency started at 12 kHz and subsequently increased by a set pattern to 20 kHz, adjusted for individual hearing thresholds. Tests and effects included a Conjunctive Continuous Performance Task (CCPT), Galvanic Skin Responses (GSR), subjective ratings of symptoms. Results are summarized in Fig. 5.2 below.

While Fletcher et al. (2018) report discomfort rating differences between ultrasound and the 1 kHz reference sound, as summarized above, they do not report lasting effects. They note, however, that “the sound pressure levels and exposure durations were limited

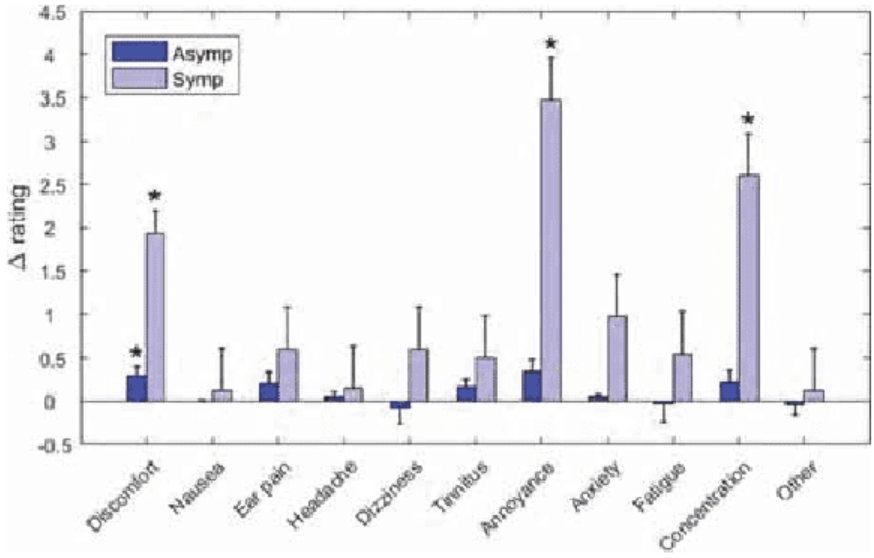

Figure 5.2: Mean difference in rating between VHFS/US and 1 kHz reference conditions for all ratings, averaged over four time periods and all participants (32 asymptomatic, 10 symptomatic). Statistically significant ($p < 0.05$) differences marked with asterisks. From (Fletcher et al., 2018, Fig. 3).

in this study by the application of the precautionary principle, and the researcher's duty of care to participants," and that, "These findings cannot be used to predict outcomes from exposures at higher sound pressure levels or longer durations."

Maccà et al. (2015) report on "significant correlations between workers exposed to audio-frequency noise and tinnitus, sensation of fullness in the ears, and hearing loss [when exposed to sound with intensities] $> 25\,\mathrm{dB}$ [referenced to $p'_{\mathrm{ref}} = 20\,\mu\mathrm{Pa}$ (B-5)] at 4 kHz." Maccà et al. (2015) also note adverse effects on hearing and other health indicators among people who deal with ultrasonic devices in the workplace, such as for cleaning, drilling, homogenizing, *etc.*, in agreement with earlier studies.

In seeking feasible mechanisms for reported adverse effects of ultrasound, Leighton (2016) notes that the "subjective symptoms reported (migraine, nausea, tinnitus, headaches, fatigue, dizziness, feelings of 'pressure') are so common and so non-specific that most clinicians are unlikely to attribute them to a physical cause." Leighton (2016) also reviews and reanalyzes the Maccá *et al.* data, agreeing with Maccá *et al.* that the data show significant increases in asthenia (loss or lack of bodily strength) and vertigo. Case histories show that

symptoms persist for a few days after exposure and cease subsequently, with no further occurrence, in the absence of exposure. The tabulated results are summarized and reproduced in Figs. 5.3 and 5.4. The overlap with complaints, symptoms, and syndromes reported in the present context is noteworthy.

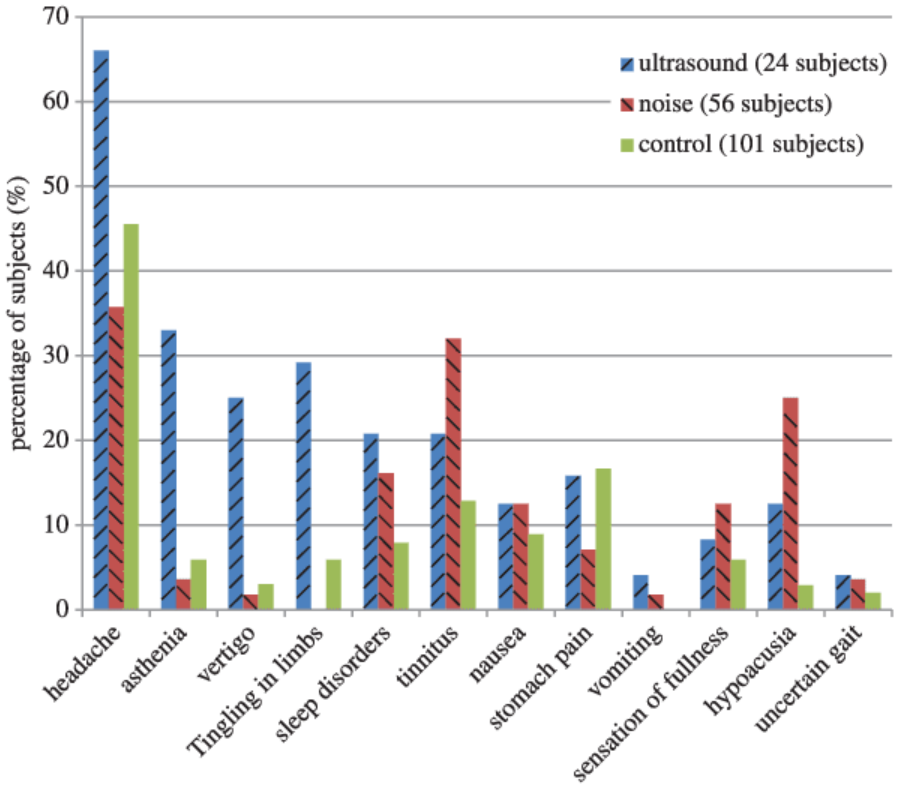

Figure 5.3: Occurrence of a range of symptoms related to ultrasound and industrial noise based on the Maccà et al. (2015) data, from (Leighton, 2016, Fig. 7).

Conflicting reports and etiologies abound, however, and (Leighton, 2016) notes that any author raising issues of possible health hazards related to ultrasound "comes at reputational risk," as many initial reports have later been attributable to psychosomatic causes. Nevertheless, the evidence suggests that VHF/US exposures elicit greater adverse effects in some individuals than others, possibly even without the need to hear the signal, or without the degree of suffering related to the degree of audibility of the VHF/US signal by an individual. As a consequence, the usable/acceptable dynamic range between what is perceptible and what

may cause adverse effects may be unexpectedly small (Leighton et al., 2020). It is worth reiterating that even among individuals who appear to show symptoms related to ultrasound noise, these symptoms are transient and do not continue well beyond exposure.

	subjects exposed to	
symptom	ultrasound	industrial noise
headache	0.0628	0.232
asthenia	**0.000131{8}**	0.518
vertigo	**0.000175{6}**	0.652
tingling in limbs	**0.000807 {7}**	—
sleep disorders	0.0625	0.115
tinnitus	0.318	**0.00366 {18}**
nausea	0.592	0.476
stomach pain	0.984	0.0876
vomiting	**(0.0394) {1}**	0.178
sensation of fullness	0.667	0.153
hypoacusia	**(0.0496) {3}**	**0.000021{14}**
uncertain gait	0.529	0.544

Figure 5.4: The p-values from the data of Maccà et al. (2015) based on cohort sizes of 24 and 56 for the number of subjects exposed to ultrasound and industrial noise, respectively. Entries with p-values below 5% are in bold and enclosed in parenthesis if they do not have at least 5 instances for the corresponding symptom. Numbers in braces denote the number of occurrences for which $p < 5\%$ (Leighton, 2016, Table 2).

Closing this part of the discussion, we estimate the characteristics of a device that could be used to cause adverse health effects. An ultrasonic beam in the tens of kHz range would experience tolerably low atmospheric attenuation, a beam directivity that can be achieved at the requisite level, and resonant emitters readily available. We will adopt 40 kHz as a nominal frequency below, but the results also hold for frequencies in the range of 20 kHz (below which the signal would be in the audible range) to 100 kHz (above which atmospheric attenuation becomes too large.)

An emitter with a characteristic dimension of $D \simeq 1\,\mathrm{m}$ would provide a diffraction-limited beam-divergence at 40 kHz of $\Delta\theta \simeq 8.5\,\mathrm{mrad}$. For a standoff distance of $x \simeq 30\,\mathrm{m}$,

such an acoustic beam would expand by 0.25 m, which is small enough compared to the $D \simeq 1$ m beam width to treat the beam as focused.

A requisite power level for harm could be estimated based on the occupational exposure limit set by OSHA for ultrasound at 40 kHz,[2] and is SPL = 115 dB (relative to 20 μPa). For the sake of this exercise, we will presume that lasting physiological damage from a one-time exposure requires ultrasound levels akin to that needed at audible frequencies, i.e., 150 dB. Conversely, we will treat this level as providing an important exposure threshold that, if not exceeded, effectively rules out ultrasound acoustic-wave energy as an admissible modality in many, if not most, cases.

Transmitting a 40 kHz beam over a distance of $x = 30$ m and then through a window pane introduces 20 dB of attenuation along the path, and another 60 dB through the window pane, as discussed in Appendix B. Harmful levels would then require a source that can emit at a Sound Pressure Level of SPL = 150 + 80 = 230 dB. While this may not require a large amount of propagating acoustic power, only of order 100 mW, coupling power from an acoustic emitter to air is inefficient. By way of example, the LRAD 500X-RE is a high-power audio-frequency device with a peak power of 154 dB and marketed to armed forces for long range transmission of audible sound as a deterrent or weapon.[3]

Transferring relevant ultrasonic power levels over a distance of 30 meters and through a closed window would require a device to broadcast 150 times as much power as is available from high-end commercial devices. Moreover, the audio power levels outside the building (including reflections) would be 1000 times higher than inside, hardly covert, and leading to acute distress on bystanders and operators.

We conclude that remote ultrasonic attacks are, therefore, highly unlikely, on technical power grounds.

[2] https://www.osha.gov/otm/section-3-health-hazards/chapter-5\#appendixc
[3] https://genasys.com/lrad_products/lrad-500x/

5.4 Concluding Remarks

We considered acoustic-energy projection as a possible covert attack modality. High intensity infrasound and ultrasound emerged as possibilities on the basis of the health effects reported to be associated with exposure to these sound waves. Among the two, effects in response to ultrasound were more significant and had a larger overlap with reported syndromes. However, such effects are not reported to persist, at least following exposure at intensity levels that have been investigated.

In terms of health effects, the challenge of etiology and attribution of biomedical effects remains, however, complicated by the likely admixture of psychogenic effects, the large variance in susceptibility between individuals to infrasound and ultrasound, and possible differences in the mechanisms responsible for exposure. Further and importantly, careful controlled experiments with human cohorts are understandably limited to exposures deemed safe for human subjects and, as a consequence, have not probed to explore permanent effects extending to damage resulting from levels that are, or could be, considered unsafe. In the case of ultrasound, these are long-established and accepted based on experience with, e.g., medical ultrasound imaging, extending to fetuses.

However, that the investigations of medical effects of exposure to infrasound and ultrasound have been conducted with due regard to safe exposure intensity levels when applied to human subjects is important. In particular, while some human subjects registered and reported discomfort and irritation, at such exposure levels **no persistent effects have been reported.**

Such reports, coupled with our theory, analysis, and measurements suggest, however, that it is difficult, if not infeasible, to project such acoustic-wave energies/fluxes into the interior of a building, where many occurrences were reported to have taken place. Specifically, infrasound cannot be delivered from a distance of tens of meters with the level of focusing consistent with our incident criteria, without equipment that is tens of meters in size. We regard that as implausible, and can rule out infrasound as a candidate modality. For ultrasound, atmospheric attenuation with typical relative humidity (RH%) levels, geometric

attenuation if impinging from the outside as spherical waves from some distance, and the small acoustic transmission through a glass window pane or wall, combine to limit possible exposure in the interior of buildings to Sound-Pressure Level (SPL) values not reported to produce lasting effects, for covert acoustic sources. As a result, acoustic energy impinging from a building exterior with no open windows does not emerge as a candidate modality inside buildings. Ultrasonic acoustic power could be beamed/focused in open spaces at SPL values that may be injurious, but not easily by covert means.

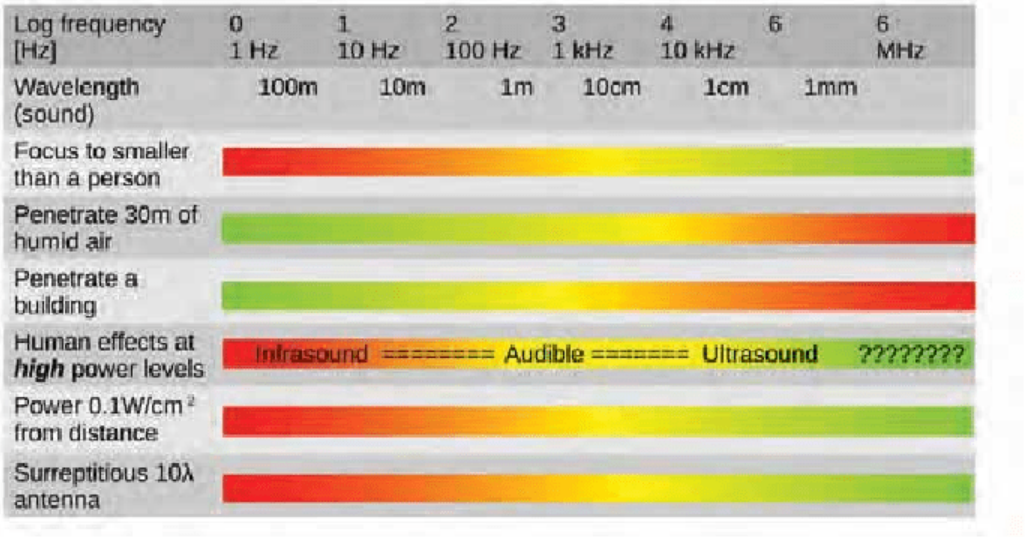

Figure 5.5: Combined assessment of acoustic waves as a potential cause for embassy incidents on all relevant criteria. An admissible modality, including the requirement of remote delivery, would require green along a vertical swath in this graph.

We summarize our assessment of sonic hypotheses in Figure 5.5. The color coding in the figure corresponds to the following: red indicates that the mechanism is inconsistent with the requirements or effects considered, yellow indicates some level of inconsistency, and green represents consistency. The fact that no frequency is green across a swath crossing the multiple rows shows that acoustic modalities can be ruled out as a cause for the embassy incidents **(Finding 5)**.

6 ELECTROMAGNETIC ATTACK HYPOTHESES

6.1 Introduction and Background

Electromagnetic (EM) radiation refers to oscillating coupled electric and magnetic fields that propagate in space as traveling waves. The wavelength λ and the frequency f of the wave have a fixed relation to one another given by $\lambda = v/f$, where v is the speed of the wave. In vacuum, EM waves travel with the speed of light $c = 2.998 \times 10^8$ m/s. As with acoustic waves, the energy flux (energy per unit area per unit time, or power per unit area) carried by the wave is proportional to the square of the wave's amplitude (here, the peak electric or magnetic field strength).

The properties of EM radiation vary with wavelength λ over the electromagnetic spectrum, ranging from radio frequency (wavelengths greater than about $\lambda \sim 1$ m, corresponding to frequencies less than about 300 MHz), to microwaves (wavelengths from about 1 m to 1 mm, and frequencies from 300 MHz to 300 GHz), to optical photons (400 to 700 nm wavelengths) to X-rays and gamma rays (wavelengths shorter than 10 nm). In the discussion that follows, we will specifically use the term "radio frequency (RF) waves" and "microwave frequencies" to mean electromagnetic radiation with frequencies in the 300 MHz - 300 GHz range (wavelengths ranging from 1 meter to 1 millimeter.) Further, within this range, the following distinctions are commonly made: the UHF band spans 300 MHz - 1 GHz (1 - 0.1 m); the SHF band spans 3 to 30 GHz (10 - 1 cm); the EHF band spans 30 to 300 GHz (10 - 1 mm).

EM radiation is generated by oscillators: charged particles accelerated back and forth by either natural or artificial means. An antenna typically creates electromagnetic radiation by driving an oscillating current of electrons in a conductor. EM radiation can be generated as a continuous wave, with a single carrier frequency, or as pulsed radiation that consist of packets of duration τ_p and pulse repetition frequency (PRF) that measures the number of

pulses per unit time, i.e. per second. Generating pulsed radiation creates a finite bandwidth around the carrier frequency that increases as the pulse duration decreases. Roughly speaking, the bandwidth BW $\approx 1/\tau_p$.

Electromagnetic radiation incident on matter will interact with charged particles within the matter, accelerating them back and forth as the wave goes by. Through this interaction, the energy carried by the wave will be transferred to the motion of particles in matter. From energy conservation, EM energy deposited in matter will be lost from the wave, and the wave will attenuate as it propagates through matter. If the irradiated matter is a receiving antenna, the resulting current from the oscillating electrons can be amplified into a measurable signal. The energy transferred to the material will often end up as heat, but in general can be converted into any other kind of energy. Energy from an electromagnetic wave can also be reflected or re-radiated by the material.

The strength of this interaction, and the resulting rate of attenuation of the incident wave, depends both on the frequency of the wave and on the nature and composition of the matter. In lossy conductors, attenuation is exponential and can be expressed as

$$I(x) = I_0 \tau e^{-x/L}, \tag{6-1}$$

where I_0 is the intensity of radiation incident on the lossy conductor, τ is the fraction transmitted into the lossy conductor, x is the depth into the lossy conductor and L is the *penetration depth*. Penetration depth L refers to the depth where the intensity drops to 1/e of its original value. The term *skin depth*, denoted δ_p, instead refers to the depth where the field or current density drops to 1/e. Because power is proportional to field squared, $L = \frac{\delta_p}{2}$. Human tissues, including skin, muscle and fat, can be treated as lossy conductors. EM waves interacting with human tissues experience an exponential attenuation, as do typical building materials that the waves pass through, analogously to acoustic waves. In addition, due to the nature of the interaction, shorter wavelengths penetrate to smaller depths within a conductor. We will return to the concept of penetration depth and its implications for the types of effects it causes on people, in more detail when we consider RF waves.

In our discussion, we will group ionizing radiation under EM waves, although ionizing radiation can consist of energetic particles as well as waves. Ionizing radiation is released by atoms and their nuclei; those in the form of electromagnetic waves include X-rays and gamma-rays, while particles can be in the form of neutrons, alpha-particles or beta-particles. Such radiation contains sufficient energy to ionize matter by freeing a bound electron.

The energy carried by an electromagnetic radiation source will spread out in space as the wave propagates from the source. If the source is isotropic (radiates equally in all directions), then the power received from the source (energy transferred per time) will decrease as

$$P(r) = \frac{P_o}{4\pi r^2}, \tag{6-2}$$

where P_0 is the emitted power and r is the distance from the source. If the source wave is *beamed*, this means that the radiator (e.g. antenna) radiates more strongly in a particular direction; this is modeled as a *gain factor* that depends on the direction around the antenna. Power will still decrease as the inverse distance squared from the source. The properties of transmitted radiation fields in both frequency and spatial power distribution can be manipulated by the shapes and sizes of transmitting antennas.

