

Diplomats' Mystery Illness and Pulsed Radiofrequency/Microwave Radiation

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Importance: A mystery illness striking U.S. and Canadian diplomats to Cuba (and now China) “has confounded the FBI, the State Department and US intelligence agencies” (Lederman, Weissenstein, & Lee, 2017). Sonic explanations for the so-called health attacks have long dominated media reports, propelled by peculiar sounds heard and auditory symptoms experienced. Sonic mediation was justly rejected by experts. We assessed whether pulsed radiofrequency/microwave radiation (RF/MW) exposure can accommodate reported facts in diplomats, including unusual ones.

Observations: (1) Noises: Many diplomats heard chirping, ringing or grinding noises at night during episodes reportedly triggering health problems. Some reported that noises were localized with laser-like precision or said the sounds seemed to follow them (within the territory in which they were perceived). Pulsed RF/MW engenders just these apparent “sounds” via the Frey effect. Perceived “sounds” differ by head dimensions and pulse characteristics and can be perceived as located behind in or above the head. Ability to hear the “sounds” depends on high-frequency hearing and low ambient noise. (2) Signs/symptoms: Hearing loss and tinnitus are prominent in affected diplomats and in RF/MW-affected individuals. Each of the protean symptoms that diplomats report also affect persons reporting symptoms from RF/MW: sleep problems, headaches, and cognitive problems dominate in both groups. Sensations of pressure or vibration figure in each. Both encompass vision, balance, and speech problems and nosebleeds. Brain injury and brain swelling are reported in both. (3) Mechanisms: Oxidative stress provides a documented mechanism of RF/MW injury compatible with reported signs and symptoms; sequelae of endothelial dysfunction (yielding blood flow compromise), membrane damage, blood-brain barrier disruption, mitochondrial injury, apoptosis, and autoimmune triggering afford downstream mechanisms, of varying persistence, that merit investigation. (4) Of note, microwaving of the U.S. embassy in Moscow is historically documented.

Conclusions and relevance: Reported facts appear consistent with pulsed RF/MW as the source of injury in affected diplomats.

Nondiplomats citing symptoms from RF/MW, often with an inciting pulsed-RF/MW exposure, report compatible health conditions. Under the RF/MW hypothesis, lessons learned for diplomats and for RF/MW-affected civilians may each aid the other.

1 Introduction

More than two dozen American diplomats in Cuba (Lederman, 2018; Perlez & Myers, 2018) and their families (Lederman & Lee, 2017), plus a smattering of Canadian diplomats in Cuba (Cochrane, 2017; Lederman, Weissenstein, Lee, & Associated Press, 2017) and their families (Panetta, 2017), reportedly developed a "mystery" illness (Associated Press in Washington, 2017; Cochrane, 2017; "Cuba's sonic attacks," 2017; Associated Press, 2017a) that "has confounded the FBI, the state department and US intelligence agencies" (Associated Press in Washington, 2017), "baffling US officials" (Lederman, Weissenstein, & Lee, 2017): "'It's just mystery after mystery after mystery'" (Lederman, Weissenstein, & Lee, 2017). Problems began in 2016, began to be widely reported in 2017, and as of January 2018, "'We are not much further ahead than we were in finding out why this occurred,' Undersecretary of State Steve Goldstein said" (Lederman, 2018). Similar problems first were recognized in China in April 2018, and "a number of diplomats at the US consulate in Guangzhou, China, had been sent home with similar symptoms" (Buckley & Harris, 2018; Harris, 2018a; Perlez & Myers, 2018; Stone, 2018)—by June's end, "at least eight" from the consulate in Guangzhou, and "at least 11" from China more broadly (Myers, 2018).

Media reports have long characterized these so-called health attacks (Associated Press, 2017a, 2017b; Robles & Semple, 2017a, 2017b) as "sonic attacks" (Associated Press in Washington, 2017; Board, 2017; "Cuba's sonic attacks," 2017; Gearan, 2017; Lederman, 2017a; Lederman, Weissenstein, & Lee, 2017; Perlez & Myers, 2018; Associated Press, 2017c).

This characterization persisted despite rejection of sonic explanations by experts (Associated Press in Washington, 2017; Lederman, Weissenstein, & Lee, 2017; Associated Press, 2017c; Zimmer, 2017a, 2017b), for example, "No single, sonic gadget seems to explain such an odd, inconsistent array of physical responses" (Lederman, Weissenstein, & Lee, 2017). According to psychoacoustics expert Joseph Pompei, "'Brain damage and concussions, it's not possible.' . . . 'Somebody would have to submerge their head in powerful ultrasound transducers'" (Lederman, Weissenstein, & Lee, 2017). Some suggested a viral hypothesis (Lederman, 2018), but this fails to explain many features of these cases, including the strange noises associated with inciting events in some, and there is not a known viral illness with a compatible profile of symptoms. Though "officials told senators the US government knew of no weapon, sonic or otherwise, that could produce

the effects seen in the Cuba patients" (Lederman, 2018), to this date, some media sources continue to reference sonic attacks (Perlez & Myers, 2018).

A different explanation is proposed that, it is suggested, better accommodates the facts, including the "odd, inconsistent array of physical responses" (Lederman, Weissenstein, & Lee, 2017) and other "mysterious" and protean features reported. Reported features are assessed for compatibility to known effects of radiofrequency/microwave radiation (RF/MW), particularly pulsed RF/MW. Symptoms and signs are assessed against symptoms and signs reported by people who report health effects from RF/MW exposure, a condition that has been termed "radiofrequency sickness" (Johnson Liakouris, 1998), "microwave syndrome" (Navarro, Segura, Portoles, & Gomez-Perretta, 2003), or to encompass people experiencing problems from exposures beyond a specific part of the electromagnetic spectrum, "electromagnetic hypersensitivity" (Genuis & Lipp, 2012; Hagstrom, Auranen, & Ekman, 2013; Hardell et al., 2008; Leitgeb, 1998; McCarty et al., 2011), "electrosensitivity" (Woolston, 2010; www.es-uk.info; www.esnztrust.org Electrosensitivity New Zealand) or "electrohypersensitivity" (Belpomme, Campagnac, & Irigaray, 2015; Carpenter, 2014; Heuser & Heuser, 2017; Johansson, 2006, 2015; Redmayne & Johansson, 2014).

2 Methods

Features of diplomats' "health attacks"—origins, symptoms, and findings—are delineated and examined in relation to evidence regarding symptoms from RF/MW.

Features to be examined for compatibility with an RF/MW-explanation include the following. Strange noises were heard by some diplomats during apparent inciting episodes (Lederman, Weissenstein, Lee et al., 2017; Stone, 2018). The noises that were heard differed markedly for different diplomats (Lederman, Weissenstein, Lee et al., 2017). Descriptions included high-pitched chirping similar to crickets or cicadas, ringing and grinding (Lederman, Weissenstein, & Lee, 2017). The noises were heard primarily at night (Lederman, Weissenstein, & Lee, 2017). Other diplomats heard no noises (Lederman, Weissenstein, Lee et al., 2017) and were not aware of any inciting episodes—just onset of symptoms. In some cases, noises were confined to "parts of rooms with laser-like specificity" (Lederman, Weissenstein, & Lee, 2017). "Others in the immediate vicinity heard nothing" (Golden & Rotella, 2018). Within the area in which a sound was perceived, it seemed to follow the person around the room (Stone, 2018).

Auditory symptoms are a prominently reported and distinctive feature (though not present in all) and include hearing loss (Associated Press, 2017b; Associated Press in Washington, 2017; Lederman, Weissenstein, & Lee, 2017; Panetta, 2017; Robles & Semple, 2017a; Wilkinson, 2017) and tinnitus (Associated Press in Washington, 2017; Harris, 2018b; Lederman,

Weissenstein, Lee et al., 2017; Panetta, 2017), and, particularly during inciting episodes in some, ear pain (Harris, 2018b; Lederman, 2018).

Other symptoms are protean and vary markedly from individual to individual—"an odd, inconsistent array of physical symptoms"—Lederman, Weissenstein, & Lee, 2017). Sleep symptoms (Associated Press, 2017a; Panetta, 2017; Swanson et al., 2018), headaches (Associated Press in Washington, 2017; Harris, 2018b; Panetta, 2017; Swanson et al., 2018), cognitive dysfunction (Harris, 2018b; Lederman, Weissenstein, & Lee, 2017; Panetta, 2017; Swanson et al., 2018), fatigue (Harris, 2018b; Panetta, 2017), and dizziness (Associated Press in Washington, 2017; Harris, 2018b; Panetta, 2017; Swanson et al., 2018) are prominent among the "nonspecific" symptoms. In some, problems were temporary and apparently recovered with time away from the exposure (Associated Press in Washington, 2017); others experienced persistent problems (Lederman & Lee, 2017; Lederman, Weissenstein, Lee et al., 2017).

Potentially objectively measurable problems with speech (Associated Press in Washington, 2017; Lederman, Weissenstein, & Lee, 2017), balance (Associated Press, 2017a; Associated Press in Washington, 2017; Lederman, Weissenstein, & Lee, 2017; Swanson et al., 2018), and vision (Associated Press, 2017a; Swanson et al., 2018), as well as epistaxis (nosebleed) (Associated Press in Washington, 2017), are a feature in some. Peculiar sensory symptoms of pressure and vibration are reported (Swanson et al., 2018). Brain injury (Associated Press in Washington, 2017; Harris, 2017a; Lederman & Lee, 2017; Lederman, Weissenstein, Lee et al., 2017), white matter abnormalities (Weissenstein, 2018), and brain swelling (Associated Press in Washington, 2017; Lederman, Weissenstein, Lee et al., 2017) have been reported.

To assess compatibility of symptoms in diplomats with those experiencing symptoms from RF/MW, we focus on those who are symptomatic in each group. "Only a minority of embassy staff were stricken" (Stone, 2018), and it is these who have been reported on and studied. The minority who are symptomatic from RF/MW exposures are the appropriate comparator.

Peer-reviewed publications are the primary source of information. However, the most authoritative source for information about symptoms and experiences of individuals is affected individuals themselves, peer review confers no benefit and has no power to adjudicate individuals' reports. For this reason, the peer-reviewed literature to address issues of science is complemented by sources that have elicited and reported on symptoms and experiences of diplomats, or of RF/MW affected individuals, extending to encompass news reports, surveys, statements of affected individuals, or, when applicable, other "gray literature." For diplomats, news and other media reports are complemented by a *JAMA* report focused on neurological symptoms in diplomats (Swanson et al., 2018). Information that references "news" rather than science also cites media sources.

Mechanisms by which RF/MW may cause reported problems are cursorily addressed. Sources of RF/MW reported to affect the comparator group, and potential RF/MW sources of diplomats' symptoms, are briefly reviewed.

3 Results

Table 1 reviews characteristics of noises reported by diplomats in inciting episodes and compatibility with RF/MW. Pulsed RF/MW in the 2.4 to 10,000 MHz range produces perceived noises that resemble sounds "such as a click, buzz, hiss, knock, or chirp," just as diplomats report (Elder & Chou, 2003). Ability to hear RF/MW "sounds" is reported to depend on high frequency hearing, and on low ambient noise (Elder & Chou, 2003) through a phenomenon termed the *Frey effect*. (Synonyms include *microwave auditory effect*, *RF hearing*, and variations of these.) This fits reports that noises were not universally perceived. The requirement for low ambient noise accounts for perception of "sounds" primarily at night (Lederman, Weissenstein, & Lee, 2017). The primary pitch perceived reportedly relates to head dimensions (Elder & Chou, 2003)—in addition to pulse waveform and other characteristics (Lin, 1980)—accounting for different "sounds" perceived by different diplomats. Sounds were localized with "laserlike" specificity in some cases, supposedly defying known physics (Lederman, Weissenstein, & Lee, 2017). This may defy the physics of sound but not the physics of RF/MW: lasers *are* electromagnetic radiation (EMR). One diplomat reported that the sound seemed to follow him within the space in which it was heard (Stone, 2018). Frey sounds also follow the person, often perceived as slightly behind the head, regardless of the body orientation relative to the source of radiation (Bolen, 1988; Elder & Chou, 2003; Frey, 1961). Covering ears did not lessen noise, consistent with RF/MR "sounds" (Tucker, 2018). Frey induction is not governed by average radiation intensity but the energy in a single pulse (Elder & Chou, 2003). (Analogously, if a jackhammer hit each 2 minutes, the low time-averaged pressure would not explain the damage.)

Table 2 reviews diplomats' symptoms and signs, and compatibility of these with RF/MW. Auditory symptoms, including tinnitus, hearing loss, and ear pain or pressure, are prominent in diplomats (Swanson et al., 2018) and in persons affected by RF/MW (Conrad & Friedman, 2013; Halteman, 2011; Kato & Johansson, 2012; Lamech, 2014). Symptoms are protean in both groups. Prevalent among nonauditory nonspecific symptoms are sleep problems, headaches, cognitive problems, and, to a lesser degree dizziness and nausea (Associated Press in Washington, 2017; Conrad & Friedman, 2013; Halteman, 2011; Harris, 2018c; Kato & Johansson, 2012; Lamech, 2014; Lederman, Weissenstein, & Lee, 2017; Swanson et al., 2018). Additional more specific symptoms that are in principle objectively measurable include problems with balance, speech, vision, and epistaxis (nosebleed) (Associated Press in Washington, 2017; Conrad & Friedman, 2013; Halteman, 2011;

Table 1: Features of Noises Reported by Diplomats during Apparent Inciting Episodes.

Diplomats' Reports	Compatibility with RF/MW
Strange noises were heard by many "of the 24 'medically confirmed'" affected U.S. diplomats (Lederman, 2018), during what were perceived as inciting episodes (Lederman, Weissenstein, & Lee, 2017).	Sound ordinarily results from air-pressure waves (which are longitudinal waves—variation occurs along the direction of travel of the wave), whereas radiation arises from electromagnetic waves (which are transverse waves—variation occurs perpendicular to the direction of travel of the wave). In each case, a frequency is defined by the number of cycles of the wave (that pass, say, a given point) per second, for the respective wave type. Though electromagnetic signals are not themselves sound, RF/MW can lead to perceived noises through the so-called Frey effect (Elder & Chou, 2003) (also called the microwave auditory effect or RF hearing). A 1976 Defense Intelligence Agency report stated, "Sounds and possibly even words which appear to be originating intracranially can be induced by signal modulation at very low average-power densities" (Adams & Williams, 1976). A 1988 Air Force Materiel Command report stated, based on knowledge at the time, that "individuals exposed to pulsed RF/MW radiation have reported hearing a chirping, clicking or buzzing sound emanating from inside or behind the head. The auditory response has been observed only for pulsed modulated radiation emitted as a square-wave pulse train. The pulse width and pulse repetition rate are factors that appear to determine the type of sound perceived. . . . James Lin . . . reports that the sensation of hearing in humans occurs when the head is irradiated at an average incident power density level of about 0.1 mW/cm² and a peak intensity near 300 mW/cm². Auditory responses have been observed for a frequency range of 200–3000 MHz and for pulse widths from 1-100 us" (Bolen, 1988). The frequency range within which sounds can be heard was broadened by 2003: it was reported that sounds can be perceived by persons exposed to RF/MW in the 2.4 to 10,000MHz range (Elder & Chou, 2003). It was noted that the same frequency did not produce the same sound from person to person. Ability to hear RF/MW-induced "sounds" (using the term to refer to the perception, not the stimulus) at all depends on individuals' high-frequency hearing (Elder & Chou, 2003), as well as on low ambient noise (Elder & Chou, 2003).
Not all diplomats heard noises (Lederman, Weissenstein, & Lee, 2017).	

Table 1: Continued.

Diplomats' Reports	Compatibility with RF/MW
Among those who heard noises, the noises reported differed markedly for different diplomats (Lederman, Weissenstein, Lee et al., 2017).	In RF hearing/microwave hearing, the "sound" perceived reportedly relates not to the radiation frequency (cycles/sec) but to head dimensions and pulse characteristics (Elder & Chou, 2003; Lin, 1980). This comports with reports that different sounds were heard by different diplomats, even if they were exposed to the same frequency (or, conceivably, frequencies) of radiation. Of note, whether sound is perceived from RF/MW is not governed by the average radiation level but the energy in a single pulse. Injury to cells (in part through membrane damage) is also materially greater with pulsed radiation (Bonnafous, Vernhes, Teissie, & Gabriel, 1999; Shil, Sanghvi, Vidyasagar, & Mishra, 2005). (Analogously, if a jackhammer hit very hard but very briefly at 2 minute intervals, the low time-averaged pressure would not explain the effects produced.) The relatively high proportion of affected diplomats reporting Frey-type noises suggests the possibility of comparatively high intensity of pulses and frequencies within the designated 2.4 to 10,000 MHz range. Frey "sounds" are "similar to other common sounds" "such as a click, buzz, hiss, knock, or chirp," consistent with sounds that diplomats reported (Elder & Chou, 2003). In a 2007 Dutch survey completed by 250 persons with electrosensitivity (ES), queries related to noise included buzzing (reported by 96), hissing (reported by 80), strong low-frequency sounds (reported by 56), and "sound of bells clanging" (reported by 28) (Schooneveld & Kuiper, 2007). The term <i>chirping</i> (if there is a Dutch equivalent) was not included among inquiries. Of note, the "strong low frequency sounds" are potentially consistent with the "blaring, grinding noise" reported by a diplomat ("blaring" indicative of "strong," and "grinding" consistent with low frequency), while the "sound of bells clanging" is consistent with reports of diplomats who awoke to hear ringing "and fumbled for their alarm clocks, only to discover the ringing [clanging] stopped when they moved away from their beds" (Lederman, Weissenstein, Lee et al., 2017).
These noises included a high-pitched "chirping," ringing and "grinding" (Lederman, Weissenstein, & Lee, 2017; Associated Press, 2017c).	

Table 1: Continued.

Diplomats' Reports	Compatibility with RF/MW
	In the Maine Smart Meter survey report (Conrad & Friedman, 2013), comments by affected persons were included. Exemplars involving Frey noises included these: "The noise I have in my head since smart meters is almost unbearable, sleep is at times impossible because it is so loud" (Conrad & Friedman, 2013) and "I became electrically sensitive almost immediately upon smart meter installation. My ears buzz, hum, and click constantly, pressure in the head and ears, . . . agitation and irritability all since the PLC smart meter was placed on my home. . . . I was able to vacation where there was no smart meter installed and it felt as if a vice had been loosened from around my head" (Conrad & Friedman, 2013). A post regarding a woman who removed her smart meter after becoming symptomatic repeated several times that the exposure caused her to hear "grinding" ("Smart meters or no power at all?" 2012), confirming this descriptor as among perceived RF/MW-hearing induced noises. Among those with ES who communicated with the UCSD ES Survey group, one stated that in proximity to "electrosmog producing devices, 'I hear sounds like beehives and similar [buzzing].'" Another stated, "The hissing in my ears is unbearable sometimes." One wrote "annoying noise" was among other symptoms. RF/MW noises do not lessen with ear occlusion, and may intensify (Frey, 1961). [After] "72 Itron AMI smart meters [were installed] near me in my townhome complex. . . I hear a constant buzzing that is driving me crazy. It keeps me awake and makes it hard to think. I am not sure if it is an actual sound, or if it is being generated inside my head, because when I put my fingers in my ears I still hear it. . . . In addition, at about every 15 or 20 minutes, a more intense whine is added that lasts about 12–15 seconds, that hurts and gives me a mild headache which stops when the whine stops. . . . When I go out into the state and regional parks around me where there are NO smart meters for miles, I no longer hear the buzzing and my heart doesn't race." Ability to hear RF/MW-induced sounds at all depends on low ambient noise (Elder & Chou, 2003). Night is generally a time of low ambient noise.
Sound doesn't lessen when cover ears (Tucker, 2018).	
The noises were heard primarily at night (Lederman, Weissenstein, & Lee, 2017).	

Table 1: Continued.

Diplomats' Reports	Compatibility with RF/MW
A sound that has been recorded in Cuba and reported to be "similar" to some sounds heard is consistent with chirping of crickets or cicadas (Lederman & Weissenstein, 2017). Frey effect sounds should not be able to be recorded.	Recorded sounds, if <i>similar</i> to what was "heard" by some, need not <i>be</i> what was "heard." (Just as Frey sounds are "similar to other common sounds," so those other common sounds can resemble the Frey sound.) The recorded sound does not cause symptoms in listeners. The sound does not fit reports by other diplomats of either the character of the sound or of strict sound localization (such as reports that when one moved from the bed, sound disappeared). Some diplomats had cited perceived sounds similar to crickets or cicadas, the recorded noises were reportedly very similar to the chirping of crickets or cicadas that are abundant along the northern coast of Cuba (Weissenstein & Rodriguez, 2017). Since Frey effects can sound like crickets chirping, presumably recordings of crickets chirping could resemble those Frey effect sounds. Dr. Allen Sanborn, an expert in Latin American cicadas, listened to a dozen recordings made by Havana diplomats, and stated, "They sounded to me like cicadas" (Golden & Rotella, 2018). Those deploying causative devices could, of course, capitalize on misguided sonic hypotheses to lead the United States astray by adding a recorded sound resembling Frey sounds; however, there seems little need to postulate this.
There was apparent laser-like localization of sounds in some cases.	For diplomats, "at least some of the incidents were confined to specific rooms or even parts of rooms with laser-like specificity, baffling U.S. officials who say the facts and the physics don't add up" (Lederman, Weissenstein, & Lee, 2017). One incident was described in media as follows: "The blaring, grinding noise jolted the U.S. diplomat from his bed in a Havana hotel. He moved just a few feet, and there was silence. He climbed back into bed. Inexplicably, the agonizing sound hit him again. It was as if he'd walked through some invisible wall cutting straight through his room. Soon came the hearing loss and speech problems" (Lederman, Weissenstein, & Lee, 2017). Even for sounds described as loud, others close by heard nothing (Golden & Rotella, 2018). In claims that "the facts and the physics don't add up" (Lederman, Weissenstein, Lee et al., 2017), it was the physics of <i>sonic</i> devices that was inconsistent. The physics of EMR is, to the contrary, compatible: lasers are themselves focused EMR. Tautologically, EMR can be focused in "laser-like" fashion. A diplomat reported that "a really odd loud noise seemed to follow him in the room" (Stone, 2018). Frey "sounds" are also reported to "follow" the listener, often perceived as slightly behind the head, regardless of the body orientation relative to the source of radiation (Bolen, 1988; Elder & Chou, 2003; Frey, 1961). In other cases, "sounds" are perceived inside or above the head (Cain & Rissmann, 1978; Elder & Chou, 2003; Ingalls, 1967).

Note: Though "sound" refers to air pressure waves, we will refer to what diplomats "heard" as (perceived) sound.

Table 2: Symptoms and Signs.

Diplomats' Symptoms and Signs	Compatibility with RF/MW
Distinctively prominent auditory symptoms	Auditory symptoms are prominent in reports of diplomats' experience, including ear pain or pressure (Swanson et al., 2018), sometimes within minutes of the perceived attack (Lederman, 2018); tinnitus (Associated Press in Washington, 2017; Harris, 2018b; Lederman, Weissenstein, & Lee, 2017; Lederman, Weissenstein, Lee et al., 2017; Panetta, 2017) and hearing loss (Associated Press, 2017a, 2017b; Associated Press in Washington, 2017; Lederman, Weissenstein, & Lee, 2017; Robles & Semple, 2017a; Swanson et al., 2018; Wilkinson, 2017). This, coupled with the strange noises in diplomats' reports, likely launched the sonic theory. These idiosyncratic features are key to winnowing potential causes. Symptoms like headache and fatigue arise with many exposures and in many conditions. New onset of tinnitus and hearing loss is far more distinctive. It is particularly so in the context of the spectrum of other reported symptoms and effects, and in the context of characteristics of instigating episodes. These distinctive auditory problems are similarly prominent in people reporting symptoms from RF/MW (Halteman, 2011; Lamech, 2014) Tinnitus and hearing loss were cited by 80% and 34%, respectively, in the UCSD survey of 202 individuals with current symptoms from EMR, with pulsed RF/MW causing symptoms in the vast majority (Golomb, 2015a). "Initial" symptoms were reported to include tinnitus in 50%, ear pain in 30%, and hearing loss in 11%. Case descriptions shared by affected individuals underscore auditory effects. From the UCSD survey: "I bought a Kindle W-Fi. I charged it not realizing the default setting was 'on.' After 5–10 minutes exposure, I became nauseated, had a headache, loud tinnitus . . . and was dizzy. I turned the Wi-Fi off and the symptoms completely resolved in 5–10 minutes" (Golomb, 2015a). A description by former educator Brinchman (2011) characterizes her abrupt development of headaches and hearing loss following introduction of pulsed RF/MW-emitting smart meters to her (and her neighbors') homes. Similarly, physicians and physician groups that assessed patients with health effects from RF/MW and recognized the connection also highlight effects on hearing. A psychotherapist in Germany with a long-time practice described a new group of patients with a physiological illness profile encompassing organic brain disease, with constellation of symptoms compatible with other reports of RF/MW injury. She was the one to discern the tie between patients' symptoms and their proximity to RF/MW sources (a connection that her patients had often missed obviating noisebo effects as a source; see Table 4), and to note recovery with removal from those sources (Aschermann, 2009). She describes "sudden hearing loss" as among the symptoms (in addition to sleep problems described as an "almost ubiquitous" headache as extremely frequent, also noting, for example, fatigue, cognitive problems, and tinnitus).

Table 2: Continued.

Diplomats' Symptoms and Signs	Compatibility with RF/MW
	A group of 114 physicians, referencing their analysis of medical complaints of 356 people in Oberfranken, signed an open Letter to the Prime Minister of Germany in 2004 (referred to as the Bamberg Appeal), stating, "The pulsed high frequency electro magnetic fields (from mobile phone base stations, from cable-less DECT telephones, amongst others), led to a new, previously unknown pattern of illnesses with a characteristic symptom complex" (Waldman-Selsam, 2004). Prominent and repeated mention is made of hearing loss: "People suffer from one, several or many of the following symptoms: Sleep disturbances, tiredness, disturbance in concentration, forgetfulness, problem with finding words, depressive mood, ear noises; sudden loss of hearing, hearing loss, giddiness; nose bleeds, visual disturbances, frequent infections, sinusitis; joint and limb pains, nerve and soft tissue pains, feeling of numbness, heart rhythm disturbances, increased blood pressure episodes, hormonal disturbances, night-time sweats, nausea. . . . It is no way only a subjective sensitivity disturbance. Disturbances of rhythm, hearing problems, sudden deafness, hearing loss, loss of vision, increased blood pressure, hormonal disturbances, concentration impairments, and others can be proved using scientific objective measures" (Waldman-Selsam, 2004). Note also the mention of "ear noises" (the Frey effect). Some studies that experimentally examine effects of RF/MW on hearing show effects, though not all do (See Table 4 for discussion of "inconsistent" effects.) A material consideration is that evidence is consistent with a vulnerable subgroup. One experimental study in humans found that 60 minutes of close exposure to EMR from a mobile phone "had an immediate effect on HTL [hearing threshold limits] assessed by pure-tone audiogram and inner ear (assessed by DPOAE) in young human subjects. It also caused a number of other otologic symptoms" (Alsanosi et al., 2013). Of note, melatonin, which can be depressed by EMR (see Table 4) and is low in those with EHS (Belpomme et al., 2015), protects against oxidative radiation injury (see Table 4), including to the inner ear (Karaer et al., 2015). Pulsed RF/MW (more than continuous) has been shown to increase tympanic temperature, even when, for instance, colonic temperature is not increased (Frei, Jauchem, & Heinimets, 1988). Since blood flow is critical for cooling and oxidative stress leads to endothelial dysfunction and may compromise blood flow, affected individuals (see below; by hypothesis those with greater oxidative stress effects) may experience greater impairment in blood flow—so less cooling and also impaired delivery (via impaired blood flow) of oxygen, glucose, and other energy substrates as well as antioxidant defenses. The downstream effects of oxidative stress (e.g., apoptosis, inflammation; see below) and impaired cell energy/ mitochondrial dysfunction (cell dysfunction and death) may contribute to auditory pathology.