Electric and magnetic fields need not be time-varying; fields which do not change amplitude with time are called static. Neither static electric fields nor magnetic fields are relevant to the present study. Static electric fields can certainly cause harm to humans, usually via the production of large voltages across the human body and resultant damaging current(s). However, the upper limit to static electric fields in everyday life is set by the dielectric breakdown of air; that is, sufficiently large fields ionize air molecules, producing sparks (as in the lightning of thunderstorms or the sparks that result when live wires come into very close proximity.) The production of large electric fields requires the application of high voltages across two or more conductors; this is inconsistent with situational reports. Additionally, although high static electric fields can be harmful, the effects are inconsistent with those reported.

Magnetic materials can be used to produce static magnetic fields. The SI unit of the magnetic field is the Tesla (T). For reference, humans are exposed continuously to the

Earth's magnetic field, which has a value on order of 50 μT. Kitchen magnets have strengths ranging from 1-100 mT; centimeter-scale permanent magnets with strengths on order of 1 T are available commercially (e.g., neodymium magnets). Clinical magnetic resonance imaging machines (MRI) produce fields on order of $3 - 7$ T and research machines can produce much higher fields, on the order of 30 T, but the bores size is too small for human bodies. Static fields are generally considered safe up to MRI strengths. Some caveats exist with respect to exposure time but, in any case, affecting a human body at any significant distance with >1T strength magnetic fields would be impractical or impossible. A review of static magnetic field effects, both homogeneous and inhomogeneous (i.e., producing field gradients) can be found in Zhang et al. (2017).

6.2 Ionizing Radiation

We first consider whether ionizing radiation is a possible attack modality.

6.2.1 Generation, focusing, absorption, and interference

Ionizing radiation is the common term used for particles or electromagnetic waves that have sufficient energy to ionize atoms by removing electrons from them. The term covers X-rays, gamma-rays, neutrons, alpha particles, and beta particles. X-rays and gamma-rays are highly energetic photons, while the rest are energetic particles. This property gives them the ability to damage cells and the DNA in cell nuclei and makes them harmful to living organisms.

X-rays are a high-energy, short-wavelength form of electromagnetic radiation. The X-ray band covers the wavelength range approximately between 10 nanometers (10×10^{-9} m, corresponding to a photon energy of 0.1 keV) to 12 picometers (12×10^{-12} m, corresponding to a photon energy of 100 keV). They are commonly produced in X-ray tubes by accelerating electrons through a potential difference, or a voltage drop, and directing them onto a target material such as tungsten.

Gamma-rays have even higher energies and shorter wavelengths than X-rays, typically smaller than 12pm (though note that some sources define smaller energy radiation as a gamma-ray if it originates from a radioactive process). The primary generation mechanisms are processes that involve the nuclei of atoms, including radioactive decays, nuclear fusion, and nuclear fission.

Alpha particles consist of two protons and two neutrons bound together (i.e., the nucleus of a helium atom). They are usually produced as a result of radioactive decay. Alpha particles are a strongly ionizing form of radiation, but at typical energies up to 8 MeV, their penetration depth is small and they can be stopped by a few to 10 centimeters of air. As a result, alphas cannot travel long distances from a source and cause harm.

Beta particles are high-energy electrons or positrons. They are more penetrating than alpha particles but still cannot be used to direct energy from longer distances or penetrate buildings.

Both X-ray and gamma-rays have high penetrating power. Depending on their energy, they can pass through several inches of a dense material like lead and up to several feet of concrete, potentially causing damage even when they travel through a wall.

X-ray beams can be collimated or reduced in size using pinholes or movable slits typically made out of tungsten or some other material with high atomic charge. A collimator works by only allowing rays coming from certain angles to pass through and forms a directional beam that way. To focus them further requires using “grazing incidence” mirrors, which involves reflecting them at grazing incidence angles. This requires specialized and often bulky optics. Gamma rays require even smaller grazing incidence angles to focus.

However, they can also be collimated. Because collimators typically consist of a honeycomb of tubes made of absorbing material, they significantly reduce the intensity of the source by admitting only a fraction of the radiation through.

The combined difficulties of generation, collimation, and penetration of ionizing radiation across the relevant distances renders this attack modality highly improbable with a portable device **(Finding 5)**.

6.2.2 Effects on the human body

The damaging health effects of short-wavelength or ionizing electromagnetic radiation, such as X-rays and $\gamma-$rays have been known for a long time. The damage due to radiation depends on the type of radiation emitted, the dose received by a person, and the parts of the body that are exposed. The radiation dose itself scales with the duration of exposure and the power of the source, and inversely with the square of the distance from the radiation source.

When ionizing radiation interacts with tissue, it causes damage to the cells as well as harmful changes to the genetic material, or the DNA, in the cell nucleus. As a result, it can cause skin burns, radiation sickness, widespread organ damage, as well as neurological symptoms, fatigue, and nausea. These symptoms are inconsistent with the medical records and reports of affected individuals, rendering exposure to ionizing radiation not a viable explanation for the reported incidents **(Finding 5)**.

6.3 Bounds on RF Wave Properties

We now turn to RF waves and assess whether they can produce effects consistent with the incidents reported. We place some immediate bounds on the properties of RF waves of potential interest using some of the Incident Consistency Criteria discussed in section 3. In addition to those listed there, the lack of reports of a heating or burning sensation during the incidents provides another bound on the RF parameter space, as we will show below.

Regarding the association of sound with RF waves, there is a mechanism, known as the Frey effect, which has been shown to induce a sensation of sound in people. We will discuss this in more detail in section 6.5.4. For now, we simply note that this mechanism requires the use of pulsed RF radiation, a PRF in the audible range, a carrier frequency in the 300MHz−10GHz range, and a sudden deposition of a small amount of energy into the ear. As a result, if the sounds reported during the incidents are to be explained through incident RF radiation, this imposes strong constraints on the type of radiation of interest.

Sound sensations produced by the Frey effect *cannot* be recorded by external electronic microphones. This is an important difference between the sensation of sound produced through the Frey effect and that produced by actual sound waves and rules out RF waves as the source of any incident with recorded sounds **(Finding 5)**.

The multiple attributes of pulsed RF waves form a multi-dimensional parameter space that is somewhat difficult to navigate. The parameters of interest include carrier frequency f, peak energy flux F_p, average energy flux F_{avg}, the PRF, and the pulse duration τ_p. We will now examine the various constraints on this parameter space and show how we can narrow it down to a small region that remains interesting.

6.3.1 Penetration Depth, Skin Heating, and the Carrier Frequency Range

We first show that the carrier frequency can be bounded by considering the penetration depth of RF into the human body. Microwave radiation incident on the human body will be partially reflected and partially absorbed. A reasonable value for the fraction of power absorbed, τ, lies between 0.4 and 0.5 (Foster et al., 2016). Because the human body is a lossy conductor, microwave energy is subject to exponential attenuation as it penetrates inward, as discussed in Section 6.1.

The penetration depth (and skin depth) of microwave radiation into tissue depends on the frequency of the radiation and the type of tissue. Gabriel et al. (1996) provides measured permittivity and conductivity vs. frequency data for various human tissues. This data can be fit to a model based on multiple Cole-Cole dispersions (Eq. 4 in Gabriel et al.

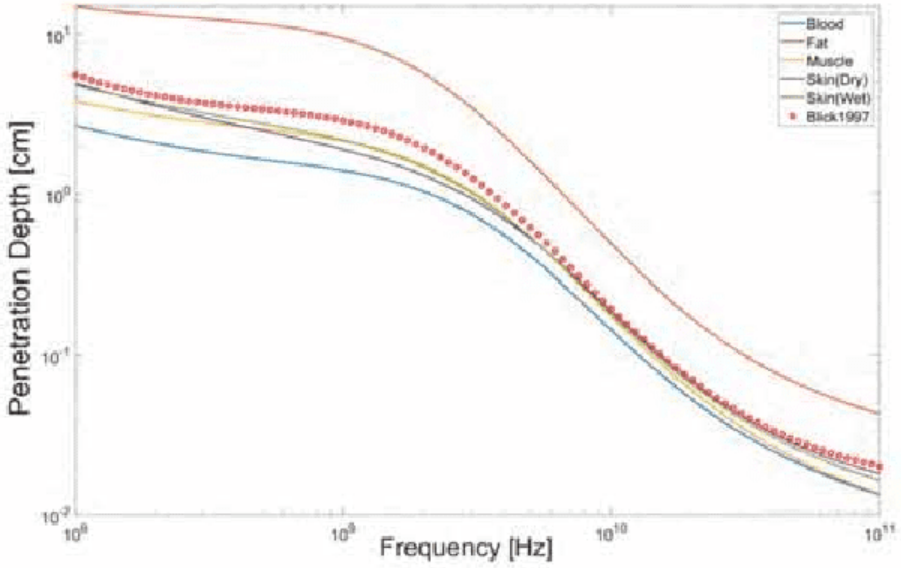

Figure 6.1: Penetration depths for various tissue types vs RF frequency as calculated by JASON, see section 6.3.1. Above $\sim 20-30$ GHz, RF waves penetrate less than 1 millimeter into tissue. Dotted line is based on reference Blick et al. (1997).

1996) to produce an equation for the relative complex permittivity, ϵ_r. Using ϵ_r calculated for each tissue, we can calculate the skin depth, δ_p using

$$\delta_p = \frac{67.52}{f}[\sqrt{\epsilon'^2 + \epsilon''^2} - \epsilon']^{-1/2} \text{ mm}, \tag{6-3}$$

where f is the frequency of the radiation in GHz and $\epsilon_r = \epsilon' - i\epsilon''$. Note that ϵ' and ϵ'' are frequency dependent. Recalling that $L = \frac{\delta_p}{2}$, we plot the penetration depth, L, for various frequencies in Figure 6.1. We also plot the penetration depth estimated using a simplified approach presented in Blick et al. (1997).

What is apparent is that for frequencies $f > 20-30$ GHz, penetration depths are less than 1 mm. That is, the incident energy will be deposited at very shallow depths and simply cause skin heating (and accompanying sensations of warmth and, for sufficiently high powers, pain). As a result, we will not consider frequencies above 30 GHz further. Conversely, for frequencies $<$ 500 MHz, penetration depths can be many centimeters, implying that energy can be deposited into the bulk of an organism. In the 500 MHz $-$ 30 GHz region (note that these extremes are arbitrary and one could consider somewhat larger or smaller

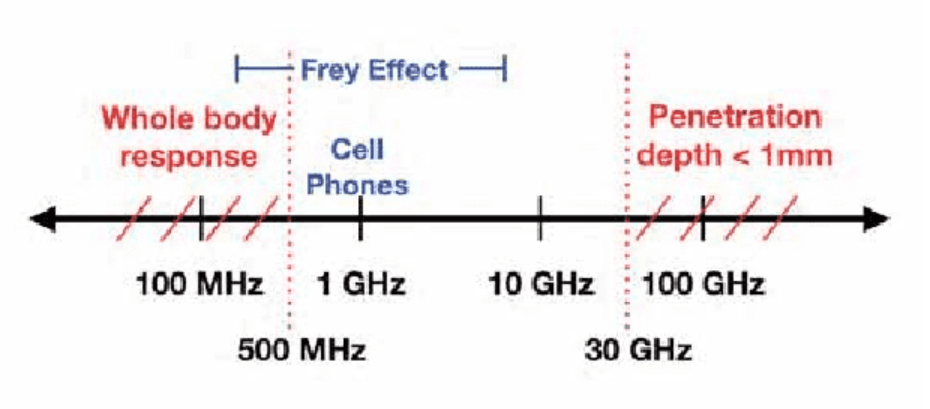

Figure 6.2: The bounds on RF frequencies imposed by consideration of penetration depth on human body.

ranges), there is the possibility of shallow penetration. We will consider these two regimes in more detail in Section 6.5 but we will focus our discussion of a remotely operated RF device to the 500 MHz−30 GHz region. We show these regimes in Figure 6.2. We also denote the frequency range for cell phones on this plot as well as for the Frey effect, which we will discuss in Section 6.5.4.

6.3.2 Peak and average flux

For pulsed RF waves, the average flux incident on a surface, F_{avg}, and the peak flux F_{p} incident during the pulse are related by a 'duty cycle': $F_{\text{avg}} = DF_{\text{P}}$, where the duty cycle D is just the ratio of pulse duration to the time between pulses. Phenomena such as heating of tissues are sensitive to the average flux because they are controlled by the total energy deposited per unit time. Some phenomena, such as the disruption of electronic devices or the Frey effect, are instead sensitive to the peak flux in the pulse. Using this fact, we can identify regions in the $(F_{\text{avg}}, F_{\text{p}})$ space that are consistent with the reported phenomena.

In Figure 6.3, we delineate regions where

- F_{avg} is large enough to produce perceptible sensations of heating and pain. Average flux (sometimes called average power density) thresholds are discussed in Section 6.5.2. Sensations of heating or skin pain are inconsistent with reports. This bound is

frequency dependent; to be conservative, we choose the highest power density value in Table 1 which is 63 mW/cm^2 at 2.45 GHz,

- $F_{\rm p}$ is large enough to cause disruption of consumer electronics, for pulse widths $\tau_p >\sim$ns,
- The region occupied by ordinary cell phones and MRI machines, which are deemed safe for human health.

The regions shown in red can be eliminated from further consideration by these arguments.

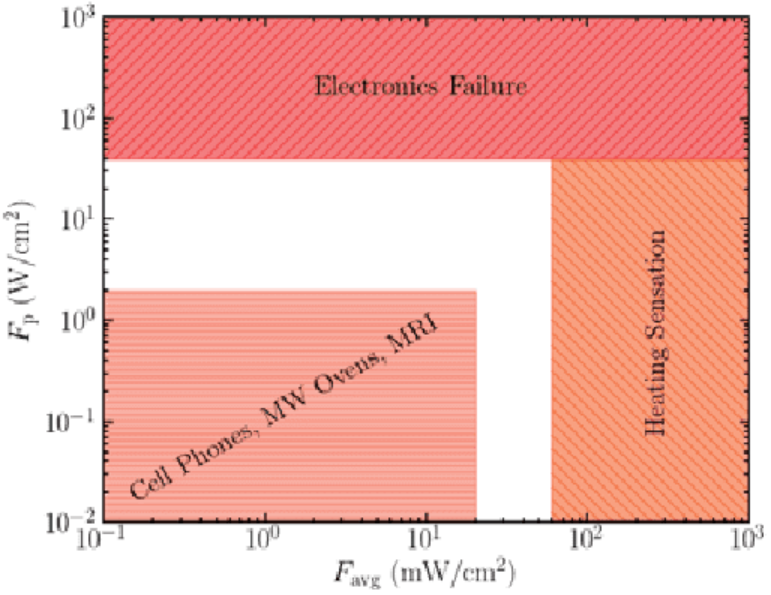

Figure 6.3: The bounds on the average flux ($F_{\rm avg}$) vs peak flux ($F_{\rm p}$) parameter space imposed by various physical and physiological effects and known safe bounds.

Several studies explored the upset and destruction of electronics by nanosecond RF pulses (Camp et al., 2001; Nitsch et al., 2004; Backstrom & Lovstrand, 2004). These studies were performed in the early 2000s; therefore, the electronics studied are representative of that era, with their coarser circuit features and less dense printed circuit boards, and likely with lower distributed capacitance compared with contemporary electronics. Camp et al. (2001); Nitsch et al. (2004), in particular, focused on sub-nanosecond irradiation, and reported upsets typically around 10kV/m (corresponding to peak fluxes of 25 W/cm^2)

and permanent damage at an order of magnitude higher (thus 2.5 kW/cm^2). We adopt here the $F_{\rm p} = 25$ W/cm^2 limit, but note that contemporary electronics may have different thresholds for disruption **(Finding 6)**. Placing firmer experimental bounds on the effect of RF waves on contemporary electronics can help narrow down this space further. **(Recommendation 8)**

6.3.3 Pulse duration and repetition frequency

We can compare incident data with information in the open literature to estimate the range of pulse width and pulse repetition frequency (PRF) that may be of interest for sensing. To begin with, the majority of literature-reported thermoacoustic effects (i.e., sounds produced by pulsed radiofrequency waves via the Frey effect) use pulse repetition rates of 1000 Hz (Elder & Chou, 2003). The highest PRF reported for a Frey effect is 20,000 Hz, but the upper end of this range is not readily reproducible. At fluxes of tens of mW/cm^2 (see Elder & Chou 2003 and Section 6.5.4) and in the $1 - 10$ GHz range, these signals would likely produce interference in communication systems (e.g., for mobile phone signals as well as 2.4 and 5 GHz WiFi.)

One last concern is the possibility of extremely fast pulses. A pulse of duration δt will spread over a bandwidth of $1/\delta$t Hz. This means that a very short pulse that encompasses only one oscillation of the carrier wave will be spread very widely in frequency. In other words, much of the energy will not be at the carrier energy. A reasonable rule of thumb is to assume each pulse must contain at least 10 oscillations of the carrier wave. For a 1 GHz carrier wave, for example, a single oscillation is 1ns and a ten oscillation pulse would have a bandwidth of 100 MHz. For a 10 GHz carrier, the smallest pulse would then be 10×10 ps = 100 ps, corresponding to a bandwidth of 1 GHz. This places a bound on the smallest pulse to reasonably consider for a given frequency.

6.4 RF Radiation Beaming, Propagation, and Absorption

6.4.1 RF beaming

Focusing RF radiation is achieved typically by using parabolic reflectors. The electric field pattern beamed from a such a reflector (or a circular phased array) is circularly symmetric and depends on angle θ from on axis. As a rule of thumb, a dish or phased array forms quite a localized central beam of angular width λ/D radians, where λ is the wavelength of the radiation, D is the antenna diameter. The ratio of the distance of an antenna of size D to the localization of focus is roughly equal to size of the antenna measured in wavelengths. The size of a beam in physical length units, for a target located at a distance L from the reflector can, therefore, be estimated as $X \approx L\lambda/D$.

For example, achieving a beam divergence of approximately 1 m at a distance of 50 m, as indicated by our incident consistency criteria, requires an antenna that is at least 50 wavelengths in size. This results in the following dish sizes for some frequencies of interest:

- 300 MHz $\sim$ 1 m wavelength: 50 m emitter diameter
- 1 GHz $\sim$ 30 cm wavelength: 15 m emitter diameter
- 3 GHz $\sim$ 10 cm wavelength (S band, cell phone): 5 m emitter diameter
- 10 GHz $\sim$ 3 cm wavelength: 1.5 m emitter diameter
- 30 GHz $\sim$ 1 cm wavelength (Ka band, 5G): 50 cm emitter diameter
- 100 GHz $\sim$ 3 mm wavelength (Active Denial System): 15 cm emitter diameter

An antenna required to localize its emission focus to $\sim$1 m at a distance of 50 m would be quite noticeable for frequencies lower than $\sim$5 GHz.

There are also techniques for creating a localized beam using the interference between two or more antennas (as is done in aperture synthesis interferometry). However, using these techniques would require tight spatial coordination between the locations of the dishes and would be logistically very challenging.