Table 2: Continued.

Diplomats' Symptoms and Signs	Compatibility with RF/MW
	In a study examining the histopathology of cochlear nuclei of rats "exposed continuously for 30 days" to "a GSM-like 2100 MHz EMF" "with a signal level (power) of 5.4 dBm (3.47 mW) to simulate the talk mode on a mobile phone," compared to a control group of rats not similarly exposed, "an increase in neuronal degeneration and apoptosis in the auditory system" was observed in the RF/MW exposed group (Celiker et al., 2016). "The histopathologic analysis showed increased degeneration signs in the study group ($p = 0.007$). In addition, immunohistochemical analysis revealed increased apoptotic index in the study group compared to that in the control group ($p = 0.002$)" (Celiker et al., 2016). In another animal study, "a prominent effect of EMS [electromagnetic stimulation] was . . . severe cochlear damage and permanent sensorimotor hearing loss in experimental animals" (Counter, 1993).
Protean symptoms	Beyond the auditory symptoms, the profile of symptoms in diplomats varies from person to person. Different people report markedly different symptoms (Lederman, Weissenstein, Lee et al., 2017). It was said that "the symptoms and circumstances reported have varied widely; making some hard to tie conclusively to the attacks" (Lederman, 2017b), and "The cases vary deeply: different symptoms, different recollections of what happened. That's what makes the puzzle so difficult to crack" (Lederman, Weissenstein, Lee et al., 2017). Reported symptoms encompass sleep problems (Associated Press, 2017a, 2017b; Panetta, 2017), headaches (Associated Press, 2017a; Lederman, Weissenstein, & Lee, 2017; Panetta, 2017; Robles & Semple, 2017a), cognitive problems (Associated Press, 2017a; Lederman, Weissenstein, & Lee, 2017), nausea (Lederman, Weissenstein, & Lee, 2017), fatigue (Panetta, 2017), and dizziness (Lederman, Weissenstein, & Lee, 2017; Robles & Semple, 2017a). Similar concerns had been raised with RF/MW injury. As Aschermann noted (translated from German), "In the <i>Deutsche Aerzteblatt</i> [official journal of the German medical association—Bundesaerztekammer] did an article ask the incredulous question: How could so many different symptoms possibly be attributed to one common underlying mechanism?" (Aschermann, 2009). Despite the protean character of symptoms, multiple survey studies verify that a strikingly reproducible suite of protean symptoms <i>are</i> reported in setting after setting, and in people citing development of symptoms in response to EMR including RF/MW (see Table 3). The profile of symptoms is strongly similar from study to study, with sleep/fatigue, headache, and cognitive problems commonly topping the list and auditory and visual symptoms, dizziness, and nausea figuring in it.

Table 2: Continued.

Diplomats' Symptoms and Signs	Compatibility with RF /MW
	A similar primary list (sometimes augmented with a few additional symptoms, often including heart rhythm problems) is mentioned in other settings. Aschermann's (2009) analyses of 65 patients cite symptoms of learning concentration and behavioral problems, headaches, insomnia, exhaustion, tinnitus, hearing loss, dizziness, nerve and soft tissue pain, "inner agitation," as well as arrhythmia problems. In the 2004 Bamberg Appeal signed by 114 physicians to the German prime minister, based on analysis of 356 patients: "The pulsed high frequency electro magnetic fields (from mobile phone base stations, from cable-less DECT telephones, amongst others), led to a new, previously unknown pattern of illnesses with a characteristic symptom complex. People suffer from one, several or many of the following symptoms: Sleep disturbances, tiredness, disturbance in concentration, forgetfulness, problem with finding words, depressive mood, ear noises, sudden loss of hearing, hearing loss, giddiness, nose bleeds, visual disturbances, frequent infections, sinusitis, joint and limb pains, nerve and soft tissue pains," also nausea, and "feeling of numbness, heart rhythm disturbances, increased blood pressure episodes, hormonal disturbances, night-time sweats. . . . The symptoms occur in temporal and spatial relationship to exposure. It is no way only a subjective sensitivity disturbance. Disturbances of rhythm, hearing problems, sudden deafness, hearing loss, loss of vision, increased blood pressure, hormonal disturbances, concentration impairments, and others can be proved using scientific objective measures" (Waldman-Selsam, 2004). Among individuals participating in a physiological provocation study examining heart rate variability with RF /MW, among 25 patients, 40% of whom believed themselves to be moderately or severely electrosensitive, "the most common symptoms of exposure to electrosmog, as identified by this group of participants, included poor short-term memory, difficulty concentrating, eye problems, sleep disorder, feeling unwell, headache, dizziness, tinnitus, chronic fatigue" (Havas et al., 2010). Of note, the same symptoms also arise in the vulnerable subgroup of persons who develop health problems following other exposures that share a documented ability to cause mitochondrial impairment and oxidative stress (Chen et al., 2017; Golomb et al., 2014; Golomb, Koslik et al., 2015; Koslik, Hamilton, & Golomb, 2014; Steele, 2000). However, the profile, which symptoms dominate, differs from exposure to exposure, based on factors such as what part(s) of the body the exposure may differentially reach and whether additional mechanisms of injury are involved that potentiate damage to one domain.

Table 2: Continued.

Diplomats’ Symptoms and Signs	Compatibility with RF/MW
	Sleep and auditory effects are clearly disproportionately represented, in diplomats and with RF/MW exposure, relative to their prevalence following other exposures that cause oxidative stress. The strong effects on sleep may relate to depressions in melatonin that can be produced with EMR / RF/MW (see Table 4). Auditory effects are addressed above. A 1990 study commissioned in response to a petition by residents who cited adverse health experiences from a shortwave radio transmitter in their small town of Schwarzenburg, funded in part by Swiss Telecom, reported that sleep disruption in association with transmitters related directly to the EMR field strength of the transmitter and affected 55% of those over age 45 (Alpeter et al., 1995; Lamech, 2014). (There the denominator is <i>not</i> restricted to those who were symptomatic.) A 1988 Air Force Materiel Command reports that “pulsed RF/MW radiation was reported to have an analeptic effect in animals. Experimental results presented by R. D. McAfee in 1971 showed that anesthetized animals could be awakened by irradiation from a pulsed 10 GHz RF/MW source. . . Experiments conducted on rats showed that these animals were aroused from states of deep sleep by irradiation” (Bolen, 1988). The prominence of auditory effects (see above for more on these symptoms) may relate in part to the absence of a skull structure to protect the inner ear, producing an incident stimulus that is of greater effective intensity. The coherence of symptoms in response to RF/MW, with findings in Cuba (and China) diplomats, adds further support to the case for a common cause within each group – and across the two groups.

Table 2: Continued.

Compatibility with RF /MW	
Diplomats' Symptoms and Signs	
Symptoms that are (potentially) objectively measurable: <i>speech</i> (Associated Press, 2017a; Associated Press in Washington, 2017; Lederman, Weissenstein, & Lee, 2017); <i>vision</i> (Associated Press, 2017a). <i>balance</i> (Associated Press, 2017a; Lederman, Weissenstein, & Lee, 2017). Nosebleeds in some (Associated Press in Washington, 2017; Golden & Rotella, 2018).	The symptoms reported in media and Swanson et al. (2018) for diplomats, extending to the more specific (e.g., dizziness/balance, vision and speech problems), are also reported in survey studies of those affected by RF /MW (see Table 3). <i>Speech</i> problems, mentioned in diplomats, were also among symptoms elicited and reported in a survey study examining effects of RF /MW following "smart meter" introduction in Australia (Lamech, 2014). Reported cases illustrate speech problems arising following RF /MW exposure. In a case referenced in the <i>LA Times</i>, a woman reported that if someone fails to turn off their cellphone on entering her home, she gets symptoms within 2 hours: "After four hours I can't speak anymore"" (Woolston, 2010). In a case described in a 2015 Australian presentation on ES (Weller, 2015), "Within hours, it felt as if someone had tied a thick rubber band around her head. Then came nausea, fatigue, ringing in her left ear—an onslaught of maladies all at once, and she had no idea why. . . . A week or two into the job, whatever was affecting her wasn't abating, and before long her speech became so jumbled that she couldn't form a complete sentence in front of an audience. . . . She went outside to inspect the place and found no fewer than 17 new 'smart' electricity meters strapped to the side of the building." In a case reported to UCSD investigators, new-onset right-sided ear pain and hearing loss attended the inciting episode (seated for 6 hours, unknowingly, directly across the wall from a bank of multiple smart meters for a building, slightly toward her right) along with vise-like headache, concentration problems, and two nights of no sleep (followed by chronic lesser sleep impairment), and, abating over months, continued to be triggered, always exclusively or predominantly on the right side, by previously tolerated RF /MW exposures thereafter. Many months later, left ear predominant ear symptoms developed for the first time. A bank of smart meters was identified to the left of where she had sat, hidden by plants so missed in an initial reconnaissance. That occasion, the only one with left predominant ear and hearing symptoms, was accompanied by speech difficulty, which resolved over about a week. In these two cases, aphasia was associated with left predominant ear symptoms (Broca's area, damage of which leads to expressive aphasia, is left prefrontal). It is an empirical question whether left-predominant auditory involvement will prove more often tied to affected speech. <i>Balance</i> is multifactorial, involving vision, muscle strength, and vestibular function, for example. In some media reports of diplomat health, the term <i>vertigo</i> is used (Harris, 2018b, 2018c). Balance and vestibular testing were performed in diplomats (Swanson et al., 2018). Clinical examinations and objective measures raised concern for balance problems in 81% (higher than the percent reporting subjective dizziness or balance problems) (Swanson et al., 2018).

Table 2: Continued.

Diplomats' Symptoms and Signs	Compatibility with RF/MW
	Vestibular function involves the same (eighth) cranial nerve as hearing. Vertigo, hearing loss, and tinnitus can arise (as adverse effects) as a triumvirate (Porto Arco, 2003; Sepcic et al., 2010). Dizziness more generally, in contrast to vertigo, is a nonspecific finding that arises with many forms of brain insult, including brain hypoperfusion (low blood flow). Of note, cerebral hypoperfusion has been reported in persons with symptoms following RF/MW (Belpomme et al., 2015). In some surveys of RF/MW-affected individuals, dizziness and balance are queried together (Lamech, 2014); other surveys use only the term <i>dizziness</i>. Individual reports of balance and dizziness problems were included among participant narrative reports in the Maine survey—for example: “Balance problems have worsened since installation of the smart meter, leading to several falls” (Conrad & Friedman, 2013) and “I could not understand the dizziness which was scary. I actually thought I had a brain tumor all of a sudden” (Conrad & Friedman, 2013). The Cuba diplomat study considered nausea as a vestibular symptom (Swanson et al., 2018). Though it need not necessarily be, it was linked to dizziness in some RF/MW/EMR affected cases: “Daily nausea and dizziness” (Conrad & Friedman, 2013). Loss of balance, with dizziness and disorientation, was identified as one of six clusters of symptoms seen in each of two smart meter surveys from different nations, with the clusters represented nearly in the same order: (1) sleep disruption, (2) headache, (3) ringing or buzzing in ears, (4) fatigue, (5) loss of concentration, memory or learning ability, and (6) disorientation, dizziness, or loss of balance (Powell, 2015). <i>Vision:</i> Vision is affected by oxidative stress and mitochondrial impairment (see Table 4, mechanisms) (Argun et al., 2014; Beatty, Koh, Phil, Henson, & Boulton, 2000; Javaheri, Khurana, O’Hearn T, Lai, & Sadun, 2007; King, Gottlieb, Brooks, Murphy, & Dunatef, 2004; Liang, Green, Wang, Alssadi, & Godley, 2004; Tolan et al., 2001), not just to the eye but to cortical systems involved in vision (Pachalska et al., 2002). Effects of these mechanisms include optic nerve damage (Javaheri et al., 2007; Qi, Lewin, Sun, Hauswirth, & Guy, 2007; Rucker, Hamilton, Bardenstein, Isada, & Lee, 2006), age-related macular degeneration (Beatty et al., 2000; Feher et al., 2005; Feher, Papale, Mannino, Gualdi, & Balacco Gabrieli, 2003; Liang & Godley, 2003; Modi, Heckman, & Saffer, 1992; Tolan et al., 2001; Yu, Wu, & Lin, 1997), retinal thinning (Sandbach et al., 2001), and cataracts (Gul, Rahman, Hasnain, Salim, & Simjee, 2008; Karslioglu et al., 2005; Ottonello, Foroni, Carta, Petrucco, & Maraini, 2000; Tarwadi & Agte, 2004; Taylor, Jacques, & Epstein, 1995). Where brain swelling ensues (see Table 4), this can affect the shape of the lens, affecting vision.

Table 2: Continued.