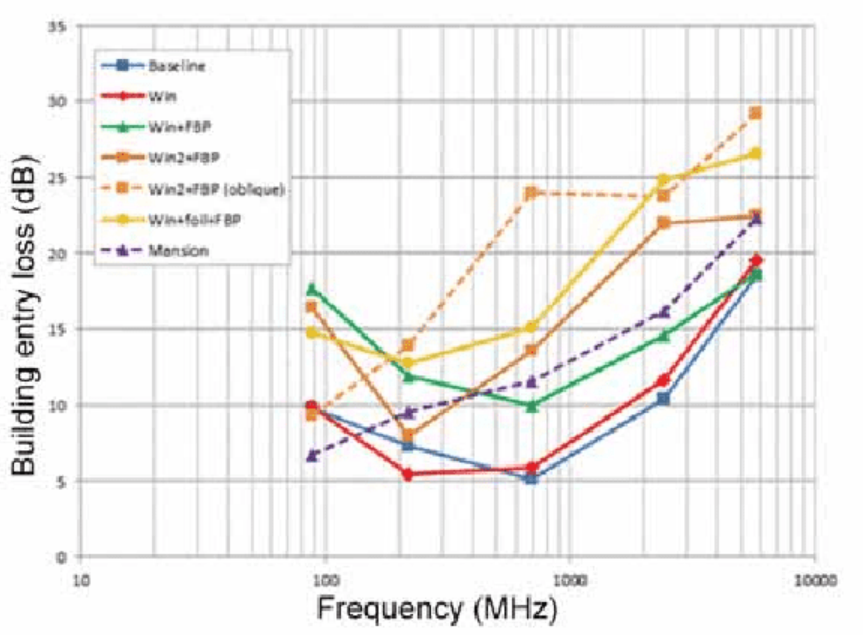

Figure 6.4: Entry loss as a function of frequency through metallized windows (Win) and fiber backed plasterboard (FBP) of a typical modern house as well as a brick "Mansion".

6.4.2 RF absorption and interference

Attenuation and entry loss of RF into buildings is described in detail by `https://www.ofcom.org.uk/_data/assets/pdf_file/0016/84022/building_materials_and_propagation.pdf`. Their Figure 1-1, reproduced as Figure 6.4, shows the attenuation of RF into a building at frequencies between 100 MHz and 6 GHz. In this frequency range, the attenuation ranges from 5 dB to 30 dB, with $\sim$10 dB variations depending on the construction material and steadily rising toward higher frequencies.

Bas et al. (2009) did a study of the penetration of 28 GHz ($\sim$1 cm wavelength, 5G cell phone) radiation from outside a building to inside, and found a typical penetration loss of 23 dB Bas et al. (2019). They used a realistic building with diverse components; the penetration through window, concrete block, and wood frame and drywall are quite different.

Albeit significant, the level of attenuation reported in these studies is not prohibitive

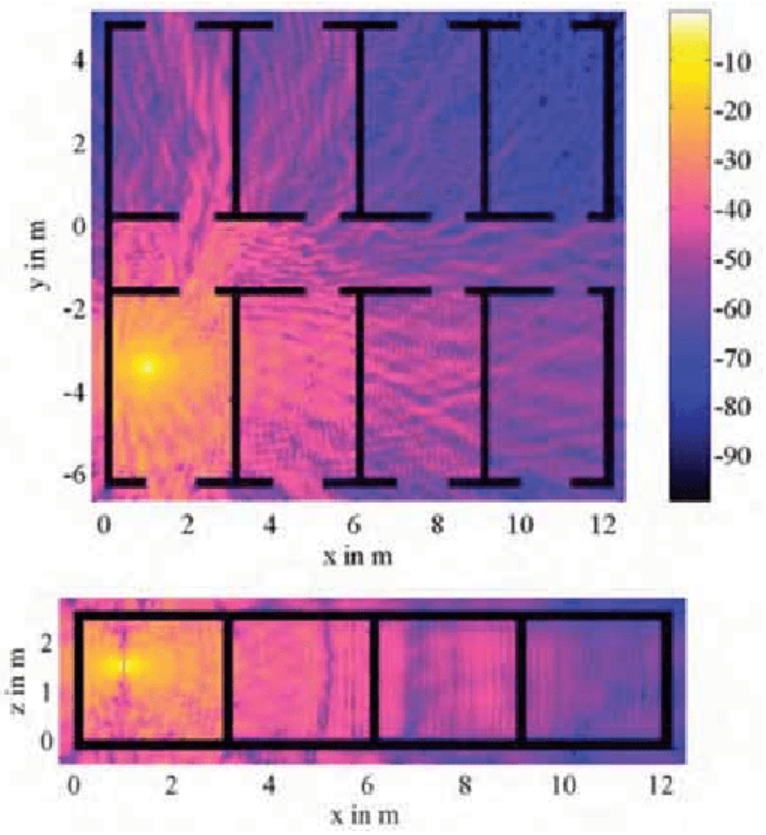

Figure 6.5: Field coverage (received E-field in dB) of building floor with inhomogeneous periodic walls for v-polarization. In this assessment, the source is inside the building, at the position of least attenuation (yellow).

at ∼GHz frequencies but penetrating effectively through a building is progressively more challenging at frequencies higher than a GHz.

RF waves exhibit a high level of interference (producing spatial variations of field strength) when passing through materials and a high degree of scattering when reflecting off ordinary surfaces. Thiel & Sarabandi (2009) calculated the effects of attenuation and scattering of 1 GHz (30 cm wavelength) wave within a typical office layout. Figure 6.5, reproduced from their Figure 6, demonstrates these effects, including attenuation with distance, attenuation because of passage through walls, and interference which creates nodes and antinodes on typical scales of a half wavelength. In particular, this figure illustrates that the signal remains collimated only when there is a direct line of sight to the source;

otherwise, scattering and interference patterns destroy collimation and spread the beam to scales comparable to the size of the room.

These interference patterns may shift as scatterers such as humans or furniture move around and, as illustrated, the power flux between a node and antinode can be an order of magnitude or more, although typically somewhat less. The location of fringes is typically different for different polarizations. Because of the sensitivity to the exact details of the layout within a building, without complete knowledge of all materials present it would be virtually impossible to predict exactly where such interference fringes will occur.

6.5 RF Effects on Human Health

We turn to the effects of RF radiation on human health, focusing in particular on the question of long-term effects. We also review the literature with an eye toward effects that can be induced without causing a sensation of pain or warmth, to be consistent with incident reports.

The study of electromagnetic interaction with biology has a long and storied history. The academic literature on microwave effects on biology, especially in the context of effects on humans, is large and spans the better part of a century and many thousands of papers, books and articles. Two of the most comprehensive and balanced synthesis of microwave effects on biological systems are Lin & Michaelson (1987) and Barnes & Greenebaum (2007).

As of this report, the prevalent opinion in the literature is that most, if not all, demonstrated in vivo effects (including thermoacoustic phenomena like the Frey effect) are due to the deposition of thermal energy by microwaves into tissue.

How thermal energy is deposited into the human body varies by frequency but is reasonably well understood. There exist detailed analytical (Adair & Black, 2003; Foster et al., 1998; Foster, 2000; Foster et al., 2010, 2018), lattice (Gowrishankar et al., 2004), and finite element models on this question, which have been demonstrated and validated (Hirata et al., 2003; Zasowski et al., 2006).

Considering the possibility of direct penetration of energy into the ear canal rather than through normal pathways of skin and tissue, some recent studies have shown that 60 - 90 GHz RF waves may be able to deliver energy deeper into the ear canal than RF waves of lower frequency (Vilagosh et al., 2018, 2020). However, this effect is very weak. "Since the absolute value of the [power flux density] is trivial at 30 GHz, the transmission has no practical significance. The higher penetration of the 60 GHz and 90 GHz pulses results in approximately 2.3% of the [power flux density] incident on the outside of the ear canal being transmitted into the middle ear at 90 GHz." (Vilagosh et al., 2020)

6.5.1 The $f < 500$ MHz Range

For frequencies where the penetration depth is on order of a body dimension, microwaves will penetrate through the entire body. The relevant thermal effect can be well modeled by a homogeneous thermal input (load) on the human body (Adair & Black, 2003), i.e., by writing a heat balance equation

$$A + M \pm W = \pm R \pm C \pm E \pm S \tag{6-4}$$

where all terms are in power units (e.g., Watts), and A, M, W, R, C, E and S are the rates of energy absorption from the RF field into the body, thermal energy production through metabolic processes, work production by or on the body, heat exchange with the environment via radiation, heat exchange with the environment via convection, heat exchange with the environment via evaporation, and heat storage in the body, respectively (Adair & Black, 2003). Additionally, microwave resonance effects are possible at these scales if the wavelength of the radiation, λ, is also on order of a relevant body dimension (Foster, 2000). Thus, to first order, incident microwaves introduce a bulk thermal energy input, which must be dealt with by the organism.

At sufficiently high RF intensities, enough thermal energy is deposited into the system to produce a measurable increase in temperature. The thermal gradients produced by such energy deposition differ from those of ambient heating. Above certain intensities, RF energy can produce morbidity, and, after thermoregulatory mechanisms are overwhelmed, mortality (Barnes & Greenebaum, 2007).

Can exogenous "whole body" inputs of thermal energy via microwaves below thresholds of perception produce long-term effects in humans?

There remain open questions about the limits of human thermoregulation (i.e., what does the human body do when provided with this "extra" thermal energy) and thermoperception (i.e., at what threshold and how does the human body perceive this "extra" thermal energy), especially for very gradual heat inputs over very long periods of time (months) and during periods where conscious detection of thermal excursions may be impaired (e.g., during sleep).

Adair et al. (2005) (and references therein) carried out a series of trials in which humans were bulk irradiated at frequencies of 100, 200, 450, and 2450 MHz at specific fluxes. At these frequencies, subjects were subjected to three fluxes (9, 12, and 15 $\mathrm{mW/cm^2}$) at a whole body average normalized specific absorption rate of 0.045 W/kg at each of three ambient temperatures, $T_\mathrm{a} = 24, 28$, and 31°C. We note, as a metric, that typical human metabolism generates heat at a rate of $\sim 0.5 - 1$ W/kg. The study included control groups that had no RF exposure. Measured responses included esophageal and skin temperatures, metabolic rate, local sweat rate, and local skin blood flow. Derived measures included heart rate, respiration rate, and total evaporative water loss.

Several conclusions from this study are noteworthy. First, subjects thermoregulated efficiently through increased heat-loss responses, particularly sweating. Second, they confirmed that RF energy at 100 and 220 MHz was absorbed into deep tissues of the body rather than on the body surface. As a result, there was an absence of the thermal sensation that occurs at frequencies at and above 2 GHz, which are derived from stimulation of temperature-sensitive neurons in the skin. Nevertheless, thermoregulatory responses were generated owing to the presence of similar temperature-sensitive neurons in the brainstem, spinal cord, and elsewhere in the central nervous system. The authors concluded that "The rapid mobilization of sweating observed during the first minute of a 220 MHz RF exposure at 15 $\mathrm{mW/cm^2}$ in all tests ... can only be understood in terms of the thermal stimulation of temperature sensitive neurons residing in critical regions of the brainstem and spinal cord." They further noted that "Calculations of localized RF energy deposition in the body,

through FDTD [Finite Difference Time Domain] modeling, indicate that there is especially high power absorption (SAR > 1.0 W/kg) in the third and fourth ventricles, the medial preoptic/anterior hypothalamic nuclei, and the cerebrospinal fluid bathing these regions." The observation that bulk heating, well below thresholds required for tissue damage (see below), can trigger hypothalamic nuclei is intriguing.

We conclude from these studies that, at flux levels of interest to the incidents, the main effect in this frequency range (< 500 MHz) is a trigger of the thermoregulation response in humans, inconsistent with direct causality for the reported incidents **(Finding 5)**.

6.5.2 The 500 MHz > f > 30 GHz range

This regime has been modeled and studied extensively (see, e.g., Foster et al. 1978, 1998; Foster 2000; Foster et al. 2010, 2018; Gowrishankar et al. 2004 and references therein). We find the set of experiments on human skin by Blick et al. (1997) and a subsequent modeling effort on those data by Riu et al. (1997) particularly useful.

At these frequencies, most of the energy is deposited within the first few millimeters of skin. Between 500 MHz and $\sim$ 3 GHz, the time constant for thermal conduction in tissue is greater than that for thermal convection of heat from blood flow, so convection dominates. The convection time constant is frequency-independent and of order 150 s (Foster et al., 1998). Above 2-3 GHz, the dominant transport mechanism for acute effects is thermal conduction of tissue, with a time constant ranging from 15 s at 10 GHz to 0.6 s at 35 GHz (Foster et al., 1998). Above 30 GHz, energy penetration is so shallow that it is effectively equivalent to pure surface heating.

The acute perception of warmth is produced by nerve endings within the first 0.6 mm of the skin and are sensitive to changes in temperature as small as $0.06 - 0.08°$C (Riu et al., 1997; Barnes & Greenebaum, 2007). For acute application of microwave energy, there is consensus in the literature on sensation and pain thresholds. Table 1, reproduced from Blick et al. (1997), presents experimentally determined human skin warmth sensation thresholds as well as the tissue penetration depth for different carrier frequencies. These

levels represent conservative values; they were determined by subjects who were aware that there was incident radiation in a controlled environment and were asked to detect sensations. In other words, higher power levels might be required for thermal perception in real scenarios where subjects are not aware energy is incident on their skin. The threshold for pain is much higher than for thermal sensation, occurring at about 46°C (Barnes & Greenebaum, 2007). Given the sensitivity of human skin to temperature changes, dermal warmth sensation thresholds provide an upper bound for the *average* flux of any incident signal for which skin warmth was not detected.

Can microwave energy modulate the activity of peripheral nerves, especially those that innervate the head, without producing sensations of warmth or pain?

Given the relative absence of reports of warmth or heat-induced pain and the fact that for frequencies above 3 GHz, the penetration depth is $\ll 1$ cm, it is natural to ask whether microwave energy can stimulate or modulate the activity of *peripheral* nerves, especially those that innervate the head (and potentially could lead to vestibular symptoms), without producing sensations of warmth or pain. We judge that this is improbable because modulating peripheral nerve activity thermally requires local temperature excursions (at the nerve depth) of 6-10°C (see below) and because microwave energy deposition at the surface will be at least the same as (for penetrant frequencies) or much higher than (for low penetration depth frequencies) at nerve depth. This would inevitably cause temperature excursions at the skin surface that would be perceived.

The fact that microwave radiation could produce responses in peripheral nerves in vivo was demonstrated 60 years ago in a seminal paper by McAfee (1962). In experiments on cats, dogs, rabbits and rats, that work demonstrated that "heating afferent peripheral fibers within exposed peripheral nerve trunks or heating cutaneous nerves lying within the tela subcutania by 3-cm microwave irradiation, infrared radiation, or by a warm-water thermode (a type of temperature sensor) results in a nociceptive response at corresponding temperatures, indicating that the response derives from thermal stimulation of these nerves

Table 1: Dermal Warmth Detection Thresholds as a Function of Frequency

Frequency (GHz)	Threshold (mW/cm^2)	Tissue Penetration Depth (mm)
2.45	63.1 ± 6.7	32
7.5	19.5 ± 2.9	6.3
10	19.6 ± 2.9	3.9
35	8.8 ± 1.3	0.8
94	4.5 ± 0.6	0.4
1.3×10^5	5.34 ± 1.1	< 0.1

.... The response occurs abruptly when a temperature averaging 46°C is reached at the nerve" (McAfee, 1962). The nerves irradiated in these experiments include radial, sciatic or trigeminal nerves. The microwave power levels incident on the animals were adjusted such that they would produce a subcutaneous temperature rise to about 45°C within 5 min. This elicited a wide range of responses consistent with pain response, including respiratory hyperpnea, pupil dilation, changes in blood pressure and pulse rate, as well as skin and visceral vasoconstriction.

The authors opine that, "...because the response to thermal stimulation may be maintained for some time..., adrenal medullary secretions may elicit any one or more of many possible secondary effects. Secretion of adrenocorticotropin (ACTH) may occur, resulting in increased elaboration of adrenal cortical hormones. Changes in the composition of the blood which characterize the stress response are expected to follow." Lastly, they observed that "...thermode heating and microwave irradiation does not elicit the spontaneous high-voltage activity associated with myelenated fibers" consistent with earlier work that "only the unmyelenated or thinly myelenated afferent sensory fibers exhibit spontaneous activity at these temperatures."

One interpretation of these results is that microwave heating produced increased firing rates in peripheral nerves, specifically those involved in nociception. That is, the microwave energy produced unpleasant sensations, ostensibly by triggering some sense of pain.

These and subsequent studies also concluded that, again, due to the low penetration depth in tissue for carrier frequencies above 3 GHz, very little energy would penetrate into

the central nervous system and, thus, the cause of the effects seen on the animals would need be restricted to peripheral structures; only minute fluxes of radiation could possibly reach the deeper structures of the central nervous system. McAffee (1971) also noted that it is the neurons themselves that respond to the specific temperature and produce reflex behavior, as opposed to the terminal end organs. The resulting reflexes and responses produced by thermal stimulation at 45°C of peripheral nerves is called nociceptive because of the unpleasant stimulations it produces. It is important to note that, for short durations, reaching a temperature of 45°C at the nerve does not cause tissue damage. McAffee (1971) estimates the flux at the skin to be of order $20 - 40$ mW/cm^2, albeit with a great degree of uncertainty. We note that these fluxes applied over 5 mins, required to reach the effect threshold of 45°C in the experiments, is equivalent to a total dose of $6 - 12$ J/cm^2.

Taken together, then, there is evidence of increased firing rates in peripheral nerves due to continuous wave microwave radiation. The literature cited would suggest that humans subjected to this energy would not show lesions but would experience sensations of heat and/or pain.

Moving from continuous waves to pulsed radiation, we review findings from a recent study that provides the energy doses and temperature excursions required to fire a peripheral nerve thermally. Using optical lasers at different frequencies, Wells et al. (2007) stimulated rat sciatic nerves in vivo and showed that direct neural activation with pulsed laser light is induced by a thermal transient. They further quantified the spatial and temporal nature of this required temperature rise, including a measured surface temperature change required for stimulation of the peripheral nerve (6°C–10°C). The temperature rise results in direct or indirect activation of transmembrane ion channels causing action potential generation and occurs at dosages of 0.3-0.4 J/cm^2 for 5 μs, 350 μs, 1 ms, 3 ms and 5 ms pulses, corresponding to fluxes during a pulse of 60 kW/cm^2, 857 W/cm^2, 300 W/cm^2, 100 W/cm^2, and 60 W/cm^2, respectively, at 0.3 J/cm^2.

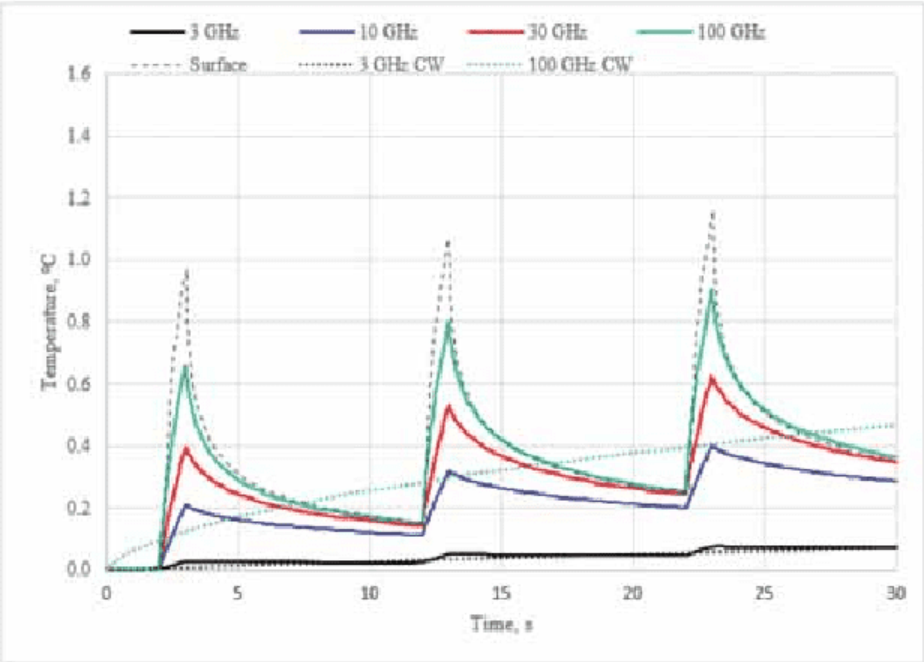

Figure 6.6: From Foster et al. (2018). "Skin surface temperature increase in 1D baseline model to pulse train at varying carrier frequencies in GHz and for the surface heating model for comparison. Pulses are 1 s duration, repeated at 1/10 Hz, peak pulse intensity 1000 W/m2. Also shown (dotted lines) are the increases in surface temperature from exposure to continuous-wave radiation at the same time-averaged power density at 3 and 100 GHz. The energy transmission coefficient was assumed to be 1 at all frequencies to facilitate comparisons of responses."

The average power is lower depending on the pulse repetition frequency. We note that these doses are about 10,000 times those reported to produce thermoacoustic phenomena (the Frey effect), which occur at $\approx 40\ \mu J/cm^2$ (see Section 6.5.4).[1]

What we can conclude by linking this with the earlier work by McAffee (1971) and others is that by modulating a microwave carrier wave (preferably at or above 1 GHz, so that the energy is only absorbed just under the skin in the peripheral nerves), it is feasible to produce a pulse that could thermally fire an action potential in a peripheral nerve. Although Wells et al. (2007) argue that thermally-induced firing (with a laser) can be repeated up to about 5 Hz PRF, this is misleading. As stated earlier, there are two thermal time constants

[1]Some care must be taken when comparing the optical and microwave powers, given the different absorption and scattering phenomena at work. The authors of the study estimate that, for 2.12 μm wavelengths, about 68% of the light incident on the peripheral nerve penetrated, entered the axon, and was converted to heat. Given this, order of magnitude comparisons appear justifiable.

for tissue near the skin: a frequency-dependent conduction time constant and a convection time constant. The convection time constant is on order of 150 s and the conduction time constant ranges from $2000 - 0.6$ s for $1 - 35$ GHz, respectively (Foster et al., 1998). For energy above 2–3 GHz in frequency, the conduction time constant dominates. Although a pulse would produce a very rapid temperature rise, the temperature decay (i.e., the time it takes for a section of tissue to cool down to the local steady-state temperature) would be set by a multiple of the conduction time constant. Thus, the fastest one could repeat a thermal firing event without slowly raising the steady-state temperature of the tissue is likely about 5τ, or one firing event every 3 seconds. This is illustrated in Figure 6.6 (Foster et al., 2018).

This time interval between pulses is much slower than the endogenous firing rates of most peripheral nerves. Just as importantly, because microwave energy deposition at the surface will be at least the same (for penetrant frequencies) or exponentially higher (for low penetration depth frequencies), pulsing in this way would inevitably cause temperature excursions at the skin surface that would be perceived. We thus conclude that pulsed-microwave induced thermal firing of peripheral nerves at fast enough rates to produce significant biological effects and explain the direct sensations and symptoms associated with anomalous health incidents is unlikely, especially in light of the absence of reports of thermal sensations **(Finding 5)**.

6.5.3 Requirement for RF-induced Traumatic Brain Injury

We conclude that radio-frequency energy is unlikely to produce Traumatic Brain Injury (TBI) without concomitant sensations of heat and or pain. In addition, we see that RF energy is unlikely to produce lesions in tissue unless power levels are sufficient to raise the temperature of tissue to $> 45°C$ (Barnes & Greenebaum, 2007). For RF energy with penetration depths on order of the centimeters to meters, such power levels would lead to general hyperthermia, because, in order to raise the temperature of a putative target such as a location in the central nervous system, the energy would increase the temperature of most of the body or head by similar amounts. For RF energy with penetration depth less than

1 centimeter, the elevation of an internal tissue location to 45°C would necessitate a much larger increase in skin surface temperature, given the exponential decay in power density as a function of tissue depth. This would produce sensations of warmth, at minimum, and pain, in all likelihood **(Finding 5)**.

6.5.4 Thermoacoustic phenomena and the "Frey Effect"

Acute auditory effects of microwave exposure have also been documented, and are often referred to as the "Frey effect." Essentially, an abrupt (typically $t < 100\mu$sec) but very low level heating of the tissue, with $\Delta T \ll 1°$C, by the absorbed RF raises the pressure in the tissue, and this launches a sound wave within parts of the auditory system inside the human head. The effect is called "thermoelastic" or "thermoacoustic". At RF frequencies $f \gtrsim 1$ GHz, the absorption length of the electromagnetic energy of the order of a centimeter, as shown in Figure 6.1, whereas that of the sound is typically much longer. As a result, this effect can be used to image the brain or other tissues acoustically (Jiang, 2020). This auditory phenomenon, characterized by the sound of clicking, buzzing, knocking, or chirping in the head, has no known adverse health consequences in and of itself (Frey & Etchert, 1985; Barnes & Greenebaum, 2007; Elder & Chou, 2003).

The Frey effect has been reported at frequencies ranging from 2.4 MHz to 10 GHz, although the upper end of this range is not readily reproducible. For example, 8,900 MHz was not effective at an average flux of 25 mW/cm^2 and peak flux of 25,000 mW/cm^2 (Frey, 1962). At 216 MHz, the threshold in average flux was found to be 4 mW/cm^2 and the peak flux to be 670 mW/cm^2 (Frey, 1963). At the lowest effective frequencies (2.4–170 MHz) reported in the literature, the peak flux thresholds were up to 9,000 mW/cm^2 (Röschmann, 1991). The lowest threshold value expressed in units of average incident flux is 0.001 mW/cm^2 (Cain & Rissmann, 1978); this value was due to the low PRF of only 0.5/s.

Overall, the hearing phenomenon has been shown to depend on the energy in a single pulse and not on average power density. Guy et al. (1975) found that the threshold for RF hearing of pulsed 2450 MHz fields was related to an energy density of 40 μJ/cm^2 per pulse, or energy absorption per pulse of 16 uJ/g, regardless of the peak flux, for pulses less than

32 ms. That amount of incident energy is expected to increase the tissue temperature by 5×10^{-6} °C (Elder & Chou, 2003).

One other important characteristic of the auditory response to microwaves is that the waves must be modulated, generally in the 7 to 10 kHz range (Chou et al. 1977, 1982b; Watanabe et al. 2000), or 8 to 15 kHz (Lin 1977a,c). If the sounds reported in the incidents arise from the Frey effect, this limits the PRF that is required in the incident radiation. However, we repeat here that any directly **recorded** audio by an external electronic microphone cannot be explained by the thermoacoustic effect.

The thermoacoustic effect has been calculated theoretically (e.g., Gournay 1966) for fluids such as water by starting from the isobaric thermal expansion coefficient

$$\beta \equiv \frac{1}{V}\left(\frac{\partial V}{\partial T}\right)_p \approx 3.6 \times 10^{-4}\,\mathrm{K}^{-1} \quad \text{at } 37°\,\mathrm{C}. \tag{6-5}$$

For a sufficiently short pulse $\Delta t < h/c_\mathrm{s}$, however, where h is the RF absorption length and c_s is the speed of sound in the fluid, it is not directly the expansion coefficient (6-5) at constant pressure that is relevant, but rather

$$\left(\frac{\partial P}{\partial T}\right)_V = \frac{\alpha}{\kappa} \approx 8 \times 10^5\,\mathrm{Pa\,K}^{-1} \quad \text{at } 37°\,\mathrm{C} \tag{6-6}$$

because the affected layer is heated at constant volume, and only later expands. (Here $\kappa = -(\partial \ln V/\partial P)_T \approx 4 \times 10^{-10}\,\mathrm{Pa}^{-1}$ is the isothermal compressibility). For example, from Fig. 6.1, $h \approx 2\,\mathrm{cm}$ at $f_\mathrm{RF} = 2.45\,\mathrm{GHz}$, and so $h/c_\mathrm{s} \approx 14\,\mu\mathrm{s}$ in water ($c_\mathrm{s} \approx 1520\,\mathrm{m\,s}^{-1}$). Indeed, it has been verified experimentally that the pressure amplitude of sound produced depends only on the energy per pulse, not the peak RF intensity (power/area), in the limit of short pulses (Foster & Finch, 1974).

Since the reference pressure level for sound is $2 \times 10^{-5}\,\mathrm{Pa}$, and ordinary conversation is typically a thousand times this (60 dB), the acoustic pressure is exquisitely sensitive to small temperature changes[2]. Thus for example, a 60 dB pressure level (0.02 Pa) would require a temperature rise of no more than 3×10^{-8} °C. Of course, a train of N pulses would heat the tissue N times as much if the length of the pulse train were short compared to the thermal diffusion time, which is $\approx 11(h/\mathrm{cm})^2$ minutes in water.

[2]This is the reference level for sound in air; in water, 1 μPa is often used instead.

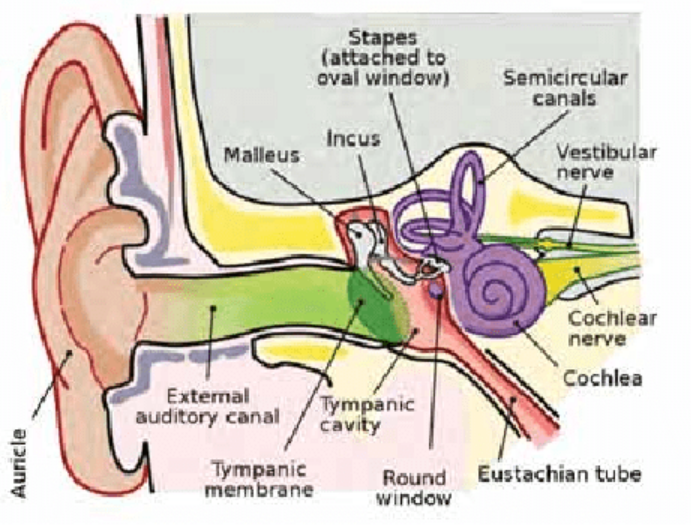

Figure 6.7: (U) The human auditory system. *Credit:* Wikipedia Commons.

Based on these calculations, we find that at tolerable levels of audibility, the thermoelastic excitation of sound waves in the brain, due to small acoustic pressure established for the Frey effect, cannot produce brain damage by mechanical means **(Finding 5)**.

The ability of a mechanical disturbance to cause damage to the brain is often quantified by the stress or strain that it produces (Wright & Ramesh, 2012). For example, according to this reference, a 50% probability of mild traumatic brain injury (TBI) is induced by a shear stress of 7.8 kPa; and a 50% probability of mild diffuse axonal injury (DAI) with a strain 0.2-0.3, corresponding to a shear stress of 26 kPa. Now the units of shear stress are the same as those of pressure, whereas even at the threshold of pain (140 dB), the pressure of audible sound is only 0.2 kPa. The strain associated with a sound wave of amplitude δp is $\delta p/\rho c_s^2$ (which is dimensionless), and $\rho c_s^2 \approx 2.3\,\mathrm{GPa}$ in water at 37 °C. In other words, at levels that are audible but not painful, the stresses and strains associated with sound waves produced in the brain by the Frey effect are orders of magnitude lower than those that have been associated with brain injury. This leads to our finding above.

A few caveats are in order here:

1. According to Howard (2021, `http://www.tulane.edu/~h0Ward/BrLg/AuditoryTransduction.html`) the mechanical advantage of the connection from the tympanum (eardrum) to

the cochlea via the ossicles (malieus, incus, & stapes in Fig. 6.7), is such that a given sound pressure level (SPL) in the air is amplified by a factor ≈ 22 in the inner ear. We have not been able to find a similar factor for the acoustic coupling from the other side, i.e., from the brain to the cochlea, which presumably would go primarily via the internal auditory meatus, the channel through which the cochlear and vestibular nerves connect the inner ear to the brain. As a result, when comparing sound waves excited in the brain to those from external sources, one should subtract at least $20\log_{10}(22) \approx 27\,\mathrm{dB}$ to account for the transduction of pressure by the auditory system. Comparing the power levels and pulse durations of their experiments with water (Foster & Finch, 1974) to those of Frey & Messenger (1973), Foster & Finch (1974) estimate that the threshold of audibility corresponds to an SPL $\approx 90\,\mathrm{dB}$ ($0.65\,\mathrm{Pa}$) in the fluid. They further estimate that this corresponds (at least at $f_{\mathrm{RF}} = 2.45\,\mathrm{GHz}$) to an incident energy in microwaves $\approx 50\,\mu\mathrm{J}$ per pulse, of which 60% would be reflected by the skull, and the remainder absorbed in the brain. The corresponding peak temperature increase per pulse would thus be

$$2 \times 0.65\,\mathrm{Pa}/(\partial P/\partial T)_V \approx 1.7\,\mu\mathrm{K}\,,$$

in which the factor of 2 has to do with the acoustic waveform (see below). This is far too small to cause direct thermal damage.

2. Most of the acoustic power produced is at frequencies $f_{\mathrm{s}} \lesssim c_{\mathrm{s}}/2\pi h$, where, again, h refers to the RF absorption length. At $f_{\mathrm{RF}} = 2.45\,\mathrm{GHz}$, where $h \approx 2\,\mathrm{cm}$, this is about 12 kHz. That is to say, a single short RF pulse should produce a very broadband acoustic signal with cutoff $\sim f_{\mathrm{s}}$. If much higher RF frequencies were used, the characteristic acoustic frequency would lie outside the range of human hearing. Therefore, a given *perceived* sound level would correspond to a larger peak pressure in the brain for the same incident microwave power (or energy per pulse). For example, at $f_{\mathrm{RF}} = 100\,\mathrm{GHz}$, $h \approx 0.3\,\mathrm{mm}$ and $f_{\mathrm{s}} \approx 1\,\mathrm{MHz}$. Furthermore, the ratio of the perceived SPL to the actual SPL in the brain is then sensitive to boundary conditions: linearly with f_{audible}/f_s for a rigid boundary condition (as may be appropriate at a few GHz where h is larger than the thickness of the skull), but as $(f_{\mathrm{audible}}/f_s)^2$ if the boundary is free (as may be appropriate at 100 GHz, where h is less than the thickness of the skin

covering the skull). This has to do with the symmetries of the acoustic waveforms produced in these two cases (see Appendix C). Thus, at $f_{\mathrm{RF}} \gtrsim 100\,\mathrm{GHz}$, audible clicks might correspond to acoustic stress levels in the brain of a few kPa, which gets somewhat closer to the thresholds quoted above for mild TBI or DAI. However, these pulses would be perceived on the skin well before they are perceived as acoustic sensations.

We provide additional mathematical details in Appendix C.

6.5.5 Are there non-thermal effects of microwaves on tissue?

There is a vast "grey literature" on effects that are a) demonstrated only in vitro or in small animals, b) inferred from epidemiological studies and reports and/or c) purported to demonstrate so-called non-thermal effects. The current consensus is that most such findings are either not reproducible, not easily scalable to humans, and/or not relevant to effects in humans. Chapter 6 of Lin & Michaelson (1987) provides a valuable resource in this area.

A number of responses have been reported that appear not to be accompanied by changes in temperature. However, in JASON's conclusion based on a review of the literature, these are all most likely caused by the deposition of thermal energy into tissue. The confusion arises in part because the body thermo-regulates, in part due to heterogeneity of tissue, in part due to the presence of tissues that have low densities of pain receptors, and in part due to poor experimental design. This discrepancy in the use of the term "non-thermal" also arises when reviewing the extensive Cold War-era literature originating in Soviet bloc countries. An early definition of non-thermal effects from that literature is based on effects not associated with a measurable local or whole organism temperature rise from an equilibrated baseline. Most Western investigators use the term thermal in a somewhat different sense, allowing for the fact that an organism can be affected thermally without a demonstrable temperature rise. The temperature rise, in fact, is taken to mean that "the functional reserves of the organism for maintaining homeostasis have been exceeded in some manner, and thermal regulation has been instituted" (Lin & Michaelson, 1987).

It has been proposed that non-thermal effects might be due to RF interaction with cell membranes, especially those of neurons, perhaps producing depolarization or the opening of ion channels. This is extremely unlikely. (See Schwan 1971 for a lucid treatment). In brief, the electric field across a cell membrane arising from the cell's normal resting potential is about 500 kV/cm. Based on the electrical characteristics of cell membranes, the electric fields produced by RF energy above 100 MHz across these membranes are between a factor of 10^5 and 10^6 smaller than 500 kV/cm. Put bluntly: "According to all modern concepts of neurophysiology about excitation, this just cannot stimulate nerves" (Schwan, 1971).

The possibility still remains that future studies will reveal reproducible *non-linear* effects arising upon excitation of tissue by radio frequency energy in the relevant frequency range. If these exist, they would occur at very high field strengths. How high these fields need to be is unknown. We can take as a guess the field strength found across cell membranes (500 kV/cm or more), although note that such fields are relatively static, while the fields discussed here are sinusoidal (or at least alternating) at GHz frequencies.

Such field strengths would require very high fluxes. In order to apply such fluxes without thermal sensation or damage, fast pulses would be required. By way of example, consider a set of conventional RF pulses studied by Foster et al. (2018), shown in Figure 6.8.

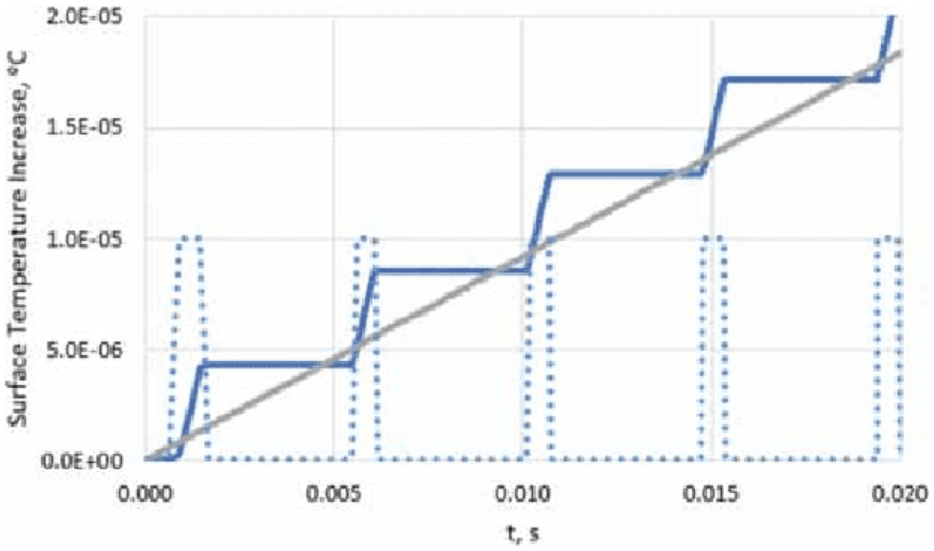

Figure 6.8: (from Foster et al. (2018))

The pulses in Figure 6.8 have a 1.9 GHz carrier frequency, a duration of $\tau_p = 0.57$ ms, a flux of of 800 W/m^2 (for a dose of 56 J/m^2), repeated 217 times per second. Each pulse produces a temperature rise of less than 5x10^{-6} °C.