Diplomats' Symptoms and Signs	Compatibility with RF/MW
	Effects of RF/MW on the eye and on vision have long been reported (Birenbaum, Groszof, & Rosenthal, 1969; Bolen, 1988; Cleary, 1980; Cutz, 1989; Daily, Wakim, Herrick, Parkhill, & Benedict, 1952; McCally, Farrell, Barger, Kues, & Hochheimer, 1986; Williams & Finch, 1974; Zaret, 1973). Particular attention has gone to effects on the lens, and on cataracts. RF/MW, via oxidative mechanisms, promotes aging of the lens, which can lead to cataracts. Cataracts have been a reported complication, sometimes in young people, among persons working with microwave radiation (Birenbaum et al., 1969; Bolen, 1988; Cleary, 1980; McCally et al., 1986; Zaret, 1973). A Swiss study (Hassig, Jud, & Spiess, 2012) documented increased cataracts in calves born near cell towers: "We examined and monitored a dairy farm in which a large number of calves were born with nuclear cataracts after a mobile phone base station had been erected in the vicinity of the barn. Calves showed a 3.5 times higher risk for heavy cataract if born there compared to Swiss average. All usual causes such as infection or poisoning common in Switzerland could be excluded." Vision problems are reported in RF/MW-affected persons. In a study in Spain, in persons in proximity to two GSM (Global System of Mobile Communications) cell tower base stations, analysis of the closer group, with exposure in the range 0.25–1.29 V/m², in a model adjusted for age, sex, and distance, showed that vision problems were elevated with an odds ratio of 5.8 (95% CI 1.7–19.8, $p = 0.005$) (Oberfeld, Navarro, Portoles, Maestu, & Gomez-Perretta, 2004). Eleven percent reported problems with eyes or vision in the Australian smart meter study. Since this includes respondents who are unaffected, rates are lower than in purely symptomatic individuals (Lamech, 2014). Twenty-Six percent of survey participants reported eye/vision problems in the Halteman smart meter impacts survey (Halteman, 2011). Vision problems were reported by 17% as "severe and new," by 38% as "moderate and new," and by 12% as "severe and worsened" in the Maine smart meter survey (Conrad & Friedman, 2013). An assessment of neurological problems in U.S. diplomats in Cuba underscores the potential importance of eye movement dysfunction (Swanson et al., 2018), which is also tied to oxidative and mitochondrial mechanisms (Chen, Li, Wu, Qi, & Wu, 1998; Dodson, Patten, Hyman, & Chu, 1976; Goto, Koga, Horai, & Nonaka, 1990; Hyman, Patten, & Dodson, 1977; Kao, 1994; Land, Hockaday, Hughes, & Ross, 1981; Pineda et al., 2004; Schaefer, Blakely, Griffiths, Turnbull, & Taylor, 2005; Smits, Westenberg, van Hal, van Engelen, & Overeem, 2012).

Table 2: Continued.

Diplomats' Symptoms and Signs	Compatibility with RF/MW
Peculiar sensory symptoms of "vibration" and "pressure" reported (Swanson et al., 2018)	<i>Epistaxis</i> (nosebleed): In a study in Selbitz, Bavaria, nosebleed was significantly more frequently reported ($p = 0.01$) in those less than 200 m from a cell phone base station than 200 m to 400 m away (Eger & Jahn, 2010). Nosebleed was a reported symptom in each of several surveys of ES and symptoms associated with RF/MW, including in a study of smart meter symptoms (Conrad & Friedman, 2013; Golomb, 2015a; Halteman, 2011; Lamech, 2014) (see Table 3). The Bamberg appeal (on behalf of 114 physicians referencing assessment of medical complaints of 356 people with symptoms from cell tower base stations and DECT phones in their homes in Oberfranken) noted the more characteristic RF/MW symptoms (above) as well as nosebleed (Waldman-Selsam, 2004). Comments from participants in survey studies include the following (all from Conrad & Friedman, 2013): "Severe headaches, gushing nosebleeds for the first time ever They all went away when the smart meter was removed"; "After the first day I was getting bloody noses and not understanding"; "Nosebleeds, nausea, dizziness, . . . ringing ears and intermittent strong agitation When I am away from wireless devices the symptoms subside"; "Had it not been for the severe nose bleeds I'm not sure I would ever have found out what was causing my health problems". "Associated sensory symptom" of "pressure" or "vibration" were reported in 43% and 14%, respectively, in a neurological evaluation of diplomats (Swanson et al., 2018). The distinctive sensory symptoms of "pressure" and "vibration" are also reported by subsets of those who report symptoms from RF/MW. Neither were commonly elicited as symptoms in surveys. However, some surveys listed head pressure separately from headache, and in some cases, it was more frequent. Eye pressure (Halteman, 2011) and ear pressure (Conrad & Friedman, 2013) have also been reported in surveys of RF/MW/EMR-affected persons. The UCSD ES survey did include "internal pressure," which was reported as a symptom in 71% of participants who cited symptoms from EMR/RF/MW (Golomb, 2015a). Spontaneous reports of vibration symptoms by different EMR/RF/MW affected persons, shared in a different survey study, include the following (all from Conrad & Friedman, 2013): "I experienced internal shaking and vibrating throughout my body" (along with sleep, mood, headache, head pressure, and other problems, after smart meter installation); "I can't think clearly, or find words when speaking; my body feels like it is vibrating"; and "Have uncontrollable jelly-like quivering throughout whole body." In online comments posted in response to articles on related topics, in which persons describe their ES symptoms, statements include "vibration through my body" (F. Wallace, 2017), and "I have a smart meter on my house and I have been experiencing strange vibrations when I watch TV or use the computer" (Wright, 2013). An email to us from an affected patient (9-2017) sharing her symptoms stated that it "feels like my brain is vibrating and spinning at night—and my tinnitus gets much worse."

Table 2: Continued.

Diplomats' Symptoms and Signs	Compatibility with RF/MW
Brain swelling in some diplomats (Associated Press in Washington, 2017; Lederman, 2017a; Weissenstein, Lee et al., 2017).	1. RF/MW may alter blood-brain barrier function via oxidative stress. (a) An analysis reported that of 100 peer-reviewed studies examining whether low-intensity RF/MW causes oxidative stress, 93 found that it did (Yakymenko et al., 2015). (b) Oxidative stress disrupts the blood-brain barrier (Al Ahmad, Gassmann, & Ogunshola, 2012; Blasig, Mertsch, & Haseloff, 2002; Enciu, Gherghiceanu, & Popescu, 2013; Haorah et al., 2007; Hurst, Heales, Dobbie, Barker, & Clark, 1998; Katsu et al., 2010; Lochhead et al., 2010; Nittby et al., 2009; Salford, Brun, Stureson, Eberhardt, & Persson, 1994; Sirav & Seyhan, 2009, 2011; Takemori, Murakami, Kometani, & Ito, 2013; Tang et al., 2015). (c) Consistent with this, blood-brain barrier disruption has been shown in multiple studies with RF/MW (Nittby et al., 2008, 2009; Salford et al., 1994; Sirav & Seyhan, 2009; Soderqvist, Carlberg, Hansson Mild, & Hardell, 2009; Soderqvist, Carlberg, & Hardell, 2009; Tang et al., 2015). Other studies have not shown blood-brain barrier effects (de Gannes et al., 2009; Finnle, Blumbergs, Cai, Manavis, & Kuchel, 2006; Finnle et al., 2002; Franke, Ringelstein, & Stogbauer, 2005; Franke, Streckert et al., 2005; Fritze et al., 1997; McQuade et al., 2009). Studies vary in many respects (e.g., exposure duration, EMR exposure characteristics, model (in vivo versus in vitro, animal, age), delay between exposure and blood-brain barrier assessment, and blood-brain barrier assessment used, for example). The blood-brain barrier is functional, and barrier function need not be affected for all substances equally. (d) Since genetics of oxidative stress management (De Luca et al., 2014) and levels of key antioxidants (Belpomme et al., 2015) relate to both RF/MW injury and oxidative stress, these factors, together with specifics of the RF/MW exposure, may guide blood-brain barrier disruption with RF/MW. (e) A study that examined gene expression in the brains of rats exposed to GSM radiation, radiation that encompasses the multiple frequencies and pulsed waveforms present in GSM exposures, identified altered gene expression of a marker of blood-brain barrier function (Belyaev et al., 2006). 2. Altered blood-brain barrier can lead to brain edema and "malignant brain edema" (Adair, Baldwin, Kornfeld, & Rosenberg, 1999; Witt, Mark, Sandoval, & Davis, 2008). (Oxidative stress-associated blood-brain barrier disruption is, for instance, thought to underlie neuroleptic-induced cerebral edema (Elmorsy, Elzalabany, Elsheikha, & Smith, 2014).)

Table 2: Continued.

Diplomats' Symptoms and Signs	Compatibility with RF/MW	
	Findings are reported to be compatible with traumatic brain injury (Harris, 2017a, 2017b, 2018c; Harris & Goldman, 2017a, 2017b; Rogers, 2017).	
		3. Among case experiences, perceived head pressure occurs with brain swelling and is reported by many with ES. As also noted in relation to the sensory symptom of “pressure,” some surveys collate head pressure separately from headache (which, in some studies, it surpasses: Conrad & Friedman, 2013; Lamech, 2014; Schooneveld & Kuiper, 2007). One survey included eye pressure (Halteman, 2011), and in one, several participants spontaneously reported ear pressure (Conrad & Friedman, 2013). Communications to the UCSD ES study included the write-in comment, “Brain feels like it’s swelling” (Golomb, 2015a). One man with severe ES who communicated with the UCSD study group and shared documentation of his approval for Social Security disability for his ES reported that the severe brain swelling he experienced in response to EMR had led an eyeball to be pushed from the socket. 1. Based on findings in an fMRI study of electrosensitive individuals it was stated that “the differential diagnosis for the abnormalities seen on the fMRI includes head injury” (Heuser & Heuser, 2017). 2. Six of the 10 ES individuals assessed reported prior head injury (Heuser & Heuser, 2017). However, 4 did not, and also showed evidence consistent with brain injury. Moreover, prior head injury is reported to also be present in at least some, but an unstated fraction of, affected diplomats (Stone, 2018). 3. Head injury could predispose to ES. Head injury, like RF/MW, promotes oxidative stress, and blood-brain barrier disturbance; and melatonin (which is low in those with ES), protects from these effects in head injury (Dehghan, Khaksari Hadad, Asadikram, Najafpour, & Shahrokhi, 2013; Ding et al., 2014; Ozdemir et al., 2005; Senol & Naziroglu, 2014), as it protects against injury from radiation (Argun et al., 2014; Bardak, Ozer Turk, Ozguner, Durmus, & Delibas, 2000; Bhatia & Manda, 2004; El-Missiry, Fayed, El-Sawy, & El-Sayed, 2007; Goswami & Haldar, 2014a, 2014b; Goswami, Sharma, & Haldar, 2013; Guney et al., 2007; Jang et al., 2013; Karaer et al., 2015; Karsliglu et al., 2005; Kim, Shon, Ryoo, Kim, & Lee, 2001; Koc, Taysi, Buyukokuroglu, & Bakan, 2003a, 2003b; Liu, Ren, Yang, Zhao, & Mei, 2014; Manda, Anzai, Kumari, & Bhatia, 2007; Manda & Reiter, 2010; Manda, Ueno, & Anzai, 2007, 2008; Naziroglu, Tokat, & Demirci, 2012; Oliinyk & Meshchyshev, 2004; Ortiz et al., 2015; Sainz et al., 2008; Sener, Atasoy et al., 2004; Sener, Jahovic, Tosun, Atasoy, & Yegen, 2003; Sharma & Haldar, 2006; Shirazi et al., 2011; Shirazi, Mihandoost, Mohseni, Ghazi-Khansari, & Rabie Mahdavi, 2013; Taysi, Koc, Buyukokuroglu, Altinkaynak, & Sahin, 2003; Taysi et al., 2008; Vasin et al., 2004; Yilmaz & Yilmaz, 2006)—and from RF/MW . . . (Ayata et al., 2004; Aynali et al., 2013; Koylu, Mollaoglu, Ozguner, Naziroglu, & Delibas, 2006; Lai & Singh, 1997; Meena et al., 2014; Naziroglu, Celik et al., 2012; Oksay et al., 2012; Oktem, Ozguner, Mollaoglu, Koyu, & Uz, 2005; Ozguner, Bardak, & Comlekci, 2006; Ozguner, Oktem, Armagan et al., 2005; Sokolovic et al., 2008; Tok, Naziroglu, Dogan, Kahya, & Tok, 2014; S. Xu et al., 2010).

Table 2: Continued.

Diplomats' Symptoms and Signs	Compatibility with RF/MW
White matter abnormalities reported (Weissenstein, 2018) in some diplomats.	4. One RF/MW affected man who communicated with the UCSD study group indicated his ES was precipitated by a serious occupational head injury. (He also had occupational exposure to EMR, but until the head injury, it had not affected him.)
	5. The study did not report the presence or absence of features indicative of greater severity of head injury, such as loss of consciousness or sequelae. Both because of this and point 5, there is no clarity about whether prior head impacts were in fact greater in number or intensity than in the general population. But it might be expected that past head injury would be a risk factor.
	6. Given findings consistent with low melatonin in those with ES (Belpomme et al., 2015), this condition (and/or common cause) may also predispose to more significant damage from a given impact and character of head injury, so there is a so greater likelihood that a given head impact causes problems and is remembered and reported as a head injury.
White matter abnormalities reported (Weissenstein, 2018) in some diplomats.	7. ES symptoms are sometimes experienced as similar to a head injury. For instance, a Rhode Island teacher likened effects experienced with RF/MW to a concussion ("Math teacher raises concerns about WIFI comparing the effects to a concussion," 2014). Just as it is important to avoid even minor head trauma following traumatic concussion until healing has occurred, so avoidance of RF/MW (or more generally EMR) aggravation may prove important following pulsed RF/MW injury. RF/MW injury may be cumulative (Sadchikova & Glotova, 1973), and in addition to the intensity-duration profile, the interval between exposures may be important in the clinical course (Zaret, 1973).
	In diplomats: "Medical testing has revealed that some embassy workers had apparent abnormalities in their white matter tracts that let different parts of the brain communicate" (Weissenstein, 2018).
	1. White matter changes were observed in some with ES, in the fMRI study of persons affected by RF/MW/EMR (Heuser & Heuser, 2017). 2. Oxidative stress and mitochondrial dysfunction (to which RF/MW can contribute; see Table 4) are associated with white matter injury (Back et al., 2005; Casta, Quackenbush, Houck, & Korson, 1997; Ikeda, Choi, Yee, Murata, & Quilligan, 1999; Miller, Lawrence, Mondal, & Seegal, 2009; Miyamoto et al., 2013; Munoz-Cortes et al., 2013; Rosenzweig & Carmichael, 2013).

Table 2: Continued.