The radiation flux F, in units of W/m^2, is related to the electric field, E (units V/m) as:

$$F = \frac{E^2}{\eta}, \tag{6-7}$$

where η is the wave impedance, (in units of Ohms or Ω), of the tissue:

$$\eta = \frac{377}{\epsilon_r^{1/2}}\ \Omega \approx 180\ \Omega, \tag{6-8}$$

where we calculated the permittivity using data from Gabriel et al. (1996) and the equation on page 44 of Hurt (1996) as cited in Table 2 of Blick et al. (1997). Using the flux of 800 W/m^2, this gives for the electric field,

$$E = (F\eta)^{1/2} = 380\ \text{V/m}. \tag{6-9}$$

Assuming all of the energy in the 800 W/m^2 entered the tissue, the resultant field at the surface of the skin would be 380 V/m or 3.8 V/cm. A field of 500 kV/cm is roughly 10^5 times larger than this value; achieving the larger field strength would require a 10^{10} larger incident flux. In order to maintain the same total energy dose per pulse (and thus keep the temperature rise the same), the pulse width would have to decrease by 10^{10}, going from 0.57 ms to 57 femtoseconds. For reasons discussed in Section 6.3, such a pulse width is below the sensation threshold and spreads out the signal over a very large bandwidth. We can approach this from the opposite direction: if we assume a 1 nanosecond pulse, a 10 GHz carrier frequency, and ignore penetration depth problems, what is the maximum achievable field strength for the same energy dose? Such a pulse would be 570,000 times shorter in duration than the 0.57 ms pulse above, allowing for a 570,000 times higher flux:

$$E = (F\eta)^{1/2} = (570000 \times 800\text{W/m}^2 \times 180\ \Omega)^{1/2} = 2.8\ \text{kV/m}. \tag{6-10}$$

This is still a factor of ≈ 200 below the field strength across cell membranes. From the above, we conclude that while it may be prudent to provide detection for very short pulses for completeness **(Finding 7, Recommendation 7)**, JASON finds it highly unlikely that

realistic pulses can produce cell membrane-level electric fields within tissue without also producing thermal sensation at the surface of the skin **(Finding 5)**.

6.6 Examples of RF Exposure

We end this section by considering instructive examples of health effects from documented and/or accidental exposures to RF waves, such as exposures to radars and directed RF signals. These incidents provide an experimental bound on the health effects due to RF at various average and peak powers. They are also illuminating in terms of health problems that may arise due to the psychological response to these incidents.

6.6.1 The Moscow Embassy Lilienfeld Study

We begin by recalling that between 1953 and 1976, beamed microwave energy was directed at the United States Embassy in Moscow, perhaps as part of an espionage attempt. Due to widespread, publicized concern over health effects of the exposure (Brodeur, 1977), researchers at the Department of Epidemiology at Johns Hopkins University, led by Abraham Lilienfeld, conducted an epidemiological study focused on the staff of the US embassy in Moscow and their families (Lilienfeld et al., 1978). An excellent and balanced review of this report and the subsequent, sometimes differing, opinions produced can be found in Elwood (2012). Because the frequencies and power levels are within ranges considered here, we cite from Elwood below.

"From 1953 to May 1975, the microwave beam came from a source in a Soviet apartment building about 100 m west of the 10-floor embassy building, affecting the west facade of the central building, with highest intensities between the third and eighth floors. The frequency was from 2.5 to 4.0 GHz and maximum exposures are given as up to 5 μW/cm^2, 9 hours per day" (Lilienfeld et al., 1978). However, Appendix 11 notes that individual exposures would have in general been much less than the maximums because of movement of personnel away from windows and to other rooms, as well as the fact that some hours of signal operation were at night (Elwood, 2012). The average fluxes noted in the Lilienfeld report are significantly lower than accepted IEEE/ANSI power density standards for cell

phones (IEEE Standards, 2019) and the radiation fluxes considered earlier when discussing thermal effects.

The conclusion of the Lilienfeld report was that, epidemiologically, there were no demonstrable health effects. Summarized more precisely in Elwood (2012): "literally hundreds of comparisons were made" and only two differences stood out from the medical record review, the increased rate of protozoal infections in Moscow male employees, and the slightly higher frequencies of the most common kinds of health conditions in the Moscow group. There were greater differences in the self-reported data on the questionnaires, but there were no conditions which were more common in Moscow and showed a relationship to estimated microwave exposure or length of service within the Moscow group..... The overall conclusion of the investigators (page 246) was: "To summarize, with very few exceptions, an exhaustive comparison of the health status of the state and non-state department employees who had served in Moscow with those who had served in other Eastern European posts during the same period of time revealed **no differences in health status as indicated by their mortality experience and a variety of morbidity measures.** No convincing evidence was discovered that would directly implicate the exposure to microwave radiation experienced by the employees at the Moscow embassy in the causation of any adverse health effects as of the time of this analysis."

6.6.2 The Norway Ship Radar Exposure

On August 24, 2012 a United States Navy destroyer passed within 70-100 meters of a Norwegian Coast Guard ship. The ships were in close proximity for approximately 7 minutes. During this period, the Norwegian Coast Guard ship experienced disturbances to instrumentation and electronics, including monitors and navigation instruments. Fire alarms were also set off. The Norwegian crew deduced that the U.S. destroyer had accidentally left its radar systems on while it passed near them and contacted the U.S. ship. The destroyer then switched off its radar. Moen et al. (2013) provide a summary of the event, estimates of exposure and a summary of the medical follow up and findings.

Of the 66 Norwegian crew onboard, 14 people were on the bridge or outside on the

deck. From November 2021 to January 2013, 29 people (including the 14 on bridge or on deck) were referred to the Hospital Department of Occupational Medicine in Bergen, Norway out of concern that the exposure had caused health problems. The cognizant medical staff utilized a subjective health complaint questionnaire at intake. A battery of clinical tests were performed, including clinical examination of blood pressure, auscultation of the chest, observation of the skin, palpation of the abdomen, neurological status and mini mental state exam, eye examinations by an ophthalmologist, as well as blood sample analysis for haemoglobin level, blood count, red cell volume, liver function (alanine transaminase), renal function (urea, creatinine), thyroid hormones (thyrotropin, thyroxine and triiodothyronine) and immunoglobulines (Immunoglobulines A, E, G and M). Among males, luteinizing hormone, follicle-stimulating hormone, testosterone, sex hormone-binding globuline, prolactin and oestradiol were analyzed. The conclusion of this study was that "the crew was found to be a healthy group of persons, with no signs of skin affection, no cataract, no general health problems, no affection of the nervous system or any abnormal blood analyses. The crew had clearly less subjective health complaints than what we find in a normal population, and the symptoms had evidently disappeared" (Moen et al., 2013).

This incident is of special interest because a) the exposure was confirmed and, based on incident reports and fundamental physics, likely of high enough power density to produce sensations of warmth on the skin; b) it resulted in documented rumor-driven health concerns and early self-reports of a variety of heterogeneous health problems by the crew (including crew who were inside the ship and could not have been exposed to radar energy); c) there was no evidence of lesions or long-term physical damage despite the high intensity exposure, even to sensitive areas including eyes and genitals; d) clinical evaluation led to a strong conclusion that the exposed population was healthy 6 months after the event. Additional detail on the event is summarized below.

Estimates of exposure

Moen et al. (2013) estimated from a review of the open literature that the radar that produced the exposure was a SPY-1D(V) and operated at a frequency of 3-4 GHz, 6MW peak power and 58 kW average power (i.e., 1% duty cycle). Additionally, the antenna gain is estimated to be 9300. The average radiation flux at the exposure site can be obtained

from

$$F = G\frac{P}{4\pi r^2} \approx 6,000\text{W/m}^2 = 600 \text{ mW/cm}^2, \tag{6-11}$$

where G is the antenna gain of 9,300, P is the average power of 58 kW and r is the distance of the exposure site from the antenna, which we take to be 85 meters as the median of the range reported ($70-100$ meters.) The corresponding peak power density is 60 W/cm^2. The authors further surmise, and JASON agrees, that the radar was likely performing a search pattern over the space swept (as opposed to maintaining a steady beam targeted at the Norwegian ship), so that the total dwell time at any spot would likely be of order seconds and thus the average exposure over several minutes at any one location would be significantly lower than the value calculated above (perhaps 10–100 times less than the value calculated above.) At 2.45 GHz, the reported threshold for warmth sensation is 63.1 ± 6.7 mW/cm^2 (see Table 1), which is 1/10 the value calculated above. We would thus expect crew members on deck (i.e., not inside the ship) would experience sensations of warmth lasting several seconds to tens of seconds.

Incident reports

"Five of the crew on the bridge and two of the crew standing outside on the deck on the same side of the ship where the American ship passed, felt a slight sensation of warmth on the face during the radar incident. This sensation lasted for a very short period, not more than seconds or minutes. Two of those on the bridge also felt a mild heat sensation on the left arm; this side of their body was turned towards the passing ship....Two of the crew were sitting inside the ship when the fire alarm went off and ran out on the deck to perform their duties during the alarms. They described a slight warm sensation on the face and on their arms" (Moen et al., 2013). No burns were reported. A physician on board the ship reported no signs of skin, eye problems, or health issues of any kind for anyone on board the ship that day and the crew reported no complaints.

Reported health issues

In the days that followed, crew members grew concerned about health effects of exposure.

Some kept health diaries. The ship was eventually diverted to Tromso on September 4, where crew were examined. No signs of disease or damage were found. At this point, a meeting was held where the crew was informed of the exposure. Subsequent to this, the ship set sail with a physician on board. During the journey, the crew began to report a wide variety of symptoms to the physician on board. "At this time the crew reported problems with headache (43%), fatigue (26%), sweating (20%), pain/burning sensation on the skin (15%), and impaired/disturbed vision (7%). Furthermore, they reported strange feelings in the ear, hearing loss, pain in testicles, nose-bleeding, nausea, and chest discomfort. The onset of symptoms showed considerable variation. One week later the physician wrote in a note that: "At the follow-up a week after the original survey, only 1 individual has health problems. The remaining crew has no problems or complaints." (Moen et al., 2013)

Subsequently, however, crew members began to report health problems again: "They all described that they talked a lot about the radar incident on board, and they wanted to know more about the exposure and the possible health effects. They all claimed that the information they were given was scarce, and that they became worried. Some of them described that they thought their symptoms developed because they discussed different possible diseases that could be caused by radiation, for instance cancer of the brain and infertility. The weeks passed and members of the crew still complained about the occurrence of different symptoms. In a note from November, more than 2 months after the incident, a marine medical officer reported to have a follow-up examination of 33 persons from the ship." As a result of this, "the crew were crew was referred for examination to the hospital in Bergen at the end of November 2012." (Moen et al., 2013) As summarized above, the subsequent clinical study found the population affected to be healthy approximately 6 months after the event.

6.7 Conclusions on the RF hypotheses

We considered directed RF energy as a possible mechanism for causing harm to individuals from a distance. We explored the frequency range, peak and average flux levels, pulse durations, and pulse repetition frequency that may be utilized toward that end evaluated

these characteristics based on multiple physiological and environmental considerations. We summarize our findings on the RF hypotheses below as well as visually in Figure 6.9.

For the carrier frequency range of interest, the main limits arise from considering the penetration depth of RF waves through the human body. Frequencies between 500 MHz and 30 GHz can penetrate below the skin and produce sensations of heat and pain. Lower frequencies can deliver thermal energy to the entire body.

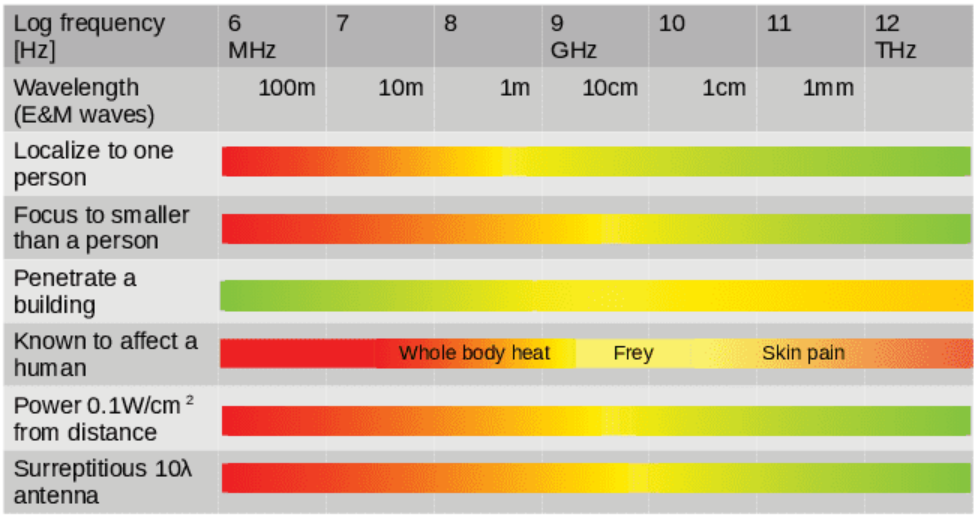

Figure 6.9: Combined assessment of RF radiation as a potential cause for embassy incidents on all relevant criteria.

For the flux range of interest, we eliminated high peak flux levels, $F_{\mathrm{p}} \gtrsim 30$ W/cm^2, that would cause a disruption of common consumer electronics devices, and low peak flux levels, $F_{\mathrm{p}} \lesssim 1$ W/cm^2, for which there are no documented health effects. We also narrowed down the range of average flux levels of interest to $20-100$ mW/cm^2 based on common daily exposures on the low end and avoiding a heating sensation on the high end of this range. However, we also note that some of these statements are based on "routine" pulse durations ($\tau_{\mathrm{p}} > 10$ ns) and, therefore, recommend a conservative approach for monitoring and allowing higher flux levels to be considered. On the lower end, we caution that routine RF clutter rises toward lower flux levels and would make individual signal detection challenging.

For pulse durations, shorter pulses (e.g., of nanosecond duration) can allow higher voltages to be induced across the cell membrane but *(i)* require higher peak fluxes to deliver the same energy and *(ii)* spread the signal over a wide bandwidth that scales as the inverse of the pulse duration. Because of this, practical considerations render pulse durations that are significantly less than a nanosecond ineffective as a mechanism.

Associating sounds reported during incidents with the "Frey effect" leads to a PRF in the audible range for human hearing. Auditory response has most commonly been elicited in the 7-10 kHz range but experiments span the range from 1Hz to (rarely) 20 kHz. Based on these studies, a PRF in the range of 1-10,000 pps provides an appropriate region of interest.

It is important to repeat here that no immediate or lasting structural damage or adverse health consequences have been reported in the literature for the signal characteristics outlined above. Any documented damage mechanism requires high flux levels that can cause large ($> 5°$C) temperature changes in tissues and nerves, which would unavoidably cause accompanying sensations of heat and pain. This is inconsistent with the reported incidents and symptoms.

We summarize our assessment of RF hypotheses in Figure 6.9. The color coding in the figure corresponds, as before, to the level of consistency between the mechanism at the given frequency and the requirements or effects considered: red denotes inconsistency, yellow denotes marginal consistency, and green denotes possible consistency. The fact that there is no frequency at which the color is green simultaneously across all rows reflects the conclusion above that there is no region in the RF spectrum that is consistent with all of the signs and symptoms reported in the embassy incidents. The region with frequencies between 500 MHz and 30 GHz cannot be conclusively ruled out but appears highly unlikely given the arguments presented in this section **(Finding 5)**. Our conclusion is based on all of the information provided to us in the briefings and the substantial JASON expertise in electromagnetic phenomenon in this frequency range. JASON will continue to engage the US national and DOD labs to ensure we have not missed any work that would impact Finding 5.

We conclude by providing thoughts on future RF surveillance. Given that the current data and analysis significantly limits but cannot conclusively rule out any offensive use of microwave irradiation, it is important to deploy technology that can continuously monitor ambient cm-wave and mm-wave microwave RF. The BlueJay detector suite described in Appendix **??** includes a first-generation monitor, covering the band 1 MHz to 10 GHz, with 55 dB dynamic range of native sensitivity (1 nW to 0.3 mW, but deliberately attenuated in practice by some 40 dB, thus 10 μW to 3 W), and with peak-hold functionality for pulsed radiation down to 100–200 ns. With relatively straightforward circuit and component-level changes, a second-generation RF monitor could extend coverage by a factor of 7 in frequency and 100 in pulse-capture speed (thus $\sim$70 GHz and 1 ns). A suggested path is described in Appendix **?? (Finding 7 and Recommendation 7)**.

7 AN INSTRUCTIVE ANALOG

Before concluding this report with our final remarks, we find it useful to underscore some of the challenges we face with the methodologies applied to the Anomalous Health Incidents in order to identify an underlying cause and to reach conclusions regarding deliberate adversarial action.

In assessing and developing methodologies that might be applied to the current situation with the embassy anomalous health incidents (AHI), we found it informative to apply our reasoning to a somewhat analogous data set. The goal here is to better understand evaluation criteria for hypothesis testing, and the identification of an appropriate comparison/control group. Also, we think it useful to have a parallel data set to which a methodology can be applied, as a way to validate the overall approach.

Of all the U.S. missions overseas, according to Department of State Office of Inspection reports ISP-I-19-14 and ISP-I-19-18, between 2012 and 2017 Colombia and Haiti reported anomalously high rates of vehicular accidents. Some of these accidents had the unfortunate consequence of injury and even death. Table 2 compares this accident data to the AHI situation, in the context of physical injury mechanism, consistent medical effects, and statistical incidence relative to an appropriate baseline rate.

One could postulate that one or more adversaries are intentionally causing accidents involving U.S. embassy vehicles on the streets of Bogota and Port-au-Prince, on the basis of this excess of incidents, and the clear connection of a physical mechanism to a long-lasting adverse health effects. How might this hypothesis be evaluated? We would look for a clear and well-defined physical mechanism, and a causally-associated cluster of medical consequences. Both of those criteria are clearly satisfied in the vehicle accident situation, much less so for AHI.

Another element of the evaluation of the hypothesis is to look at the rate of events compared to the background, and see if there is an excess. There were bound to be two embassies that had accident rates above that of other U.S. missions, and these two were selected on that basis. As a result, the excess of events above the average for U.S. missions

Table 2: (U) A comparison of the reported excess of accidents involving U.S. embassy vehicles in Colombia and Haiti to AHI. If we require a clear physical mechanism and a clear set of medical effects connected to it, any methodology that assigns a likelihood to nefarious intentional action should be applied to both data sets, as a validation mechanism.

	Vehicle Accident Analog	**AHI**
Is there a clear physical process that can lead to the reported lasting health effects?	Y	?
Is there a common physiological/medical outcome consistent with the physical insult?	Y	?
Is the frequency of events larger than seen in other U.S. embassies?	Y	?
Is the frequency of events larger than seen in the domestic US population?	Y	?
Can we conclude that this situation is the consequence of deliberate adversarial action?	?	?

is not significant. A more meaningful comparison might be to compare the accident rate and medical consequences to that of broader background populations. One indicator is the incidence of vehicular fatalities in the U.S., in Colombia, and Haiti. The deaths per year per 100,000 population for those countries are 10.9, 21.3, and 15.5, respectively. But are any of these the right control group? Are there adequate statistics to assess whether the embassy cars collide more often than the typical car in the cities in which they drive?

This analog problem has some lessons we can extract: whatever methodology is applied to the AHI situation to assess the likelihood of intentional adversarial action should also be applied to analogous situations where we think we know the answer, to validate the logical process and reasoning. Second, the definition of one or more appropriate control groups is non-trivial, and requires careful consideration. Finally, beware of selection effects. In this analog problem, it is easy to recognize that the two embassies were chosen due to high accident rates. Other selection biases can be much more subtle and lead to the conclusion of a spurious signal.

8 COMBINED ASSESSMENT AND CONCLUDING REMARKS

We conclude by summarizing our analyses and findings on the embassy health incidents and potential mechanisms that could be responsible for them.