Diplomats' Symptoms and Signs	Compatibility with RF /MW
	Among potential mechanisms, oxidative stress increases vulnerability of proteins (and, e.g., lipids, DNA, RNA) to autoimmune attack, which can include attacks on myelin (Gelderman et al., 2007; Iborra, Palacio, & Martinez, 2005; Iuchi et al., 2010; Kalluri, Cantley, Kerjaschki, & Neilson, 2000; Kumagai, Jikimoto, & Saegusa, 2003; Liu et al., 2003; Maes et al., 2013; Profumo, Buttari, & Rigano, 2011; Shah & Sinha, 2013; Wang, Cai, Ansari, & Khan, 2007). Indeed, antibodies directed to O-myelin were reported in a subset of the 675 persons with ES who were included in a French study (Belpomme et al., 2015), affirming one mechanism by which white matter changes might occur. 3. Following GSM radiation exposure (study cited previously), examination of gene expression in rat brain showed alterations in myelin-related products (myelin-related glycoprotein) (Belyaev et al., 2006).

Lamech, 2014; Lederman, Weissenstein, & Lee, 2017; Swanson et al., 2018). Peculiar sensory symptoms are reported in both, including pressure and vibrations (Conrad & Friedman, 2013; Swanson et al., 2018). Reported brain findings have included brain swelling, problems consistent with traumatic brain injury, and white matter abnormalities. Each such feature is also observed in those with symptoms ascribed to RF/MW.

Table 3 lists symptoms commonly reported in diplomats, together with percentages reporting each symptom, for symptoms assessed in the neurological appraisal of Cuba diplomats or mentioned in news reports (Associated Press in Washington, 2017; Harris, 2018c; Lederman, Weissenstein, & Lee, 2017; Swanson et al., 2018). These symptoms (when elicited) are ranked by prevalence in surveys of persons exposed to specific sources of RF/MW or with symptoms ascribed to EMR exposure (Conrad & Friedman, 2013; Halteman, 2011; Kato & Johansson, 2012; Lamech, 2014). Fractions of symptomatic diplomats who report each symptom (Swanson et al., 2018) appear similar to fractions of those symptomatic with EMR symptoms, who do so. Comparing rates in diplomats (Swanson et al., 2018) to those in a peer-reviewed study of EMR-affected individuals (Kato & Johansson, 2012) on symptoms tallied in both, symptom rates were: headache, 81% versus 81%; cognitive problems, 81% versus 81%; sleep problems, 86% versus 76%; irritability, 67% versus 56%; nervousness/anxiety, 52% versus 56%; dizziness 67% versus 64%; and tinnitus, 57% versus 63% (Kato & Johansson, 2012; Swanson et al., 2018). Thus, rates conform closely.

The rates of symptoms reported for diplomats appear within reported variation for studies of persons affected by RF/MW/EMR. Sleep problems were reported somewhat less frequently in EMR-affected persons in the Kato study (76%), than in diplomats, but reported sleep problems, or their by-product, fatigue (for which prevalence was not recorded in the diplomat study), dominate the number one symptom position in studies of RF/MW affected persons (see Table 3), and prevalence of sleep problems was higher than for diplomats in some other studies of RF/MW-affected persons (Golomb, 2015a). Of note, the Kato study was performed in Japan, where the traditional diet is rich in fish, which supplies the long-chain omega-3 fatty acids that reportedly benefit sleep and reduce irritability (Conklin et al., 2007; Peet & Horrobin, 2002), the two symptoms that were more than 3% lower than in affected diplomats.

The protean character of symptoms in diplomats (Lederman, 2017a), as for RF/MW-affected individuals, has led some to infer that a single cause cannot account for all. But a number of reports, in a number of nations and settings, tie RF/MW exposure (in vulnerable individuals) to each of the problems reported in diplomats. The coherence of findings in those citing affects of RF/MW, with findings in diplomats, supports a common cause within each group and across the two groups. Of note, a protean suite of generally the same symptoms, though in a different distribution, is reported in other conditions that are tied to mitochondrial alteration and oxidative

Table 3: Symptoms in Diplomats: Comparison to Symptom Rankings in Survey Studies That Report Symptoms with EMR or in Those with ES.

Citation	Cuba Diplomats	Australia, 2014	United States, 2011				France, 2002	Japan, 2012	United States, 2015 ^a	Netherlands, 2007	Sweden, 2006	Finland, 2013	Turkey, 2017
			(Wireless Utility Meter Safety Impacts Survey)	2013 ^a (Maine Smart Meter Health Effects Survey & Report)	2013 ^a (Maine Smart Meter Health Effects Survey & Report)	2013 ^a (Maine Smart Meter Health Effects Survey & Report)							
Study of diplomats (Swanson et al., 2018) News media	NA	Smart meter exposure	Smart meter exposure	Smart meter exposure	Smart meter exposure	Proximity to cell phone base station	ES	ES	ES	ES, acute phase	ES, acute phase	ES, acute phase	Cell phone use symptoms during
EMR- or ES-related characteristic													

Table 3: Continued.

	Cuba	Australia, 2014	United States, 2011 (Wireless Utility Meter Safety Impacts Survey)	United States, 2013 ^a (Maine Smart Meter Health Effects Survey & Report)	France, 2002	Japan, 2012	United States, 2015 ^a	Netherlands, 2007	Sweden, 2006	Finland, 2013	Turkey, 2017
Sample characteristics	About 24 U.S. and 2 Canadian diplomats to Havana reporting symptoms attributed to "health attacks" in news: 24 U.S. embassy community members with neurological findings often seen after mild traumatic brain injury/concussion (Swanson et al., 2018)	92 residents of Victoria, Australia, after exposure to smart meter radiation	318 U.S. respondents from 28 states	210 respondents, 68% ES (142) ^b	530 people living near cellular phone base stations	75 Japanese with ES or sensitive to EMF	202 persons with current ES	250 Dutch respondents with ES	22 with ES-ranked symptoms; most common were listed (not ranked)	194 with ES	2150 students in 26 high schools in Turkey
All have symptoms	Yes	No	No	No	No	Yes	Yes	Yes	Yes	Yes	No

Table 3: Continued.

	Cuba Diplomats	Australia, 2014	United States, 2011 (Wireless Utility Meter Safety Impacts Survey)	United States, 2013 ^a (Maine Smart Meter Health Effects Survey & Report)	France, 2002	Japan, 2012	United States, 2015 ^a	Netherlands, 2007	Sweden, 2006	Finland, 2013	Turkey, 2017
		#5	#5	#2/#4	#4, #7	#3 (81%)	#3 (85%)	#2, #13	Yes	#7, #10	#4, #5
Cognitive	81% Swanson et al. (2018). Also see Lederman (2017a); Panetta (2017); Associated Press (2017d).										
Stress	67% irritability;	#11	#2	#8/#7 (agitation)	#6 (irritability)	#9 and #10. For #6 in “initial “irritation” and “anxiety” (56% and 55%).	#5 (80%)				
anxiety	57% nervousness; 52% more emotional; 29% sadness.										
irritability	Swanson et al. (2018)										
Tinnitus	57% Swanson et al. (2018). Also see Lederman, Weinstein, Lee et al. (2017), Panetta (2017).	#3	#4	#3/#2	Not queried (except as “hearing”)	#7 (63%)	#5 (80%)	Not in main symptom list, but based on number affected in auditory symptom list, #13	Not queried		

Table 3: Continued.

			United States, 2011 (Wireless Utility Meter Safety Impacts Survey)	United States, 2013 ^a (Maine Smart Meter Health Effects Survey & Report)	France, 2002	Japan, 2012	United States, 2015 ^a	Netherlands, 2007	Sweden, 2006	Finland, 2013	Turkey, 2017
	Cuba Diplomats	Australia, 2014	#6	#10/#9	#1	#1 (and possibly #5, "sluggish" in the head (85%))	"Exhaustion" was a write-in symptom (not queried).	#1	Yes	#6	#1
Fatigue	Not elicited ^c in Swanson et al. (2018). Mentioned in news media; Panetta (2017)										
Dizziness or balance	67% ^d Swanson et al. (2018). Also see Lederman, Weissenstein, Lee et al. (2017); Panetta (2017); Robles and Sample (2017a)	#7	#7	#7/#7	#14	#6 (64%)	#4 Initial: 49%	#11	Yes	#12	#9

Table 3: Continued.

	Cuba Diplomats	Australia, 2014	United States, 2011 (Wireless Utility Meter Safety Impacts Survey)	United States, 2013 ^a (Maine Smart Meter Health Effects Survey & Report)	France, 2002	Japan, 2012	United States, 2015 ^a	Netherlands, 2007	Sweden, 2006	Finland, 2013	Turkey, 2017
Vision problems	76% Swanson et al. (2018). Also see Associated Press (2017a).	#12	#8	#10/#11	#12	—	#8 in initial Symptoms (38%)	#6	—	#13 (photosen- sitivity)	#10
Nausea	Associated Press in Washington (2017); Lederman, Weinstein, Lee et al. (2017); Panetta (2017)	#9	#12	—	—	—	#9 “Gastroin- testinal symptoms” (64%). Nausea not separately asked.	—	Yes “Symptoms from the gas- trointestinal tract.”	#20	#15
Epistaxis (nose bleed)	Not elicited in Swanson et al. (2018). Mentioned in news/media: Associated Press in Washington (2017); Golden & Rotella (2018).	#17	#13	#15 in symptoms that intensified. New onset in several write-ins.	—	—	“Nosebleeds” as a write-in symptom (not queried).	—	—	—	—

Table 3: Continued.

	Cuba Diplomats	Australia, 2014	United States, 2011 (Wireless Utility Meter Safety Impacts Survey)	United States, 2013 ^a (Maine Smart Meter Health Effects Survey & Report)	France, 2002	Japan, 2012	United States, 2015 ^a	Netherlands, 2007	Sweden, 2006	Finland, 2013	Turkey, 2017
Hearing loss	43% Swanson et al. (2018). Also see Associated Press (2017b); Associated Press in Washington (2017); Panetta (2017); Robles & Semple (2017a); Wilkinson (2017)	#18 (with ear pain)	—	—	#5	—	#11 (34%)	#3	—	—	#14
Speech problems	Not elicited ^e in Swanson et al. (2018). Mentioned in Associated Press in Washington (2017)	#30	—	—	—	—	f	—	—	—	—
Comment			g	h	i		j	k		l	

Table 3: Continued.

Note: — = Not queried. Surveys in the smart meter era were prioritized for inclusion; proximity of emitting devices to homes may make these more comparable to diplomat experience. Studies of ES were also prioritized, as these focus on those who are symptomatic, providing symptom rates better suited for comparison to those in affected diplomats. Other studies on similar themes report similar findings. (An exception is that older studies from Scandinavia that focused on exposure to video display terminals from that time report high rates of skin problems.) For instance, in a 2007 study of 85 persons living near the first mobile phone station antenna in Menoufiya governorate, Egypt reported that "the prevalence of neuropsychiatric complaints as headache (23.5%), memory changes (28.2%), dizziness (18.8%), tremors (9.4%), depressive symptoms (21.7%), and sleep disturbance (23.5%) were significantly higher among exposed inhabitants than controls: (10%), (5%), (5%), (0%), (8.8%), and (10%), respectively ($P < 0.05$)." Sleep, headache, and cognitive again topped the list in frequency (Abdel-Rassoul et al., 2007).

Some studies focus not on ranking, but dose-effect/distance relation. For instance, in Selbitz, Bavaria, those within 200 m of a cell phone base station were compared on reported symptoms to those 200 m to 400 m away and were found to report significantly more sleep problems, headache, concentration problems, "cerebral affections," depression, auditory/vestibular problems, visual problems, GI problems, dizziness, and nosebleed along with cardiovascular problems, joint problems, infections, and skin problems ($p = 0.01$ for dizziness and nosebleed, $p = 0.001$ for the rest; Eger & Jahn, 2010). A 2003 survey study of the "microwave syndrome" "in Murcia, Spain, in the vicinity of a Cellular Phone Base Station working in DCS-1800MHz" reported that symptoms included fatigue, irritability, headache, nausea, insomnia, depression, discomfort, difficulty in concentration, memory loss, visual dysfunction, auditory dysfunction, dizziness, (and several other symptoms) (Navarro et al., 2003). These were more prevalent within 150 m of the station, relative to more than 250 m, in most cases significantly so. It was noted that symptoms abated with removal from the RF/MW source (Navarro et al., 2003). A follow-on study examined rates of problems in relation to measured electric fields and showed significance for 13 of 16 assessed symptoms, with symptom odds ratios as high as 59 (Oberfeld et al., 2004).

Our rankings do not include as a symptom "onset of electromagnetic hypersensitivity syndrome" or "aggravation of electromagnetic hypersensitivity syndrome." We used the highest ranking if several cognitive queries were used (e.g., memory problems or concentration difficulties) or several head queries were used (e.g. headache, head pressure, heat or strange sensation in head), and exclude later exemplars of the category in ranking the lower-ranked items.

^aThere was no barrier to participation from outside the United States, but participants are predominantly from the United States.

^bSixty-eight percent of participants had ES ($N = 142$) of whom 63% felt certain their exposure to smart meter was responsible for initiating the ES. Of the 49 who were ES before smart meter exposure, all 49 (100%) stated that smart meter exposure made their ES not only worse but "much worse." "Though fatigue was not elicited, it is noted that a number reported a "good day bad day" pattern in which mental or physical exertion on one day led to exacerbation for several days.

^dSeparates out balance (67%) and dizziness (63%) and includes nausea (7%) in this category.

^eSpeech problems were not elicited, but speech audiometry, speech therapy, and speech pathology consultation are each mentioned totaling at least six references.

Table 3: Continued.