The results drawn from medical and incident data are inconclusive at this time and do not point clearly to the presence of a new medical syndrome associated with AHI or to a single cause underlying them. Instead, currently reported collection of symptoms could result from a combination of structural causes, including those arising from other known illnesses, functional disorders, as well as psychogenic response to stressful events.

Upon evaluation of incident data, JASON concluded that the incidents are highly heterogeneous; approximately 15% of the 200 cases share moderately common characteristics and could potentially fit the working case definition. We used these incident files to arrive at a set of "incident consistency criteria" that we used for evaluating potential causes. These included the physical requirements for operations in the described environments and the sensations and symptoms that have been reported. We also noted sensations and symptoms reported to be absent in order to help establish a fully consistent link between potential mechanisms and outcomes. Similarly, we used the medical data to identify symptoms and delineate symptoms that should and should not be present in conjunction with a mechanism.

Focusing on mechanisms that could deliver energy from a distance, we evaluated acoustic and electromagnetic waves with regard to their potential to produce physiological damage as well as any transient sensations that could be consistent with those reported and precipitate functional disorders. We summarize our combined assessment in Table 3. Color coding indicates the level of consistency between the mechanism and the physical requirements, sensations, or symptoms considered: red denotes inconsistent/highly unlikely, yellow denotes somewhat inconsistent/unlikely, and green denotes possibly consistent/likely.

Based on the fact that no mechanism is green across all columns, JASON judges none of these mechanisms to be the likely cause underlying the health incidents. In particular, acoustic waves at all frequencies, ionizing radiation, electromagnetic waves below 500 MHz

and above 30 GHz can be ruled out with high confidence because there is at least one aspect of generating or delivering that energy, or its effects on human health that is highly inconsistent with the incidents. Pulsed RF waves with carrier frequencies between 500 MHz and 30 GHz cannot be conclusively ruled out but appear unlikely given the "yellow" designation in multiple columns: the radiation flux needed to cause any physiological change in human tissues, nerves, or organs, even temporarily, would have led to a sensation of skin heating or pain, in contradiction to the reported sensations during incidents. In addition, the difficulty of keeping an RF beam at these frequencies focused through structures such as walls and the unlikely association of sounds produced by pulsed RF (e.g., through Frey effect) with any damage mechanism render this possibility unconvincing. Finally, for incidents with recorded sounds, RF as a source can be ruled out conclusively.

Future data, especially if collected in a uniform and quantitative manner, may help with the resolution of these anomalous incidents. Data collection falls into two categories. First is searching directly for harmful signals through persistent monitoring. This effort should be focused on potential signals that have not already been ruled out. Second is a redesigned uniform incident questionnaire with numerical evaluation criteria that can be employed at all embassies to provide an initial filtering of events and help guide appropriate medical and monitoring response. We recommend that only incidents that meet a threshold to be included in an AHI database, to provide a robust sample on which studies and hypothesis testing can be carried out. We also emphasize that judging a case to be inconsistent with an anomalous health incident carries no implications about its importance or severity. An inner ear infection, stroke, or onset of PPPD can be serious conditions that require medical attention even if they are not AHI.

Regarding adversarial intent, it is not possible to conclude at this time that the events reviewed by JASON are the result of intentional attacks that cause physical harm. However, it is not possible, either, to rule out mechanisms that do not cause any physical harm but which might constitute harassment and lead to health conditions and functional disorders, for example through unpleasant sounds or pressure sensations. Given this, and in the interest of protecting embassy personnel and their families, it would be prudent to be vigilant against tactics intended to produce anxiety and trauma, with an intent to either

disrupt operations and/or cause long-term harm. The US government could minimize the effects of such tactics, if present, through open communication, education, and appropriate rapid medical response to any conditions that develop.

Table 3: An Assessment of Potential Causes[1]

	Consistent Sensations	Inconsistent Effects	Biological Mechanism	Barrier Penetration	Focusing	Production
Ionizing Radiation	neuro symp. nausea fatigue	skin burns rad sickness organ damage no sounds	cell and DNA damage	can penetrate thick walls	can be directed	bulky equipment
100 MHz RF	tingling muscular eff	body heat sensation	neuro effects unestablished	can penetrate building	beam unlikely	large antenna
1−10 GHz RF	sounds (Frey) potential nerve stimulation	skin heat electr/comm disruption	neuro effects require high flux	medium penetration capability	no beam through barriers	medium antenna
30 GHz RF	potential thermal effects	skin pain electr/comm disruption	neuro effects unestablished	penetration challenging	no beam through barriers	small antenna
Infrasound	headache nausea dizziness	whole body sensation	pressure &vibrations on organs	minor loss through walls	no focusing	very large equipment
Audible Sound	hearing loss headache	confusion dizziness no sound cases	ear damage at high dB	significant dB loss thru building	directed not localized	large equipment for high dB
Ultrasound	pressure, dizziness tinnitus, headache nausea, sounds	long-term effects	hyperthermia acoustic cavitation	significant dB loss thru building	directed not localized	large equipment for high dB

1. A scoring of potential causes based on several biological and physical criteria in the context of the incident and medical data relevant to the reported events. Color coding indicates the level of consistency between the mechanism and the physical requirements, sensations, or symptoms considered: red denotes inconsistent/highly unlikely, yellow denotes somewhat inconsistent/unlikely, and green denotes possibly consistent/likely. "Inconsistent Effects" column refers to signs and symptoms that should have been present for a given mechanism but are not reported *or* symptoms that are reported but cannot be produced by that mechanism. There is an implied power level in this table for each of these remote energy mechanisms, which is discussed in detail in the report, but assumed here to be the most optimal power level for its relevance for the reported incidents and symptoms.

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A APPENDIX: Sample Incident Intake Questionnaire

This questionnaire is included to serve as an example for the types of quantitative, structured, and hypothesis-related questions that can be asked during an incident report. It is not meant to be complete nor optimized; we recommend expanding and modifying it as needed prior to deploying it widely. For example, some of the situational questions that exist in the current reports are not repeated in their entirety here.

It is important to administer the same questionnaire to all personnel reporting incidents as soon as possible after an event and equally important to ask them to answer every question. A question left blank could lead to ambiguities about whether the individual did not feel that symptom or did not know how to answer that question, defying the attempt at gathering uniform and structured data.

1. Which of the following symptoms, if any, did you experience during the event?

   ☐ Sound

   ☐ Pressure (specify where on your body)

   ☐ Heat (specify where on your body)

   ☐ Pain (specify where on your body)

   ☐ Smell (describe)

   ☐ Physical sensation such as buffeting, vibration, etc.

   ☐ Ringing in ears

   ☐ Nausea

   ☐ Dizziness

   ☐ Headache

2. If you answered yes to Sound in Question 1, check all that apply.

☐ high-pitch

☐ low pitch

☐ continuous

☐ intermittent

☐ directional (specify how)

☐ clicky. Specify if repetitive click, fast clicks, or slow clicks.

☐ specify volume. Barely audible, conversation level, loud, or very loud.

☐ Did you put your hands over your ears? If yes, did it make a difference?

3. If you answered yes to Heat in Question 1, please answer the following.

Circle the one that applies to the heat level: Slight flush, hot, very hot, painful

Circle the one that applies to the onset: Sudden onset, slow rise, intermittent

4. If you answered yes to Dizziness or Nausea in Question 1, please answer the following.

Circle the one that applies to the severity: General wooziness, sudden sweep through your head, complete disorientation

Circle the one that applies to the onset: Sudden onset, slow rise, intermittent

Please specify the duration.

5. If you answered yes to headache in question 1, please specify type:

☐ whole head

☐ localized (specify where in your head)

☐ throbbing

☐ squeezing

☐ stabbing

6. Specify the duration of the event.

7. Did the sensations and symptoms change when you moved?

☐ Yes ☐ No ☐ Maybe

If you answered yes or maybe, which ones and how?

8. Was anyone else present?

☐ Yes ☐ No

If yes, how far away were they from you?

If yes, did they have the same experience? ☐ Yes ☐ No ☐ I don't know.

9. Check any malfunction of electronics near you that was coincident with the event.

☐ Computer reboot or a monitor change

☐ Noise out of any device with audio function, such as computer, phone, etc. If yes, specify.

☐ Phone reboot, display changes, or disruption of communication

10. Check all that apply regarding your location during the incident.

☐ Indoors

☐ Outdoors

☐ Single residence home

☐ Apartment building

☐ Embassy or consulate

☐ Other (please specify)

11. Please estimate the distance to the following:

Nearest building:

Nearest unit if within the same building:

Nearest street:

If indoors, nearest exterior wall or window:

12. How often do you get headaches?

☐ fewer than once per month

☐ once per month

☐ once per week

☐ several times per week

13. Have you ever suffered from or been diagnosed with any of the following? Check all that apply.

☐ Sinus infections

☐ Hearing problems

☐ Tinnitus

☐ Migraines

☐ Vertigo

☐ Blood pressure problems (specify)

☐ None of the above

14. How were you feeling otherwise on the day of the incident? Report any illness or other issue not typical for you on that day or in the days prior.

15. Have these symptoms

- ☐ gotten better
- ☐ stayed the same
- ☐ gotten worse since the event?

If you responded "stayed the same" or "gotten worse", please describe duration and severity.

16. Please describe if anything else out of the ordinary happened at the same time and just before the event.

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B APPENDIX: Acoustic Waves, Fundamentals, and Detailed Calculations

B.1 Sound Speed and Propagation

Acoustic waves are characterized by a sound speed,

$$a_{\rm s} \;=\; \sqrt{\left(\frac{\partial p}{\partial \rho}\right)_s}, \tag{B-1}$$

the square root of the partial derivative of pressure at constant entropy with respect to density. Under the assumption of an ideal gas, at ordinary temperatures, and using the composition for dry air, this leads to

$$a_{\rm s} \;\simeq\; 343\,{\rm m/s} \;=\; 343\,{\rm Hz}\cdot{\rm m} \;=\; 34.3\,{\rm kHz}\cdot{\rm cm}\,. \tag{B-2}$$

Moist air, at a particular relative humidity (RH%), supports sound speeds that are somewhat higher than that of dry air. Relative humidity plays an important role in the propagation of high-frequency sound, as we'll discuss below.

The wavelength of the wave is related to its speed and frequency by

$$\lambda \;=\; \frac{a_{\rm s}}{\nu}\,. \tag{B-3}$$

Considering a few values at different frequencies, we have $\lambda \simeq 50\,{\rm m}$ at an infrasound frequency of $\nu = 7\,{\rm Hz}$, 20 m at 17 Hz, 34 cm $\simeq$ 1 ft at an audio frequency of 1 kHz, 1.7 cm at an ultrasonic frequency of 20 kHz, 1 cm at 34 kHz, and sub-millimeter wavelengths at MHz frequencies often used for medical and material imaging (Fig. 5.1).

Using the standard equations for acoustic energy propagation, we define the *acoustic energy flux* as

$$\mathbf{j}_{\rm ac} \;=\; p'\mathbf{u}'\,, \tag{B-4}$$

with units of $[\,{\rm W/m^2}\,]$ (e.g., Thompson, 1972), where p' is the small acoustic pressure perturbation in the medium and $\mathbf{u}'$ is the perturbation in the velocity field introduced by the acoustic excitation. The (average) threshold of audibility for (healthy) human ears is used as an acoustic-flux/-power reference and accepted as

$$\mathbf{j}_{\rm ac,ref} \;=\; 10^{-12}\,{\rm W/m^2}\,. \tag{B-5}$$

For healthy ears, this level is very nearly frequency-independent, especially at lower frequencies within the audible acoustic frequency band.

This level is used to scale acoustic flux on a decibel (dB) sound power level (SPL),

$$\mathrm{SPL} \;=\; 10\,\log_{10}\left(\frac{\mathbf{j}_{\mathrm{ac}}}{\mathbf{j}_{\mathrm{ac,ref}}}\right), \tag{B-6}$$

yielding $\mathrm{SPL} = 0\,\mathrm{dB}$ as the audible acoustic-flux threshold.

Finally, we define the *acoustic impedance*, which measures the resistance of a medium to the propagation of acoustic waves, as the ratio

$$Z_{\mathrm{ac}} \;\equiv\; \frac{p'}{|\mathbf{u}'|}\,. \tag{B-7}$$

For small perturbations, the impedance is equal to $Z_{\mathrm{ac}} = \rho_0\, a_{\mathrm{s},0}$, where ρ_0 is the density of the unperturbed medium and $a_{\mathrm{s},0}$ is its sound speed.

Because the acoustic flux is quadratic in acoustic pressure, we may also express SPL in terms of the latter, [1]

$$\mathrm{SPL} \;=\; 10\,\log_{10}\left(\frac{p'^2}{p'^2_{\mathrm{ref}}}\right) \;=\; 20\,\log_{10}\left(\frac{p'}{p'_{\mathrm{ref}}}\right), \tag{B-8}$$

with (B-5)

$$p'_{\mathrm{ref}} \;\simeq\; 20\,\mu\mathrm{Pa}\;(\mathrm{rms})\,, \tag{B-9}$$

corresponding to $\mathbf{j}_{\mathrm{ac,ref}}$ in (B-5) in air.[2]

B.2 Acoustic reflection and transmission

Acoustic reflection at boundaries between two different media occurs for many reasons. For semi-infinite media on either side of an interface, such phenomena can be ascribed to differences in the acoustic impedance (B-7), Z_{ac}, of the substance comprising the two media.

[1] We note that 'SPL' is often referred to as the 'sound pressure level'. We prefer 'sound power level' in this document to avoid the confusion of the factor of 2 in its expression in terms of dB in (B-8).

[2] Notable is how small audible rms threshold pressure levels are, as a fraction of ambient pressure, *e.g.*, $p'_{\mathrm{ref}}/p_{\mathrm{atm}} \simeq 2\times10^{-5}/10^{5} \simeq 2\times10^{-10}$. Pain threshold is of the order of 140 dB, *i.e.*, human ears serve as sensors with a dynamic range over 14 orders of magnitude. Some animal ears are known to be better yet.

In non-dispersive media, such as dry air, the sound speed (and therefore, its impedance) has only a weak dependence on the sound wave frequency. The characteristic specific acoustic impedance of air for audible acoustic waves at $T = 25°\mathrm{C}$ is

$$Z_{\mathrm{ac,air,au}} \approx 410\,\mathrm{kg}/(\mathrm{m}^2 \cdot \mathrm{s})\,, \tag{B-10a}$$

and only slightly higher for ultrasound waves, *i.e.*,

$$Z_{\mathrm{ac,air,us}} \approx 430\,\mathrm{kg}/(\mathrm{m}^2 \cdot \mathrm{s})\,. \tag{B-10b}$$

Attenuation and dissipation by various mechanisms are more significant at high frequencies, as described below, especially in dispersive media.

The acoustic impedance of a concrete (wall) for ultrasound waves, for example, is

$$Z_{\mathrm{ac,us,conc}} \approx 5 - 8 \times 10^6\,\mathrm{kg}/(\mathrm{m}^2 \cdot \mathrm{s})\,, \tag{B-10c}$$

measured with a transducer with a central frequency of $\nu = 0.5\,\mathrm{MHz}$ (Lotfi et al., 2013). For glass, a typical value is,[3]

$$Z_{\mathrm{ac,glass}} \approx 14 \times 10^6\,\mathrm{kg}/(\mathrm{m}^2 \cdot \mathrm{s})\,. \tag{B-10d}$$

At boundaries between media with different acoustic impedances, some of the wave energy is reflected and some is transmitted. The acoustic *amplitude* reflection coefficient, $\mathcal{C}_{\mathrm{ac,r}}$, i.e, the ratio of the reflected pressure p'_{r} to the incident pressure p'_{i}, depends on the acoustic impedances of the two media on either side of the boundary, $Z_{\mathrm{ac},1}$ and $Z_{\mathrm{ac},2}$.

For waves at normal incidence to the boundary surface, the wave-amplitude reflection coefficient at normal incidence is given by

$$\frac{p'_{\mathrm{r}}}{p'_{\mathrm{i}}} = \mathcal{C}_{\mathrm{ac,r}} = \frac{Z_{\mathrm{ac},2} - Z_{\mathrm{ac},1}}{Z_{\mathrm{ac},2} + Z_{\mathrm{ac},1}}\,, \tag{B-11a}$$

and can be both positive and negative, *i.e.*, inducing a wave phase flip in the latter case. Reflection of the sound intensity (energy-flux) is given by $\mathcal{C}^2_{\mathrm{ac,r}}$.

[3] `https://www.bostonpiezooptics.com/ultrasonic-properties`.

Absent absorption/attenuation in the media and at their interface, the total energy flux on the two sides of the interface will be conserved and we'll have

$$\mathcal{C}_{\rm ac,r}^2 + \mathcal{C}_{\rm ac,t}^2 = 1\,, \tag{B-11b}$$

where $\mathcal{C}_{\rm ac,t}$ is the wave-amplitude transmission coefficient. At a typical interface between air and a solid wall, for example, this expression would give a near-zero transmitted acoustic flux, indicating that the vast majority of the acoustic wave flux would be reflected rather than transmitted into and through a semi-infinite adjoining high-$Z_{\rm ac}$ medium.

The relations above are for normal incidence. Acoustic waves incident at an angle will both reflect and refract with angles and amplitudes reminiscent of Snell's Law of reflection and refraction for optics (*e.g.*, Thompson, 1972, Sec. 4.9).

An analysis of transmission of acoustic waves through finite-thickness slabs and boundaries with dissimilar materials, *e.g.*, walls, window panes, *etc.*, needs to take both the thickness of the finite-thickness slab, say, d and the acoustic wavelength (B-3), λ, or wavenumber $k = 2\pi/\lambda$, into account.

Considering a glass slab, as in a window pane, sound speed is much higher in glass than in air, with $a_{\rm ac,glass} \simeq 4540\,{\rm m/s}$, for example, yielding a sound-speed and a corresponding wavelength ratio (at the same frequency) with respect to air of

$$\frac{a_{\rm ac,glass}}{a_{\rm ac,air}} = \frac{\lambda_{\rm ac,glass}}{\lambda_{\rm ac,air}} \simeq \frac{4540}{340} \simeq 13.4\,. \tag{B-12a}$$

An ultrasonic acoustic wave with a

$$\lambda_{\rm ac,air}(\nu{=}20\,{\rm kHz}) \simeq 1.7\,{\rm cm} \tag{B-12b}$$

wavelength (B-3) in air, will then have a wavelength in glass given by

$$\lambda_{\rm ac,glass}(\nu{=}20\,{\rm kHz}) \simeq 13.4 \times 1.7\,{\rm cm} = 22.8\,{\rm cm}\,. \tag{B-12c}$$

As a consequence, standing-wave resonances in window panes cannot occur at even ultrasonic frequencies of concern. Moreover, a typical window glass-pane thickness, d, will be much smaller than $\lambda_{\rm glass}$, even at ultrasonic frequencies, and, as a consequence, will respond as a slab comprised of an incompressible material, impeding transmission by its areal density $[\,{\rm kg/m^2}\,]$, as discussed in App. B.4.

Glass window panes are mounted in frames and, therefore, also respond to the equivalent of drum-membrane frequencies that will be much lower, also depending on the damping offered by the window-pane mount, *e.g.*, low-damping aluminum vs. high-damping wood, *etc.* Such frequencies will also depend on the glass thickness, density, elasticity, mounting details, whether single-pane, double-pane with the latter using purposely different panes to avoid cross-resonances, etc.

Ultrasonic waves are attenuated when propagating over long distances in air, especially moist air, as discussed below, further impeding the ability to project them at a distance at high frequency.

B.3 Sound attenuation and localization

Sound propagates effectively in air with an intensity (*e.g.*, acoustic flux) that can decrease either geometrically, by spreading, as from a point source at distances large compared to its wavelength, or attenuated by dissipation through the action of bulk viscosity and heat conductivity.