^f "Aphasia" was a write-in symptom (not queried).
^g Seventy-three percent women; 93% over age 40; 43% over age 60; 78% from California; 49% characterize selves as EMF sensitive.
^h The first number is severe or moderate and new; the second number is severe and new. Pressure in head and headaches were queried separately. The overlap is uncertain. The higher ranking (pressure in head) was used. Concentration and memory were queried separately. The overlap is uncertain. The higher ranking (concentration problems) was used.
ⁱ Memory and concentration were queried separately, ranked #4 and #7 in the original. Combined might be higher. The higher ranking is used. This analysis provides values at different distances. Orderings for the closest distance are used. Ordering shifts slightly with longer distances, but in general, the more frequently reported symptoms remain the more frequently reported.
^j Ratings are based on (videotaped) Commonwealth Club slide presentation. Additional symptoms were elicited but not presented.
^k Notes buzzing ears, hissing sounds, loss of hearing, strong low-frequency sounds, earaches, and sound of bells clanging in 96, 80, 64, 545, 38, and 28 participants
^l This assesses acute symptoms. It also gives fractions of who report those symptoms before the acute phase, but it is unclear whether someone who reports a symptom (say, headaches, dizziness) before exposure had those symptoms only occasionally.
Note: Percentages are given for diplomats (chosen for being symptomatic) and rankings for studies of persons reporting symptoms with EMR/ RF /MW (not restricted to acute stage).

stress (Golomb et al., 2014; Golomb & Evans, 2008; Golomb, Koslik, & Redd, 2015), mechanisms that each promote the other (Lee & Wei, 1997; Wei & Lee, 2002). RF/MW is tied to these mechanisms (Barnes & Greenebaum, 2015, 2016; Gao, Hu, Ma, Chen, & Zhang, 2016; Turedi et al., 2015; Yakymenko et al., 2015; Yuksel, Naziroglu, & Ozkaya, 2016; Zhu et al., 2014). However the distinctive prominence of sleep and auditory symptoms, the peculiar somatic sensory experiences of pressure and vibration, and the noises perceived during apparent inciting episodes are relatively distinctive features—distinctive to diplomats' reports and reported RF/MW problems.

Table 4 reviews several mechanism considerations. Central to this is the critical role of oxidative stress and the relevance of oxidative stress to potential auxiliary mechanisms, such as mitochondrial dysfunction, blood-brain barrier disruption, membrane alterations, impaired blood flow, apoptosis, effects on voltage-gated calcium and anion channels, and triggering of autoimmune reactions. (In some cases, effects are reciprocal—oxidative stress promotes mitochondrial dysfunction, calcium channel effects, inflammation, and autoimmunity—which in turn can promote oxidative stress.) One analysis found that of 100 evaluated studies that examined the relationship of low-level RF/MW to oxidative stress in biological systems, 93% supported a connection (Yakymenko et al., 2015). A role for oxidative stress in RF/MW/EMR-affected persons is cemented by evidence that gene polymorphisms adverse to antioxidant defense are significantly more prevalent in persons experiencing symptoms from RF/MW/EMR (De Luca et al., 2014). In addition, levels of a particular antioxidant, melatonin, known to be critical for RF/MW and broader EMR defense are consistently low in affected persons (assessed by a urinary metabolite) (Belpomme et al., 2015). Oxidative stress has been tied to each of the symptoms and conditions reported in diplomats and RF/MW-affected persons.

Also noteworthy is the repudiation of psychogenic causation in the evaluation of diplomats (Stone, 2018; Swanson et al., 2018), which holds for RF/MW-affected persons as well. Case narratives for those affected by RF/MW underscore that for many, symptoms developed and progressed when affected parties as yet had no knowledge that an RF/MW-emitting device had been introduced or that one could cause problems (Conrad & Friedman, 2013; Golomb, 2015a). A Swiss Telecom-funded study found that sleep problems related to the electromagnetic field strength of the transmitter and did not correlate with personality traits tied to worry about health (Altpeter et al, 1995; Lamech, 2014). The circumstance that some report being affected severely by levels of exposure that cause others no problem is reviewed in the context of effect modification, variations in antioxidant defenses, and demonstrated variable involvement of secondary mechanisms such as autoimmune activation (Belpomme et al., 2015). In fact, analogous marked differences in harm or development of health effects are well known for other exposures, such as peanuts, penicillin, and pesticides. For EMR-affected persons (De Luca et al., 2014), as for many other

Table 4: Mechanism Considerations.

Oxidative stress, mediated by free radicals, is involved in RF/MW injury.	Oxidative stress refers to a kind of injury against which “antioxidants” relatively protect, in which “reactive oxygen species” or “free radicals” produce changes/damage that can affect, for instance, lipids, proteins, DNA, and RNA. Mitochondria, the primary source of energy for cells (and they regulate many other phenomena such as steroid hormone production and apoptosis) are a leading source and target of oxidative stress (Gruber, Schaffer, & Halliwell, 2008; Kowald, 2001; Lee & Wei, 1997; Sastre, Pallardo, & Vina, 2003; Wei, 1998). That is, mitochondrial injury not infrequently accompanies oxidative stress and has been shown with RF/MW (see below). RF/MW produces oxidative stress. As above, in an analysis of 100 studies examining if low-level RF/MW produced oxidative injury, it was reported that about 93 found that it did (Yakymenko et al., 2015). Oxidative stress and mitochondrial dysfunction are implicated in the symptoms and health effects that have been reported by diplomats and RF/MW-affected persons (Adamczyk-Sowa et al., 2014; Berr, Balansard, Arnaud, Roussel, & Alperovitch, 2000; Bonne & Muller, 2000; Brubaker, Mohney, & Pulido, 2009; Carelli, Ross-Cisneros, & Sadun, 2002; Feng et al., 2010; Fetoni et al., 2013; Finsterer, 2008; Fukui et al., 2002; Hoshino, Tamaoka, Ohkoshi, Shoji, & Goto, 1997; Ikeda-Douglas, Zicker, Estrada, Jewell, & Milgram, 2004; Insel, Moore, Vidrine, & Montgomery, 2012; Jeyakumar, Williamson, Brickman, Krakovitz, & Parikh, 2009; Kilic, Selek, Erel, & Aksoy, 2008; Koga & Nataliya, 2005; Koillinen, Jaaskelainen, & Koski, 2009; Kuruppu & Matthews, 2013; Liang et al., 2004; Manwaring et al., 2007; Massin et al., 1995; Neri et al., 2006; Ottonello et al., 2000; Reynolds, Laurie, Mosley, & Gendelman, 2007; Riordan-Eva, 2000; Rosen, 2008; Sandbach et al., 2001; Savastano, Brescia, & Marioni, 2007; Seidman, Khan, Bai, Shirwany, & Quirk, 2000; Sharma et al., 2013; Someya et al., 2009; Tiwari & Chopra, 2013; Vurucu et al., 2013; D. Wallace, 2001; Yamasoba et al., 2007; Zhang et al., 2013; Zoric et al., 2008). For instance, oxidative stress is tied to tinnitus, antioxidants modestly alleviate it, and markers of oxidative stress in tinnitus are reported to be greater in jugular blood (near the ear) than the more commonly measured brachial blood (Neri et al., 2006; Savastano et al., 2007; Van Campen, Murphy, Franks, Mathias, & Toraason, 2002). Two findings substantially cement a role for oxidative stress in RF/MW health effects. First, persons who are “electrosensitive” (i.e., who experience symptoms at levels of radiation that many others tolerate) are significantly more likely to harbor gene variants that confer less avid protection against oxidative injury (De Luca et al., 2014). This is an extremely important finding. People cannot manipulate their genes in response to suggestibility and did not know their genes when they reported their sensitivity status. This powerfully supports a causal role for oxidative stress in the injury experienced.
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Table 4: Continued.

Second, a French study in electrically and chemically sensitive individuals (93% with ES), found consistently low levels of a urinary melatonin metabolite (Belpomme et al., 2015). Since melatonin is an antioxidant that protects against damage to many toxins, but has been shown in numerous studies to be particularly vital for defense specifically against oxidation injury due to radiation across the electromagnetic spectrum (Argun et al., 2014; Bhatia & Manda, 2004; El-Missiry et al., 2007; Goswami & Haldar, 2014b; Goswami et al., 2013; Griefahn, Kunemund, Blaszkewicz, Lerchl, & Degen, 2002; Guney et al., 2007; Inaïda et al., 2000; Jang et al., 2013; Karaer et al., 2015; Karslioglu et al., 2005; Kim et al., 2001; Koc, Tayşi, Buyukokuroglu, & Bakan, 2003a, 2003b; Manda, Anzai et al., 2007; Manda & Reiter, 2010; Manda et al., 2008; Naziroglu, Tokat, & Demirci, 2012; Ortiz et al., 2015; Sener, Atasoy et al., 2004; Sener, Jahovic et al., 2003; Sharma & Haldar, 2006; Shirazi et al., 2011, 2013; Tayşi et al., 2003, 2008; Vasin et al., 2004; Yilmaz & Yilmaz, 2006), including due to RF/MW (Ayata et al., 2004; Aynali et al., 2013; Koylu et al., 2006; Lai & Singh, 1997; Meena et al., 2014; Naziroglu, Celik et al., 2012; Oksay et al., 2012; Oktem et al., 2005; Ozguner et al., 2006; Ozguner, Oktem, Armagan et al., 2005; Sokolovic et al., 2008, 2013; Tok et al., 2014), this dovetails with the genetic data to compellingly support a role for oxidative stress and to show that those with ES (those who experience symptoms with radiation that others tolerate) are also experiencing greater cellular and subcellular injury from this radiation.

Many studies show the importance of antioxidant defenses, including melatonin, in protection against RF/MW injury. For instance, melatonin and, to a lesser degree, caffeic acid protect against cell phone-induced oxidative stress in rats, and melatonin increased the activity of other endogenous antioxidant enzymes, superoxide dismutase (SOD), glutathione peroxidase (GPx) and catalase, which were depressed with the cell phone radiation (Ozguner et al., 2006). Melatonin protected against laryngo-tracheal oxidative injury from wireless (2.45 GHz) radiation in rats (Aynali et al., 2013). It also protected against skin oxidative injury in an experimental mobile phone model in rats (Ayata et al., 2004). It protected against 900 MHz microwave radiation-induced lipid peroxidation in rats (Koylu et al., 2006); reversed the oxidative damage of microwaves to rat testes including protecting testosterone level and sperm count, and protecting against DNA fragmentation (a marker of cell death) (Meena et al., 2014) and protected against oxidative damage from cell phone radiation to rat brain (Sokolovic et al., 2008). Melatonin protects against oxidative damage from Wi-Fi to the lens of rats (Tok et al., 2014). Vitamins E and C protect against "900 MHz radiofrequency-induced histopathologic changes and oxidative stress in rat endometrium" (Guney, Ozguner, Oral, Karahan, & Mungan, 2007). Ginkgo biloba protected against cell phone-induced oxidative injury in rat brain (Ilhan et al., 2004). And so on.

Table 4: Continued.

	Antioxidants work together, for instance, to recycle one another to the reduced form in which they are active as antioxidants. The importance of antioxidant defenses in protection against radiation injury from RF/MW extends what is well known for injury from radiation throughout the electromagnetic spectrum, including so-called ionizing radiation (which includes gamma)—for instance, “A positive correlation was found between GPx activity, glutathione content and cell survival following ionizing irradiation”; Bravard et al., 2002). Glutathione depletion increased with gamma radiation–induced DNA damage (Dutta, Chakraborty, Saha, Ray, & Chatterjee, 2005) and cell death (Dethmers & Meister, 1981). Glutathione determined the survival “shoulder” for X-ray radiation in hypoxic cells (Evans, Taylor, & Brown, 1984), and melatonin protected against X-ray-induced lung injury (Jang et al., 2013). Melatonin protected against radiation–induced cataract (Karslioglu et al., 2005) and increased activity of other critical antioxidant enzymes, SOD and GPx. SOD protected against fractionated radiation–induced esophagitis (and reduced the effect of that radiation on glutathione) (Epperly et al., 2001). Melatonin protected against UVB radiation–induced oxidative skin injury (Goswami & Haldar, 2014a, 2014b), as did glutathione (Hanada, Gange, & Connor, 1990) and chocolate, which is rich in antioxidant polyphenols (Williams, Tamburic, & Lally, 2009). Melatonin has specifically been reported to protect the inner ear against radiation injury in rats exposed to “radiotherapy” at 4 KHz to 6 KHz (Karaer et al., 2015) A role for oxidative stress in radiation injury transcends labels of “ionizing” versus “nonionizing,” and “thermal” versus “nonthermal” radiation. For this reason, those labels are of questionable utility in understanding radiation damage. A number of studies report that EMR, including but not limited to RF/MW, can depress melatonin (Bergqvist et al., 1997; Burch, Reif, & Yost, 1999, 2008; Fernie, Bird, & Petitclerc, 1999; Griefahn et al., 2002; Halgauge, 2013; Qin et al., 2012; Reiter, 1993a, 1994; Weydahl, Sothorn, Cornelissen, & Wetterberg, 2000). Evidence suggests that (like virtually all other biological effects), a subgroup is more vulnerable (Parry et al., 2010; Wood, Loughran, & Stough, 2006). (Note that sunlight, which provides EMR of a kind “expected” evolutionarily, is well recognized to govern (depress) melatonin, toward producing day-night and seasonal effects.) Light (a portion of the electromagnetic spectrum) inhibits melatonin as part of establishing circadian and seasonal rhythms (Gammack, 2008; Glickman, Byrne, Pineda, Hauck, & Brainerd, 2006; Navara & Nelson, 2007). Evolution did not plan for man-made radiation sources, and one hypothesis is that such radiation sources may induce similar effects in some people.
Radiation may depress melatonin—more so in some—and, in part through depressed melatonin, may depress other antioxidants	

Table 4: Continued.

"EMF [electromagnetic fields] are known to affect Ca²⁺ homeostasis and suppress melatonin activity in a wide wavelength range. Ca²⁺ ions in pinealocytes are involved in regulation of cAMP synthesis that mediates conversion of serotonin into melatonin. Their leakage from pinealocytes results in a decrease of the cAMP level and thereby suppresses production of melatonin" (Rapoport & Breus, 2011). Longterm radar workers reportedly have increased serotonin and depressed melatonin, consistent with this impaired conversion and effects in the RF/MW frequency range (Singh, Mani, & Kapoor, 2015). Electronic repair workers have also been reported to have lower melatonin than controls and more sleep problems (El-Helaly & Abu-Hashem, 2010).	Melatonin and its derivatives, though better known for effects on sleep, provide a critical antioxidant defense system that protects against toxicity of an extraordinary array of toxins and conditions (Abdel Moneim et al., 2015; Antunes Wilhelm, Ricardo Jesse, Folharini Bortolatto, & Wayne Nogueira, 2013; Bandyopadhyay, Ghosh, Bandyopadhyay, & Reiter, 2004; Baxi, Singh, Vachhrajani, & Ramachandran, 2013; Chabra, Shokrzadeh, Naghshvar, Salehi, & Ahmadi, 2014; Chen, Gao, Li, Shen, & Sun, 2005; Ebaid, Bashandy, Alhazza, Rady, & El-Shehry, 2013; El-Missiry et al., 2014; Fuentes-Broto et al., 2010; Garcia-Rubio, Matas, & Miguez, 2005; Jindal, Garg, Mediratta, & Fahim, 2011; Korkmaz, Uzun, Cakatay, & Aydin, 2012; Laothong et al., 2010; Mehta et al., 2014; Skaper, Floreani, Ceccon, Facci, & Giusti, 1999; Sousa & Castilho, 2005; et al., 2014; Shokrzadeh et al., 2014; Thomas & Mohanakumar, 2004; Uygun et al., 2013; S. C. Xu et al., 2010; L. Zhang et al., 2013; Aranda et al., 2010; Carrillo-Vico et al., 2005; Das, Belagodu, Reiter, Ray, & Barik, 2008; El-Sokkary, Nafady, & Shabash, 2010; Esrefoglu, Gul, Ates, & Selimoglu, 2006; Esrefoglu, Gul, Emre, Polat, & Selimoglu, 2005; Fagundes, Gonzalo, Arruebo, Plaza, & Muriello, 2010; Y. K. Gupta, Gupta, & Kohli, 2003; Hu, Yin, Jiang, Huang, & Shen, 2009; Kacmaz et al., 2005; Kerman et al., 2005; Omurtag, Tozan, Sehirli, & Sener, 2008; Ozacmak, Barut, & Ozacmak, 2009; Ozacmak, Sayan, Arslan, Altaner, & Aktas, 2005; Ozelcik, Soyoz, & Kilinc, 2004; Rao & Chhunchha, 2010; Rezzani, Buffoli, Stacchiotti, & Bianchi, 2005; Sadir, Deveci, Korkmaz, & Oter, 2007; Sahna, Parlakpinar, Turkoz, & Acet, 2005; Sahna, Parlakpinar, Vardi, Cigremis, & Acet, 2004; Saravanan, Sindhu, & Mohanakumar, 2007; Suke et al., 2006; Tunes, Montilla, Del Carmen Munoz, Feijoo, & Salcedo, 2004; Wang, Wei, Wang et al., 2005; Wang, Wei, Zhang et al., 2005; Watanabe et al., 2004; Zavodnik et al., 2004) (Abdel-Wahab, Arafat, El-Mahdy, & Abdel-Naim, 2002; Bagchi et al., 2001;
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Table 4: Continued.

Behan, McDonald, Darlington, & Stone, 1999; Bruck et al., 2004; Cadenas & Barja, 1999; Chen, Lin, & Chiu, 2003; Dabbeni-Sala, Floreani, Franceschini, Skaper, & Giusti, 2001; El-Sokkary, 2000; Gazi, Altun, & Erdogan, 2006; Hara et al., 2001; Herrera et al., 2001; Karbownik & Reiter, 2002; Lankoff, Banasik, & Nowak, 2002; Martin et al., 2002; Mayo, Tan, Sainz, Lopez-Burillo, & Reiter, 2003; Mayo, Tan, Sainz, Natarajan et al., 2003; Montilla, Tunez, Munoz de Agueda, Gascon, & Soria, 1998; Mor et al., 2003; Morishima et al., 1998, 1999; Ortega-Gutierrez et al., 2002; Othman, El-Missiry, & Amer, 2001; Popov et al., 2015; Princ, Maxit, Cardalda, Batlle, & Juknat, 1998; Sener, Kacmaz et al., 2003; Sener, Paskaloglu et al., 2004; Sener, Sehirli, & Ayanoglu-Dulger, 2003; Shen et al., 2002; Shifow, Kumar, Naidu, & Ratnakar, 2000; Shokrzadeh et al., 2015; Soyoz, Ozcelik, Kilinc, & Altuntas, 2004; Spadoni et al., 2006; Sutken et al., 2007; Tomas-Zapico et al., 2002; Tunez et al., 2003).
For this reason, to the extent that EMR does depress melatonin, it is expected to potentiate the array of adverse health outcomes tied to these toxins, and other sources of injury.
Again, melatonin specifically protects against radiation injury at frequencies across the electromagnetic spectrum (Bardak et al., 2000; Cruz et al., 2003; Dogan et al., 2017; Goswami & Haldar, 2014a; Guney et al., 2007; Jang et al., 2013; Karaer et al., 2015; Kim et al., 2001; Koc et al., 2003a, 2003b; Koylu et al., 2006; Liu et al., 2014; Manda, Anzai et al., 2007; Manda & Reiter, 2010; Manda et al., 2008; Naziroglu, Celik et al., 2012; Oliinyk & Meshchyshen, 2004; Ortiz et al., 2015; Sener, Atasoy et al., 2004; Sener, Jahovic et al., 2003; S. Sharma & Haldar, 2006; Sokolovic et al., 2008, 2013; Taysi et al., 2003, 2008; Tok et al., 2014; Yilmaz & Yilmaz, 2006).
A study examining gene expression in rat brain reported that brain expression of N-acetyltransferase-1, the rate-limiting enzyme in melatonin production (Reiter, 1993b), had significantly reduced expression following 915 MHz GSM-consistent RF/MW radiation (encompassing pulsed RF/MW) in rats, fold difference 0.48 ± 0.13 , $p < 0.0025$ (Belyaev et al., 2006).
Suppressed melatonin or sleep deprivation in turn increases damage to the pineal gland (Lan, Hsu, & Ling, 2001), which produces most of the circulating melatonin. Thus, sufficiently depressed melatonin can beget still further depressed melatonin—and heightened vulnerability to injury from future EMR exposure. The ability to sustain adequate melatonin production in the face of EMR/RF/MW, may be a critical determinant of pineal vulnerability. The pineal gland has high antioxidant needs (Lan et al., 2001; Razygraev, 2010), and in the absence of such protections, it is vulnerable to involution (Lin'kova, Poliakova, Kvetnoi, Trofimov, & Sevost'anova, 2011; Polyakova, Linkova, Kvetnoy, & Khavinson, 2011).

Table 4: Continued.

Age-related involution of the pineal gland may help to explain why more middle-aged persons are reportedly affected by ES than younger people (Gruber, Palmquist, & Nordin, 2018), though presumably younger adults may be more exposed to technology. (Older persons, however, may have had more years of EMR exposure and injury may be cumulative (Sadchikova & Glotova, 1973).)

Melatonin supports the levels and activity of other antioxidants, including in the setting of radiation exposures (Karslioglu et al., 2005; Ozguner et al., 2006; Tok et al., 2014). Modest exposure to oxidative stressors (including from radiation) in persons or animals or plants whose system is not overwhelmed can lead to antioxidant upregulation, a phenomenon called *oxidative preconditioning*, seen with many sources of limited oxidative stress, including limited exposure to radiation (Chen, 2006). In part because of this, the net effect of an oxidant exposure on antioxidant levels depends on factors like intensity and duration of exposure, other oxidative exposure (so, mitochondrial dysfunction state), and the status of antioxidant defenses, as well as time from exposure to assessment. Some studies in some systems show antioxidant upregulation (Irmak et al., 2002) or mixed direction effects on different antioxidants (Tok et al., 2014), but many show depression of assessed antioxidants following EMR exposure (Duan et al., 2013; Goswami & Haldar, 2014a, 2014b; Martinez-Samano, Torres-Duran, Juarez-Oropeza, Elias-Vinas, & Verdugo-Diaz, 2010) or specifically RF/MW exposure (Akpınar, Oztürk, Ozen, Agar, & Yargicoglu, 2012; Bahreymi Toossi et al., 2017; Ceyhan et al., 2012; Esmekaya, Ozer, & Seyhan, 2011; Guney et al., 2007; Megha et al., 2015; Ozguner, Altinbas et al., 2005; Oktem et al., 2005; Ozguner et al., 2006; Ozguner, Oktem, Armagan et al., 2005; Ozguner, Oktem, Ayata, Koyu, & Yilmaz, 2005; Tok et al., 2014; Yurekli et al., 2006). Such depressions, coupled with melatonin depressions, may increase vulnerability to future EMR exposures, particularly where genetics provide for less effective variants of one or more antioxidants (De Luca et al., 2014).

It is expected that mitochondrial impairment (J. Gruber et al., 2008; Lee & Wei, 1997; Sastre et al., 2003; Wei, 1998) or brain inflammation (sometimes itself a result of oxidative stress, amenable to reduction with melatonin; Guney et al., 2007; Halliday, 2005), since associated with greater production of free radicals and an expected less favorable balance of oxidative stress to antioxidant defenses, may be a risk factor for problems with the added oxidative stress from RF/MW or from the depression in antioxidant defenses to which RF/MW may contribute.

Table 4: Continued.

RF/MW may depress xenobiotic protections	RF/MW is reported to depress butyrylcholinesterase (McRee, 1980), a key xenobiotic defense; low levels are tied to higher cardiovascular and all-cause mortality (Calderon-Margalit, Adler, Abramson, Gofin, & Kark, 2006).
Oxidative stress contributes to auxiliary mechanisms of radiation injury, such as mitochondrial dysfunction.	Oxidative stress contributes to multiple documented auxiliary mechanisms of RF/MW damage that likely contribute to health effects in subsets, including membrane alterations—cell membranes (Benderitter, Vincent-Genod, Pouget, & Voisin, 2003) and mitochondrial membranes (Shonai et al., 2002; Thomas, Gebicki, & Dean, 1989; Vayssier-Taussat et al., 2002; Wang et al., 2002), blood-brain barrier disruption (Al Ahmad et al., 2012; Baricello et al., 2011; Freeman & Keller, 2012; Gasche, Copin, Sugawara, Fujimura, & Chan, 2001; Haorah, Knipe, Leibhart, Ghorpade, & Persidsky, 2005; Haorah et al., 2007; Hurst et al., 1998; Lochhead et al., 2010; Nittby et al., 2009; Salford et al., 1994; Zehender et al., 2013), effects on voltage gated calcium channels (Cui et al., 2012) affected by and affecting oxidative stress—(Nishiyama, Nakano, & Hitomi, 2010; Pall, 2015)—but also on voltage-gated anion channels that are an important part of the outer mitochondrial membrane (Ferrer, 2009) potentially contributing to mitochondrial impairment and amplification of oxidative stress, EEG spiking (Naziroglu, Celik et al., 2012), impaired mitochondrial function (Aitken, Bennetts, Sawyer, Wiklendt, & King, 2005; Xu et al., 2010)—bidirectionally related to oxidative stress (Houston, Nixon, King, De Iulius, & Aitken, 2016; Mancuso, Coppede, Migliore, Siciliano, & Murri, 2006; Wei & Lee, 2002)—and protected by melatonin (Tan, Manchester, Qin, & Reiter, 2016), impaired blood flow—e.g., via oxidative stress-driven endothelial dysfunction (Engin, Sepici-Dincel, Gonul, & Engin, 2012; Indik, Goldman, & Gaballa, 2001; Jarasuniene & Simaitis, 2003; Loscalzo, 2002), autoantibodies (Ahsan, Ali, & Ali, 2003; Fiorini et al., 2013; Gilgun-Sherki, Melamed, & Offen, 2004; Kirkham et al., 2011; Kumagai et al., 2003; Maes et al., 2013; Ryan, Nissim, & Winyard, 2014), and apoptosis (Aoki et al., 2001; Bresgen et al., 2003; Espino et al., 2010; Filomeni, Cardaci, Da Costa Ferreira, Rotilio, & Ciriolo, 2011; France-Lanord, Brugg, Michel, Agid, & Ruberg, 1997; Li et al., 2015; Li et al., 2008; Salido & Rosado, 2009; Yalcinkaya et al., 2009; Zhang, Zhang, Rabbani, Jackson, & Vujaskovic, 2012)—programmed cell death, which in turn triggers inflammation and coagulation activation (Reutlingsperger & van Heerde, 1997). Laboratory correlates for some of these were reported in ES participants in the French study: about 15% of those with ES had elevated markers of blood-brain barrier permeability; 29% in those with ES (23% in those with ES and multiple chemical sensitivity, MCS) had antibodies to O-myelin (Belpomme et al., 2015).

Table 4: Continued.

Melatonin considerations: RF/MW/EMR versus diplomats	While depressions in a melatonin metabolite were the norm in participants with ES in a French study (Belpomme et al., 2015), this need not necessarily be the case for diplomats, even if a related cause (pulsed RF/MW) and related processes (e.g., tied to oxidative stress) are involved in symptom induction. In persons with “ES,” lowered defenses are needed for nominally modest exposures to produce problems. But if exposures in affected diplomats were more intense or otherwise injurious, lowered defenses would not be required to produce injury. To evaluate this, it may be prudent to assess urine melatonin metabolites at the time diplomats are identified with symptoms.
Psychogenic illness has been dismissed	Psychogenic causation has been repeatedly suggested as the basis for diplomats’ symptoms (Buckley & Harris, 2018; Myers, 2018; Stone, 2017). This has been correctly dismissed, however, for the Cuba and China diplomats (Harris, 2018c; Stone, 2018; Swanson et al., 2018). Psychogenic causation has similarly been suggested for symptoms from RF/MW (Maisch, 2012) and has been similarly repudiated (Aschermann, 2009; Tressider, 2017). The Swiss Telecom-funded study that documented a relation of sleep problems to transmitter field strength also showed that symptoms were not related to a health-worrying personality (Altpeter et al., 1995; Lamech, 2014). The concordance of symptom profiles across studies, the emergence of RF/MW problems in people unaware of the exposure or its potential for problems, the concordance of symptoms and objective signs with known documented mechanisms of RF/MW injury, the presence of objective markers, and ties to genetics that each cohere with known mechanisms of RF/MW injury (Belpomme et al., 2015; De Luca et al., 2014; Havas et al., 2010) effectively preclude a psychogenic basis for the problem—were such a diagnosis meaningful. (See below, in the entry for study inconsistency, for provocation studies.) The notion that chronic symptoms can arise from psychogenic sources dates to Freud, who also pioneered the flaws associated with its application (Crews, 2017). The foundation is substantially circular, a mechanism has never been physiologically defined or substantiated (much less documented to be operating in cases where the label is applied), and the label is deployed without the most basic scrutiny of the tacit assumptions (Golomb, 2015b). Historically, many conditions that were presumed psychogenic (such as ulcers, seizures) were recognized as organic as evidence emerged (Golomb, 2015b).