Dissipative (as opposed to geometric) attenuation of acoustic intensity/flux per unit propagation length, x, *i.e.*,

$$I(x) \,=\, I_0\, \mathrm{e}^{-\alpha x}. \tag{B-13a}$$

The attenuation coefficient, α, has units of reciprocal length and may be approximated with a nominally quadratic scaling with frequency in dry air, i.e.,

$$\alpha(\nu) \,=\, \alpha_0 (\nu/\nu_0)^2, \tag{B-13b}$$

where α_0 is the attenuation specified at the reference frequency ν_0.

Attenuation generally depends on the gas-mixture bulk viscosity and heat conductivity with a frequency dependence that is more complicated in mixtures with molecules that have energy modes with characteristic frequencies in the vicinity of sound frequencies. An important example in our context is moist air where sound attenuation depends on relative humidity (Fig. B.1), and other air-mixture constituents (*e.g.*, Harris, 1966; Bass et al., 1995).

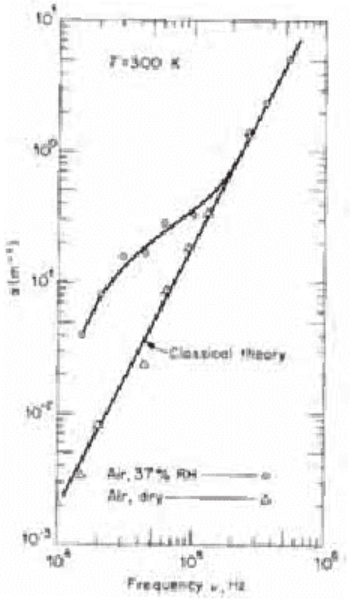

Figure B.1: Sound attenuation in dry and moist air, also illustrating the quadratic dependence of the attenuation coefficient for dry air. Data from Sivian (1947). Figure from Thompson (1972, Fig. 4.38).

Dissipative attenuation begins to become significant at frequencies of tens of kHz and above, as can be seen in Fig. B.1, with attenuation of 20 kHz sound in dry air of order $\alpha_{\text{dry air}} \simeq 6 \times 10^{-3}/\text{m}$ and $\alpha_{\text{air, 37\%RH}} \simeq 8 \times 10^{-2}/\text{m}$. For a *collimated* acoustic beam traversing a distance $x = 30\,\text{m}$, this gives

$$\frac{I(\nu{=}20\,\text{kHz}, x{=}30\,\text{m})}{I_0} \simeq \text{e}^{-8\times10^{-2}\times30} = \text{e}^{-2.4} \simeq -10\,\text{dB}\,, \tag{B-14a}$$

for RH=37% air, and $\alpha_{\text{air, 37\%RH}}(40\,\text{kHz}) \simeq 0.15/\text{m}$

$$\frac{I(\nu{=}40\,\text{kHz}, x{=}30\,\text{m})}{I_0} \simeq \text{e}^{-0.15\times30} = \text{e}^{-4.5} \simeq -20\,\text{dB}\,, \tag{B-14b}$$

illustrating the importance and role of water vapor content in sound attenuation in air.

Ultrasound *can* be projected and localized/focused in air in open spaces from such distances, and perhaps from beyond, if generated with a large D/λ transmitter of adequate power. Such a beam, projected from a distance into the interior of a building, might rely on transmission through a glass window pane, and would undergo a total attenuation that

is at least as high as the sum of the attenuation in air and that through the window:

$$\underbrace{-10\,\mathrm{dB}}_{20\,\mathrm{kHz,\ air}} + \underbrace{-40\,\mathrm{dB}}_{\mathrm{window}} \simeq \underbrace{-50\,\mathrm{dB}}_{20\,\mathrm{kHz,\ total}}\,, \tag{B-15a}$$

and

$$\underbrace{-20\,\mathrm{dB}}_{40\,\mathrm{kHz,\ air}} + \underbrace{-40\,\mathrm{dB}}_{\mathrm{window}} \simeq \underbrace{-60\,\mathrm{dB}}_{40\,\mathrm{kHz,\ total}}\,. \tag{B-15b}$$

as also shown graphically in Fig. B.2.

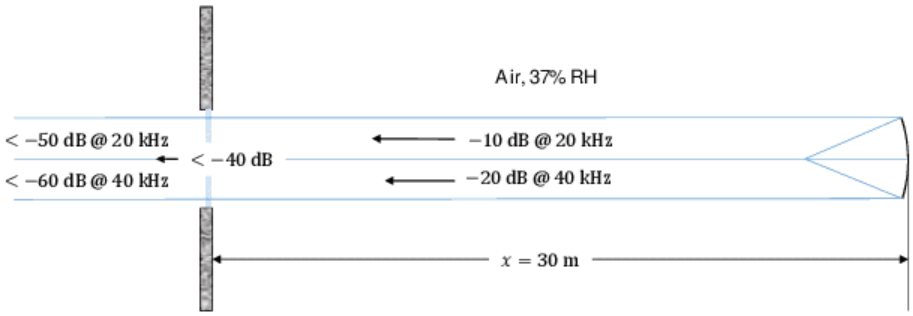

Figure B.2: Notional sketch for the attenuation of a 20 kHz and a 40 kHz focused acoustic wave, emanating from a source at a distance $x = 30\,\mathrm{m}$, propagating through air with a 37% RH, transmitted through a glass window pane into an adjacent interior of a building.

Acoustic energy flux spreading as a spherical wave across $x = 30\,\mathrm{m}$, from a localized source that can be treated as a point source at the distance, with I_0 measured, say, at $x_{\mathrm{src}} = 1\,\mathrm{m}$ from the emitter, would have an amplitude that would spread as $1/r$ and undergo an additional geometric intensity attenuation,

$$\left(\frac{x_{\mathrm{src}}}{x}\right)^2 = \frac{1}{30^2} = -29.5\,\mathrm{dB}\,, \tag{B-15c}$$

in an open space, *i.e.*, excluding multi-path effects. This yields a total attenuation at least as high as

$$\underbrace{-39.5\,\mathrm{dB}}_{20\,\mathrm{kHz,\ air}} + \underbrace{-40\,\mathrm{dB}}_{\mathrm{window}} \simeq \underbrace{-79.5\,\mathrm{dB}}_{20\,\mathrm{kHz,\ total}}\,, \tag{B-15d}$$

and

$$\underbrace{-49.5\,\mathrm{dB}}_{40\,\mathrm{kHz,\ air}} + \underbrace{-40\,\mathrm{dB}}_{\mathrm{window}} \simeq \underbrace{-89.5\,\mathrm{dB}}_{40\,\mathrm{kHz,\ total}}\,. \tag{B-15e}$$

See Fig. B.3.

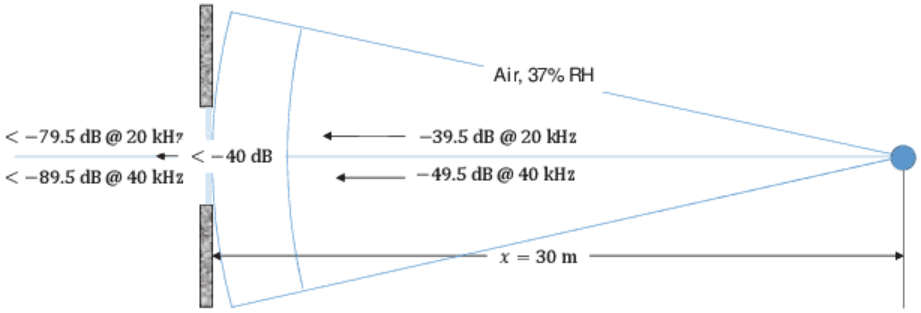

Figure B.3: Notional sketch for the attenuation of a 20 kHz and a 40 kHz spherical acoustic wave, emanating from a source at a distance $x = 30$ m, propagating through air with a 37% RH, transmitted through a glass window pane into an adjacent interior of a building.

In a realistic situation, the total attenuation would be bracketed by these bounding values imposing, in either case, substantial requirements to the projection of significant acoustic energy/power into the interior of a building or a vehicle, through closed, glass-pane windows, on the transmitter power and size, if emission is intended to be covert/undetected.

Infrasound has a very low attenuation propagating through even moist air, with an intensity that primarily decreases as a result of geometric dispersion, i.e., as a spherical wave, for example, or refraction/ducting characteristics, diffuse scattering/reflection from rough/irregular surfaces, etc. As a consequence and because of its low attenuation, infrasound can propagate to great distances and is useful in monitoring distant natural or man-made explosions in the atmosphere, excitation by seismic waves, volcanic eruptions, exploiting long-distance wave-guide ducting by the layered terrestrial atmosphere.

Generally, as a wave, sound can be focused, for example, to spatial extents scaled by its wavelength. As a consequence, infrasound on human spatial scales cannot be localized to meter-/sub-meter-scaled regions. In contrast, ultrasound can form beams and be collimated (*e.g.*, Fig. B.2), or even focused, by large-aperture reflectors or transmitters if the diameter of their aperture, D, scaled by the sound wavelength is large, *i.e.*, if $D/\lambda \gg 1$, akin to optics.

With short attendant wavelengths, ultrasound enables high-resolution medical imaging. Such applications have helped establish safe operating limits and intensities, extending

to fetuses, albeit employing direct, well-coupled means to human tissue.

B.4 Sound Transmission through a Slab

Transmission of sound at normal incidence through a slab of thickness d, *e.g.*, a glass pane or a wall, can be estimated in an ideal case where the transverse extent of the slab can be taken as infinite and we have a right-going incident wave with a left-going reflected harmonic wave (pressure) amplitude given by at $x = 0$,

$$p'_{\rm i} \quad \text{and} \quad p'_{\rm r}\,, \tag{B-16a}$$

respectively, a right-going and a left-going wave through the slab material at x, given by,

$$p'_{+}\mathrm{e}^{ikx} \quad \text{and} \quad p'_{-}\mathrm{e}^{-ikx}\,, \tag{B-16b}$$

with $kd = 2\pi d/\lambda$, and an outgoing, right-propagating transmitted wave in air, given by, as it emerges at $x = d$,

$$p'_{\rm t}\mathrm{e}^{ikd} \tag{B-16c}$$

as depicted in Fig. B.4.

Given the incident-wave amplitude, $p'_{\rm i}$, we can solve for the other four amplitudes by matching the pressure and velocity on the left and right slab faces, with $u' = \pm p'/Z_{\rm ac}$ for right- and left-going waves (**??**), respectively, *i.e.*,

$$\begin{aligned} p'_{\rm i} + p'_{\rm r} &= p'_{+} + p'_{-} \\ \frac{p'_{\rm i} - p'_{\rm r}}{Z_{\rm ac,air}} &= \frac{p'_{+} - p'_{-}}{Z_{\rm ac,slab}} \end{aligned} \tag{B-17a}$$

at $x = 0$, and

$$\begin{aligned} p'_{+}\mathrm{e}^{ikd} + p'_{-}\mathrm{e}^{-ikd} &= p'_{\rm t}\mathrm{e}^{ikd} \\ \frac{p'_{+}\mathrm{e}^{ikd} - p'_{-}\mathrm{e}^{-ikd}}{Z_{\rm ac,slab}} &= \frac{p'_{\rm t}\mathrm{e}^{ikd}}{Z_{\rm ac,air}}\,, \end{aligned} \tag{B-17b}$$

at $x = d$, or, if

$$\zeta = \frac{Z_{\rm ac,slab}}{Z_{\rm ac,air}}\,, \tag{B-17c}$$

then

$$\begin{aligned} p'_{\mathrm{i}} + p'_{\mathrm{r}} &= p'_{+} + p'_{-} \\ p'_{\mathrm{i}} - p'_{\mathrm{r}} &= \frac{1}{\zeta}\left(p'_{+} - p'_{-}\right) \end{aligned} \tag{B-17d}$$

at $x = 0$, and, at $x = d$ with $\varphi = \mathrm{e}^{ikd}$,

$$\begin{aligned} p'_{+}\varphi + p'_{-}\varphi^{-1} &= \varphi\, p'_{\mathrm{t}} \\ p'_{+}\varphi - p'_{-}\varphi^{-1} &= \zeta\,\varphi\, p'_{\mathrm{t}}\,. \end{aligned} \tag{B-17e}$$

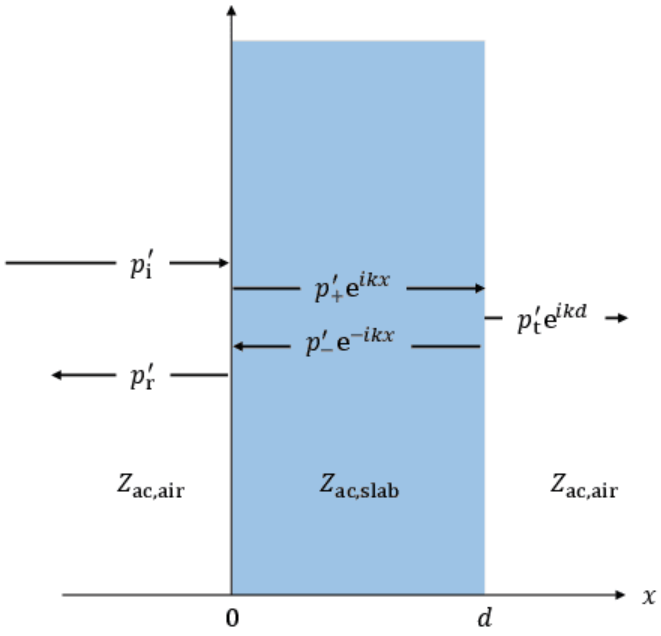

Figure B.4: Transmission of a sound wave at normal incidence on a slab, at a frequency ν, with a wavenumber $k_{\mathrm{air}} = 2\pi/\lambda_{\mathrm{air}} = 2\pi\nu/a_{\mathrm{air}}$ in air, through a slab of thickness d, with a wavenumber $k = k_{\mathrm{slab}} = 2\pi/\lambda_{\mathrm{slab}} = 2\pi\nu/a_{\mathrm{slab}}$ in the slab.

Solving (B-17e) for $p'_{\pm}$ in terms of p'_{t}, we have

$$\begin{aligned} p'_{+} &= \frac{1}{2}\left(1+\zeta\right) p'_{\mathrm{t}} \\ p'_{-} &= \frac{1}{2}\left(1-\zeta\right) p'_{\mathrm{t}}\varphi^{2}. \end{aligned} \tag{B-18a}$$

Substituting in (B-17d), we then have in terms of the transmitted wave

$$\begin{aligned} p'_{\mathrm{i}} + p'_{\mathrm{r}} &= \frac{1}{2}\left(1+\zeta\right) p'_{\mathrm{t}} + \frac{1}{2}\left(1-\zeta\right) p'_{\mathrm{t}}\varphi^{2} \\ p'_{\mathrm{i}} - p'_{\mathrm{r}} &= \frac{1}{\zeta}\left[\frac{1}{2}\left(1+\zeta\right) p'_{\mathrm{t}} - \frac{1}{2}\left(1-\zeta\right) p'_{\mathrm{t}}\varphi^{2}\right] \end{aligned} \tag{B-18b}$$

or,

$$\begin{aligned} p'_{\rm i} + p'_{\rm r} &= \frac{1}{2}\left[(1+\zeta) + (1-\zeta)\,\varphi^2\right] p'_{\rm t} \\ p'_{\rm i} - p'_{\rm r} &= \frac{1}{2\zeta}\left[(1+\zeta) - (1-\zeta)\,\varphi^2\right] p'_{\rm t}\,. \end{aligned} \tag{B-18c}$$

Adding the two, we solve for the incident amplitude, $p'_{\rm i}$ required to yield a transmitted amplitude $p'_{\rm t}$, *i.e.*,

$$\begin{aligned} p'_{\rm i} &= \frac{1}{4}\left(1+\frac{1}{\zeta}\right)\left[(1+\zeta) + (1-\zeta)\,\varphi^2\right] p'_{\rm t} \\ &= \frac{(\zeta+1)^2 - \left(\zeta^2-1\right)\varphi^2}{4\zeta}\, p'_{\rm t}\,, \end{aligned} \tag{B-18d}$$

noting that $p'_{\rm t}/p'_{\rm i} \to 1$ as $d \to 0$ and $\varphi \to 1$, as it must. The corresponding intensities are then given by,

$$\begin{aligned} \left|\, p'_{\rm i}\,\right|^2 &= \frac{\left[(\zeta+1)^2 - \left(\zeta^2-1\right)\varphi^2\right]\left[(\zeta+1)^2 - \left(\zeta^2-1\right)\varphi^{-2}\right]}{16\zeta^2}\,\left|\, p'_{\rm t}\,\right|^2 \\ &= \frac{(\zeta+1)^4 - (\zeta+1)^2\left(\zeta^2-1\right)\left(\varphi^2+\varphi^{-2}\right) + \left(\zeta^2-1\right)^2}{16\zeta^2}\,\left|\, p'_{\rm t}\,\right|^2 \\ &= \frac{(\zeta+1)^4 - 2\,(\zeta+1)^2\left(\zeta^2-1\right)\cos(2kd) + \left(\zeta^2-1\right)^2}{16\zeta^2}\,\left|\, p'_{\rm t}\,\right|^2, \end{aligned} \tag{B-18e}$$

for lossless interface transmission and reflection, and propagation through the slab medium.

As a consequence, even at ultrasonic frequencies, a typical window-pane thickness ($d \approx 3/32'' \simeq 0.24\,\text{cm}$) is much smaller than the corresponding wavelengths in glass and the argument in the cosine in (B-18e) will be small, *i.e.*,

$$\cos(2kd) \;\approx\; 1 \;-\; 2\,(kd)^2 \;+\; \ldots\,. \tag{B-19a}$$

In that limit, we'll then have,

$$\begin{aligned} \left|\, p'_{\rm i}\,\right|^2 &\simeq \frac{(\zeta+1)^4 - 2\,(\zeta+1)^2\left(\zeta^2-1\right)\left[1 \;-\; 2\,(kd)^2\right] + \left(\zeta^2-1\right)^2}{16\zeta^2}\,\left|\, p'_{\rm t}\,\right|^2, \\ &= \left[1 \;+\; \frac{(\zeta+1)^2\left(\zeta^2-1\right)}{4\zeta^2}\,(kd)^2\right]\left|\, p'_{\rm t}\,\right|^2, \\ &\simeq \left[1 \;+\; \frac{1}{4}\,(\zeta kd)^2\right]\left|\, p'_{\rm t}\,\right|^2, \end{aligned} \tag{B-19b}$$

the latter relation for $\zeta \gg 1$.

Substituting for ζ from (B-17c), we then have,

$$\begin{aligned} |\, p_{\rm i}' \,|^2 &\simeq \left[1 + \frac{1}{4} \left(\frac{Z_{\rm ac,glass}\, kd}{Z_{\rm ac,air}} \right)^2 \right] |\, p_{\rm t}' \,|^2 \\ &= \left[1 + \frac{1}{4} \left(\frac{\rho_{\rm glass}\, 2\pi\nu d}{\rho_{\rm air}\, a_{\rm air}} \right)^2 \right] |\, p_{\rm t}' \,|^2 \end{aligned} \tag{B-19c}$$

or,

$$|\, p_{\rm i}' \,|^2 \simeq \pi^2 \left(\frac{\rho_{\rm glass}\, d}{\rho_{\rm air} \lambda_{\rm air}} \right)^2 |\, p_{\rm t}' \,|^2 \,, \tag{B-19d}$$

noting that $\rho_{\rm glass} d$ is the areal density $[\,{\rm kg/m^2}\,]$ of the glass slab.

Substituting $\rho_{\rm glass} \simeq 2.5 \times 10^3\,{\rm kg/m^3}$, $d \simeq 3/32'' = 0.24\,{\rm cm}$, $\rho_{\rm air} \simeq 1.2\,{\rm kg/m^3}$, and $\lambda_{\rm air}(20{\rm kHz}) = 1.7\,{\rm cm}$, we then have,

$$\pi^2 \left(\frac{\rho_{\rm glass}\, d}{\rho_{\rm air} \lambda_{\rm air}} \right)^2 \simeq 8.2 \times 10^5 \,, \tag{B-20}$$

yielding a transmitted acoustic intensity ratio for $\nu = 20\,{\rm kHz}$ sound of

$$\left| \frac{p_{\rm t}'}{p_{\rm i}} \right|^2 \simeq 1.2 \times 10^{-6} \simeq -59\,{\rm dB} \,. \tag{B-21}$$

and less at higher frequencies, or for a thicker glass plane.

Transmission of acoustic energy through an actual window with a glass pane can be somewhat higher because the glass pane also acts as a drum membrane and can transmit by vibrating as such as well through its mounting, frame, etc.

C APPENDIX: Mathematical Details of the Frey Effect

For simplicity, consider a plane-parallel geometry with RF pulses normally incident from $x < 0$, and the tissue of interest (brain or skin) a semi-infinite medium lying at $x > 0$. A very short pulse heats this tissue (very slightly) at $t = 0$, resulting in an initial pressure profile

$$\delta p(x, 0) = \delta p_{\max} e^{-x/h}, \quad x > 0. \tag{C-1}$$

Here the prefix "δ" serves as reminder that the pressure perturbation is much less than the background (atmospheric) pressure, which we presume to be uniform. We neglect nonlinearities and treat this problem to first order in δp. Insofar as the sound speed in the tissue is constant,[1] the general solution to the one-dimensional acoustic wave equation is

$$\delta p(x, t) = g_+(x - t) + g_-(x + c_s t),$$

in which $g_\pm$ are arbitrary functions describing right- (g_+) and left-going (g_-) waves. The corresponding velocity perturbation is

$$\delta v(x, t) = \frac{g_+(x - c_s t) - g_-(x + c_s t)}{\rho c_s},$$

in which $\rho \approx 1\,\mathrm{g\,cm^{-3}}$ is the density of the tissue. By hypothesis, $\delta v(x, 0) = 0$ for all $x \geq 0$, and it therefore follows that g_+ and g_- are the same function, i.e. $g_+(\xi) = g_-(\xi) \equiv g(\xi)$.

A rigid boundary: In this case $\delta v(0, t) = 0$ for all $t \geq 0$. It then follows that $g(-c_s t) = g(+c_s t)$, i.e. $g(\xi)$ is symmetric about $\xi = 0$. Matching this to the initial condition (C-1), one sees that for $x \gg h$, the acoustic pulse propagating into the tissue has the pressure profile

$$\delta p(x, t) = \frac{\delta p_{\max}}{2} \exp(|x - c_s t|/h)\,. \tag{C-2a}$$

The factor 1/2 comes from the fact that $g(x \pm c_s t)$ contribute equally to δp at $t = 0$, but $g(x + c_s t)$ becomes exponentially small at $x \approx c_s t$ at $t \gtrsim h/c_s$.

[1] We ignore the slight change in sound speed c_s produced by the heating itself.

A free boundary: In this case $\delta p(0,t) \approx 0$ for $t > 0$, so that[2] $-g(-\xi) = g(\xi)$, i.e. $g(\xi)$ is antisymmetric around $\xi = 0$. Instead of eq. (C-2b), the pulse propagating into the tissue becomes

$$\delta p(x,t) = \frac{\delta p_{\max}}{2} \exp(|x - c_s t|/h) \times \mathrm{sign}(x - c_s t)\,. \tag{C-2b}$$

To determine how much of this is audible, consider first the temporal Fourier transforms of these pulses. At some fixed station $x \gg h$, let $\tau = t - x/c_s$ be a shifted time variable, and $\delta p(\tau)$ the pressure profile measured at this station. The Fourier transform of this is

$$\begin{aligned}
\delta\tilde{p}(f) &\equiv \int_{-\infty}^{\infty} \delta p(\tau) e^{2\pi i f \tau} \\
&= \frac{f_s}{f^2 + f_s^2} \frac{\delta p_{\max}}{2\pi} && \text{for a rigid boundary;} && \text{(C-3a)} \\
&= \frac{-if}{f^2 + f_s^2} \frac{\delta p_{\max}}{2\pi} && \text{for a free boundary.} && \text{(C-3b)}
\end{aligned}$$

Here, again, $f_s = c_s/2\pi h$. Notice that in the case of a free boundary, the amplitude near DC (i.e. $f = 0$) is small, because $\delta p(\tau)$ integrates (almost) to zero.

The frequency response of the human ear is complicated and dependent on age, but to demonstrate the scalings mentioned in the caveats above, a simple and mathematically convenient low-pass filter will do, such as

$$H(f) = \frac{f_0^2}{f^2 + f_0^2}\,. \tag{C-4}$$

Here f_0 is a cutoff frequency, which might be 20 kHz for a healthy young person with clean habits, and rather less for the author of this note. The normalization has been set so that $H(0) = 1$.

[2] More precisely, $-g(-\xi) = g(\xi) \times (1-r)/(1+r)$, with $r = (\rho c_s)_{\mathrm{air}}/(\rho c_s)_{\mathrm{tissue}} \ll 1$.

The audible (low-pass-filtered) pressure profile is then

$$\begin{aligned}\delta\hat{p}(\tau) &= \int_{-\infty}^{\infty} \delta\tilde{p}(f)H(f)e^{-2\pi i f\tau}\,df \\ &= \frac{\delta p_{\max}}{2} \times \frac{f_0}{f_s^2 - f_0^2}\left(f_s e^{-2\pi f_0|\tau|} - f_0 e^{-2\pi f_s|\tau|}\right) \qquad \text{if rigid;} \qquad \text{(C-5a)} \\ &= \frac{\delta p_{\max}}{2} \times \frac{f_0^2}{f_s^2 - f_0^2}\left(e^{-2\pi f_0|\tau|} - e^{-2\pi f_s|\tau|}\right)\operatorname{sign}(\tau) \qquad \text{if free.} \qquad \text{(C-5b)}\end{aligned}$$

For $f_s \gg f_0$, the peak pressure occurs when $f_s^{-1} \ll \tau \ll f_0^{-1}$ and is reduced from its unfiltered value $\delta p_{\max}/2$ by factors $\approx f_0/f_s$ and the square of this for rigid and free boundaries, respectively.

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D APPENDIX: BlueJay 2nd-gen RF Detector

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E APPENDIX: Microwave Pulser

Given that there appears to have been no upsets of electronic devices (cellphones, televisions, computer and audio equipment) at the time of the reported events, one is led to ask whether the source could have been ultra-short pulsed RF, say with pulse widths in the nanosecond or sub-nanosecond range. Such RF spreads its energy over a broad frequency range (~ 1 GHz or more), consequently with greatly reduced spectral energy density: so the resonances of wiring within nearby electronic devices would be less strongly driven. Another way to think about it is that the distributed series inductances and shunt capacitances[1] of the victim device more easily attenuate the ultra-short pulses.

So it may be useful to conduct some animal model tests with a source of pulsed microwaves, for example ~ 10 GHz nanosecond-scale pulses at the kilowatt power level. From the briefings we received, it appears that such test sources are not available, or at least they have not been used to explore physiological effects *in vivo*.

We have identified commercially available technology that can satisfy these specifications; we detail these below.

E.1 Commercially Available Components

What's needed is a source of low-level ns-pulsed microwave RF, followed by a microwave power amplifier of adequate bandwidth to preserve the pulse.

E.1.1 The low-level pulsed RF

A first idea for an inexpensive pulsed source might be a CW oscillator, gated by an RF switch. The oscillator is no problem, but the switch *is*: PIN diode switches are slow, and even the fastest GaAs switches have rise and fall times in the tens of nanoseconds. Another way to switch RF is to use a mixer, driving the (dc-coupled) IF port with the gating pulse: the oscillator's RF drives the RF port, and the gated burst emerges from the LO port.

[1] Contemporary electronic devices make extensive use of ground planes in their multilayer circuit boards, to ensure good signal integrity and to suppress coupling and radiated RF.

This *does* work, but typically the RF–LO isolation in the OFF state is unimpressive, with typical values of 40–50 dB. We've used this technique in applications where some OFF-state feedthrough is acceptable. For our application here we could use a Marki Microwave MM1-0212HSM (or -LSM) GaAs mixer (datasheet attached), with typical OFF-state isolation of 57 dB over 2–12 GHz, driven by a low-level X-band signal generator.

A better source, if cost is not constrained, is an arbitrary function generator ("arb"); for this task something like the Tektronix AWG70001B (datasheet attached), which clocks its DAC at 50 Gsps and can generate waveforms to 20 GHz. This is high-end instrumentation (as the saying goes, "if you have to ask the price, you can't afford it"), but a well equipped test lab might have one, and in any case one can always rent a unit for a set of limited tests.

E.1.2 The power amplifier

Microwave power amplifiers tend to be narrowband, with tuned elements or cavities. But there are available some kilowatt-scale amplifiers with 10% to 20% bandwidths.

An example of the latter is the L5850 helix traveling wave tube (TWT) from L3-Harris: its specified band is 8.5–10.5 GHz, with 60 dB RF gain and 2 kW output power; it is specified for "CW/Pulsed" operation, so it is running cool when driven with nanosecond pulses at, say, audio rates (which would generate an audible Frey effect). So it takes just 2 mW drive (+3 dBm) for full 2 kW output. TWTs require high voltages (here −11 kV and +7.7 kV), but they do the job.[2] For higher frequencies a gyrotron (a Russian invention) could be used. For example, the VGB-8193 amplifier from CPI (Communications & Power Industries) has 1.6 GHz bandwidth at 95 GHz, with 55 kW (peak) output power and 33dB gain.

A solid-state amplifier uses more friendly operating voltages; a suitable unit is the VSX3696 from CPI. It has a 10% bandwidth (9–10 GHz), 58 dB gain, and a saturated peak RF output of 1.8 kW; it runs on a single 50 V supply (datasheet attached).

These examples are based on limited searching on the Internet. Greater power levels

[2]We requested a full datasheet, but have not received a response.

and shorter pulse widths can likely be obtained with commercially available components.

E.1.3 Getting to *Mega*watts

There is a substantial Russian literature on techniques for generating ns-scale repetitively pulsed microwaves at megawatt peak RF levels. For example, an article by Efremov et al. ("Generation and Radiation of High-Power Ultrawideband Nanosecond Pulses") reports E_pR products of 750 kV in nanosecond L-band pulsed radiation, and antenna far-field patterns of some 25° FWHM (with impressively low sidelobes). This and similar publications use nonlinear techniques of pulse shortening – spark-gap switches (singly or in sequence), or ferrite-loaded nonlinear transmission lines, or avalanche breakdown of semiconductor diodes.[give cites?] Some of these publications feature compact sources ("Small-Sized Nanosecond Source of Powerful Wide-Band VUV–UV Radiation")

Figures E.1–E.3 show representative data from another Efremov et al. publication ("High-Power Sources of Ultra-wideband Radiation with Subnanosecond Pulse Lengths"). Notable are the high radiated power levels (Fig. E.1), the sub-nanosecond RF pulse width (Fig. E.2), and the good directivity of the antenna array (Fig. E.3).

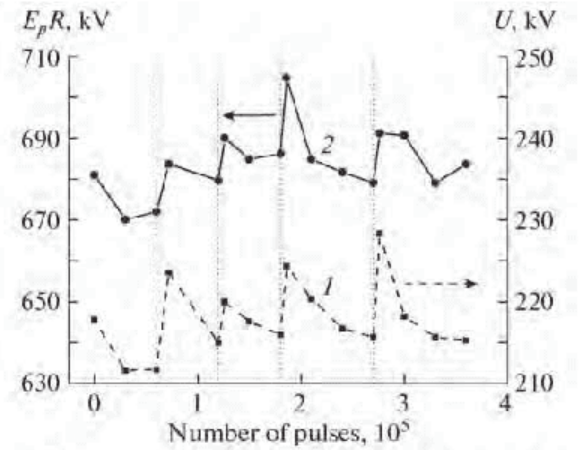

Fig. 14. (*1*) Amplitude of a bipolar voltage pulse and (*2*) the effective radiation potential as functions of the number of pulses.

Figure E.1: Radiated potential ($E_R \cdot R$) and drive pulse voltage of Efremov et al. (2010).

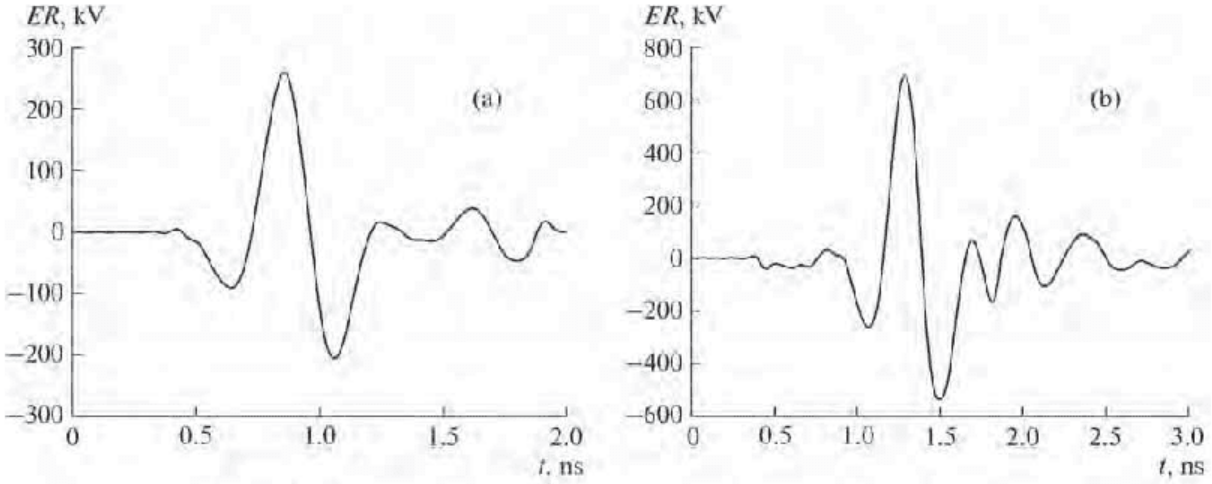

Fig. 13. Oscillograms of pulses radiated by (a) the single antenna and (b) the array.

Figure E.2: RF pulse waveforms of Efremov et al. (2010).

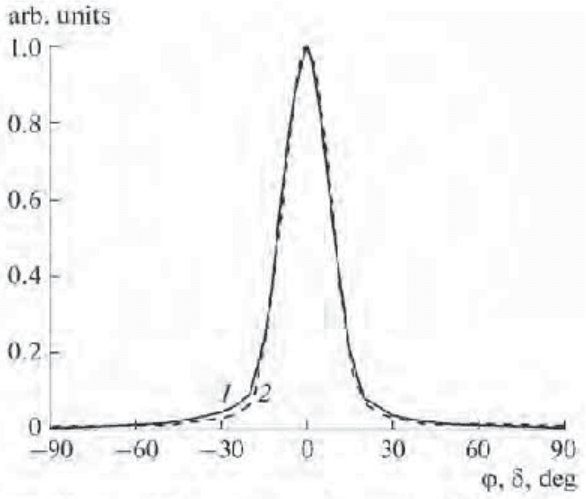

Fig. 11. Directivity patterns of the arrays in the (*1*) *H* and (*2*) *E* planes.

Figure E.3: RF radiation pattern of the array of Efremov et al. (2010).

Additionally, some published articles from the same organization (Institute of High-Current Electronics, Tomsk, Russia) refer to observations of "different biological objects" to pulsed RF (e.g., Romanchenko et al., "Gyromagnetic RF source for interdisciplinary research")

E.2 Portable Power Sources

Could a carryable battery provide the dc power needed for a kilowatt-to-megawatt ns-pulsed mm-wave illuminator? The *average* energy requirements are surprisingly modest, and in fact there are abundant battery packs, intended for video camcorders, lighting equipment, and the like that would do the job in a compact human-carried package.

To evaluate this, condider a baseline scenario of a $P_{\rm pk} = 10\,{\rm kW}$ (peak power) mm-wave (say 95 GHz) source producing a train of $\tau = 10\,{\rm ns}$ RF pulses at a $f_{\rm rep} = 1\,{\rm kHz}$ rep rate, with a conservative $\eta = 10\%$ dc-to-RF efficiency. The RF frequency and pulse width are sized to avoid detection by currently fielded surveillance devices and to allow narrow beamforming in a compact radiator, and the rep rate is chosen in the range of perceived audio symptoms.

The *average* power required is $P_{\rm avg} = P_{\rm pk}\tau f_{\rm rep}/\eta$, or 1 watt. Scaling up to 1 *mega*watt peak power raises the average dc power requirement to 100 W. For suitable battery packs one might go to the canonical website that caters to professional photographers (B&H Photo and Electronics Corporation: bhphotovideo.com). There are listed many choices, for example the Bescor PRB-9NC battery belt (12V, 9A-hr), the Cool-Lux 12V 200 W-hr LCE battery belt, or the larger (shoulder-slung) Bescor 12V 20A-hr battery pack for studio lighting. These retail for $77, $330, and $215 respectively, and would power the megawatt configuration for about an hour of continuous operation.

One could extend this theme to a mobile (van-carried) illuminator, where some two orders of magnitude more battery energy would be feasible by simply configuring series-parallel arrangements of the exemplar battery packs above. However, there are battery packs intended for substantially larger power and energy requirements. For example, lead-acid batteries intended for marine applications can be purchased (e.g., from Amazon) with

ratings of 24V/77A-hr (thus 1.8kW-hr), or 12V/250A-hr (thus 3kW-hr), providing an order of magnitude or more of energy storage compared with the belt-packs above. Another example is the Tesla "powerwall," with 13.5kW-hr capacity; it includes charging and dc-to-ac inverters, that is, 120V/240V 60 Hz ac for charging and during discharge. Although You can't buy these things singly, the established price is $10k, and they are large and heavy (4.5 cu-ft, 250 lb); but they establish a convenient benchmark for considering what could be run from batteries in a vehicle.

Of course, one could as well operate a generator (from the vehicle's engine, or an auxiliary power generator), producing a few kilowatts of continuous power. However, given that a human-portable battery pack would suffice, in terms of power requirements even for a pulsed megawatt illuminator, the only essential role of a van would be to house the RF hardware itself, which is likely to be bulkier and heavier than the power source; moreover, one would want to conceal from sight the antenna and RF hardware.

F APPENDIX: Classified (S)

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G APPENDIX: Classified (TS//SCI)

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H APPENDIX: Briefings

List of Summer 2021 briefings to JASON.

- [redacted], "NIH Review of Current UHI, Medical Responses and Bio Markers"
- [redacted], "Non-Thermal EM Effects - NASA"
- [redacted], "Diplomats' Mystery Illness and Pulsed Radiofrequency Radiation"
- [redacted], "Overview and Analysis of AHI's to Date, In Depth Review of Select Events, and Proposals for New Sensor Technologies"
- [redacted], DARPA
- [redacted] "Keystone UBI, Biological Response Program Overview – DARPA"
- [redacted], "Directed Energy – NASIC"
- [redacted], "LLNL Energy Delivery and Protective Materials"
- [redacted], "UHI Sources and Detections – NSA R4"
- [redacted], [redacted] "Deployed Sensors, Collected Data Presentation – DOS"

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