Table 4: Continued.

Not all are affected—a subset of embassy personnel (Stone, 2018) and of RF/MW exposed	How might some people experience symptoms and signs of injury from what seem to be “low levels” of an exposure, seemingly well below levels that other people tolerate? For toxins, we designate an LD50 (Baioimy, Attia, Soliman, & Makrum, 2015; Jagetia & Baliga, 2003; Jagetia, Venkatesh, & Baliga, 2004; Pal & Chatterjee, 2006; Shafiee et al., 2010; Shimoda, Akahane, Nomura, & Kato, 1996) (dose lethal in 50%) or an LD5. This reflects the recognition that for each potentially toxic exposure, there is a range in which some will experience an outcome and others will not. One can also define an SD50 (symptoms in 50%)—or an SD25, or SD5. It would be surprising if a highly useful and lucrative technology were not pushed as far into this intensity range as possible. Genetic variations in a range of free radical detoxification systems, competition for those systems, alterations in gene expression based on prior exposures, differences in vulnerability of the tissue affected (via factors like mitochondrial “heteroplasmy,” past injury of that organ), and variations in secondary mechanisms triggered by oxidative stress provide among the mechanisms by which variability is produced. The de facto intensity of the “same” exposure may differ radically (no pun intended) from person to person.^a A further mode of variability arises from immune activation. Considering a more familiar allergen, one person can eat a jar of peanut butter without a problem, while another is hospitalized for exposure to a crumb of peanut. As above, oxidative stress can modify substances in a fashion that makes them vulnerable to autoimmune attack. Immune or autoimmune activation is a documented feature in a subset of those citing symptoms from RF/MW/EMR (Belpomme et al., 2015). “Effect modification” refers to differences in effect in different individuals, and it is the rule rather than the exception in biology. Particular considerations are germane when the exposure has potential for prooxidant or antioxidant effects (Golomb, 2018). Many prooxidants can be antioxidant at low doses in some people (via “oxidative preconditioning” in which low-level exposure to prooxidants may upregulate native antioxidant defenses; this can lead to net antioxidant effects in persons whose defenses are not already overwhelmed or maximally upregulated—as above). Conversely, many substances thought of as antioxidants are prooxidant in some settings, often including high dose (Azam, Hadi, Khan, & Hadi, 2003; Bowry, Mohr, Cleary, & Stocker, 1995; Gerster, 1999; Hiramoto, Ohkawa, Oikawa, & Kikugawa, 2003; Hu, Chen, & Lin, 1995; Kontush, Finckh, Karten, Kohlschutter, & Beisiegel, 1996; Lee, Kim, Park, Chung, & Jang, 2003; Palozza, Luberto, Calviello, Ricci, & Bartoli, 1997; Young & Lowe, 2001). So the same exposure can produce even opposite-direction effects in different persons. Exemplifying the principle, statin cholesterol-lowering drugs are net antioxidant in many people (often tested in nonelderly males without metabolic syndrome factors),
Effect modification	

Table 4: Continued.

	but are reproducibly prooxidant in a subset, and prooxidant dominance is tied to side effects (Sinzinger, Lupattelli, & Chehne, 2000; Sinzinger, Lupattelli, Chehne, Oguogho, & Furborg, 2001). These side effects, attended by net prooxidant effect (Sinzinger et al., 2000; Sinzinger et al., 2001) arise disproportionately with higher doses and in persons with conditions like older age and metabolic syndrome factors, that are statistically tied to mitochondrial impairment (Golomb & Evans, 2008). Side effects, too, occur disproportionately in women (Golomb & Evans, 2008). Women show higher rates of adverse effects from many drugs and environmental toxins (and many medical procedures); they are also more often affected by EMR (Gruber et al., 2018; Levallois et al., 2002; Rössli, Möser, Baldinini, Meier, & Braun-Fahrlander, 2004; Santini et al., 2002; Schooneveld & Kuiper, 2007). There are many potential sources of effect modification from genetics (De Luca et al., 2014), level of exposure, and past and current environment that influence biology. Some exposures may cause mitochondrial injury or oxidative stress or depress concentrations of antioxidants, boosting vulnerability. Others may have protective effects. Many drugs and chemical exposures cause oxidative stress, cause mitochondrial injury (which also increases intracellular oxidative stress), depress antioxidant defenses, and/or compete for or inhibit detoxification systems. Through these and other mechanisms, these exposures may magnify harm from RF/MW and vice versa. Preliminary evidence comparing Swedish ES-affected persons versus controls identifies higher levels of some organic pollutants in those with ES (Hardell et al., 2008), though larger studies are needed. Chemical exposures that cause oxidative stress compete for or inhibit detoxification systems may magnify harm from RF/MW and vice versa. Melatonin and glutathione (and other antioxidants) can be "radioprotective" (Bravard et al., 2002; Jensen & Meister, 1983; Shirazi et al., 2013; Simone, Tamba, & Quintiliani, 1983). (Here the root <i>radio</i> refers to radiation, not specifically to radiofrequency radiation.) Other agents or conditions can be "radiosensitizing." As might be expected, glutathione depletion can be radiosensitizing, though the status of other antioxidants may be important (Hodgkiss, Stratford, & Wafar, 1989; Koch & Skov, 1994; Vallis, 1991; Vos, van der Schans, & Roos-Verheij, 1986). The tie between low melatonin (assessed by the principal metabolite) and ES in the French study (Belpomme et al., 2015) supports the expectation that melatonin depletion is radiosensitizing as well. Radiosensitization is used therapeutically to enhance killing by radiation of tumor cells (Yi, Ding, Jin, Ni, & Wang, 1994), but its existence there is a reminder that chemicals interact with radiation to modify
Chemical exposures may serve as one source of effect modification	

Table 4: Continued.

radiation effects. Radiation itself may be radiosensitizing—as potential effects on antioxidant systems, reviewed elsewhere, suggest—and reportedly ultrahigh-frequency radiation is a particularly effective radiosensitizer (Holt, 1995). Oxidative stress is an important, but not the only, means by which radiosensitization occurs (Park et al., 2005), consistent with multiple downstream mechanisms of injury. Of note, because critical systems that are involved in radiation defense (e.g., melatonin, glutathione, and other antioxidant systems) are also involved in defense against toxicity of chemicals and drugs (Mitchell & Russo, 1987) and because factors that adversely affect antioxidant:oxidant balance may be adverse for oxidative stress-mediated injury from either type of source, it is expected, as it is observed, that there will be overlap between chemical and electrical sensitivity (Belpomme et al., 2015; Golomb, 2015a). Two illustrations where we can see the radiosensitizing effect occur with ultraviolet (uv) light, since due to its high frequency, the effect is primarily on the skin. Photosensitizing agents and radiation recall are the illustrations. Photosensitizing or phototoxic or photoallergic agents are agents that magnify damage observed with uv radiation. (For simplicity we use <i>photosensitizing</i> to encompass each of these.) In some cases, radiation breaks down a chemical to something toxic. Drugs may also photosensitize, for instance, by augmenting one of the mechanisms of radiation injury, such as oxidative stress or mitochondrial dysfunction (Shea, Wimberly, & Hasan, 1986). Fluoroquinolone antibiotics, which can cause serious problems in a vulnerable subset through oxidative stress and mitochondrial dysfunction (Golomb et al., 2015), are strongly reported to photosensitize and to be phototoxic (Agrawal, Ray, Farooq, Pant, & Hans, 2007; Akter et al., 1998; Bilski, Martinez, Koker, & Chignell, 1996; Boccumini, Fowler, Campbell, Puertolas, & Kaidbey, 2000; Burdge, Nakielna, & Rabin, 1995; Chetelat, Albertini, & Gocke, 1996; Ferguson & Johnson, 1990, 1993; Fujita & Matsuo, 1994; Granowitz, 1989; Kimura, Kawada, Kobayashi, Hiruma, & Ishibashi, 1996; Man, Murphy, & Ferguson, 1999; Nedorost, Dijkstra, & Handel, 1989; Oliveira, Goncalo, & Figueiredo, 2000; Scheife, Cramer, & Decker, 1993; Snyder & Cooper, 1999; Trisciuglio et al., 2002; Wagai & Tawara, 1991; Wagai, Yamaguchi, Sekiguchi, & Tawara, 1990). Fluoroquinolones have been tied to development of persistent phototoxicity (following withdrawal of the drug; Sailer et al., 2011)—that is, ongoing higher vulnerability to this radiation—consistent with evidence that a vulnerable group experiences persistent damage from fluoroquinolones in which oxidative stress and mitochondrial injury play a role (Golomb et al., 2015). This “vulnerability” may be acquired, as mitochondrial injury can be cumulative, and a serious reaction sometimes follows a previous course of

Table 4: Continued.

fluoroquinolones with a milder and time-limited reaction or none at all (Golomb et al., 2015). (Mitochondrial injury from radiation can also be cumulative; Prithivirajsingh et al., 2004.) Fluoroquinolones have led to reported "photosensitivity" reactions to fluorescent lighting (Jaffe & Bush, 1999). Statins, which as elsewhere are sometimes prooxidant (Sinzinger et al., 2001) and sometimes mitochondrially toxic (Golomb & Evans, 2008), are also sometimes linked to photosensitivity (Morimoto, Kawada, Hiruma, Ishibashi, & Banba, 1995; Thual, Penven, Chevallier, Dompmartin, & Leroy, 2005). (The information that follows about photosensitivity in Smith-Lemli-Opitz syndrome explains one reason that statins can be prooxidant, though they also have antioxidant mechanisms.) Given oxidative mechanisms of radiation injury that apply across the electromagnetic spectrum, it is expected that some agents that photosensitize may sensitize to other forms of radiation, potentially including RF/MW. Others have noted that photosensitizing drugs have played an apparent role in other radiation injury (Dawson, Brown, & Tellefsen, 2009). (Data we have presented, but not published, showed that past use of fluoroquinolones was significantly tied to the development of ES. Past adverse effects to fluoroquinolones, which signify oxidative-mitochondrial injury to a point producing symptoms (at least, they surpassed the symptom threshold for a time), showed a particularly strong connection (Golomb, 2015a).) There are also disease conditions tied to the magnified photosensitivity (Murphy, 2001). Where these are tied to depressed antioxidant defenses, or increased mitochondrial injury, they might be predicted to be tied to increased risk of ES development (accounting for radiation exposure). In Smith-Lemli-Opitz syndrome, which many studies have tied to photosensitivity, cholesterol levels are low (Anstey, 1999, 2001, 2006; Anstey, & Azurdia, Rhodes, Pearse, & Bowden, 2005; Anstey et al., 1999; Anstey & Taylor, 1999; Azurdia, Anstey, & Rhodes, 2001; Charman et al., 1998; Chignell, Kukielczak, Sik, Bilski, & He, 2006; Eapen, 2007; Martin, Taylor, Trehan, Baron, & Anstey, 2006; "[A new congenital photosensitivity syndrome. Smith-Lemli-Opitz syndrome]," 1999). Cholesterol transports critical fat-soluble antioxidants (Golomb & Evans, 2008). In the phenomenon of "radiation recall," injury to tissue initially caused by radiation can be made to reappear by another agent with shared mechanisms of injury (e.g., oxidative stress and mitochondrial injury), such as fluoroquinolone antibiotics, best recognized for skin reactions, since we are able to see these (Cho, Breedlove, & Gunning, 2008; Jain, Agarwal, Laskar, Gupta, & Shrivastava, 2008; Wernicke, Swistel, Parashar, & Myskowski, 2010).

Table 4: Continued.

Hypothesis: One possible vulnerable group	Evidence supports a relationship between genetics of intellectual promise, and a different condition in which oxidative stress and mitochondrial impairment play a critical role: autism spectrum disorder (ASD; Frye, Delatorre et al., 2013; Frye, Melnyk, & Macfabe, 2013; Frye & Rossignol, 2011; Rose et al., 2012; Rossignol & Frye, 2012). (EMR exposure has been considered as a possible factor (Herbert & Sage, 2013a, 2013b.) It was found that gene profiles that increase risk of ASD (polygenic risk) are tied to higher intelligence in the general population (Clarke et al., 2015): “We report that polygenic risk for ASD is positively correlated with general cognitive ability ($\beta = 0.07$, $P = 6 \times 10^{-7}$. . .), logical memory and verbal intelligence,” findings that were replicated in a different sample by positive relation to full-scale IQ (Clarke et al., 2015). This supports a line of reasoning by which impaired cell energy, through oxidative stress and mitochondrial dysfunction, may disproportionately affect the “best and the brightest,” on whom society differentially depends—with implications for vulnerability to RF/MW. Many mechanisms tied to high function are tied to high energy demand. Higher energy demand may create greater vulnerability in the setting of impaired energy supply. (It is the chasm between demand and that guides degree of injury.) Many drugs and chemical exposures cause oxidative stress, cause mitochondrial injury (which also increases intracellular oxidative stress), depress antioxidant defenses, and/or compete for or inhibit detoxification systems. Through these and other mechanisms, these exposures may magnify harm from RF/MW and vice versa.
Are provocation studies contributory?	Several so-called provocation studies have been conducted in persons with ES; some focus on symptoms, some on objective markers. In most of those that focus on symptoms, those with ES fail to reliably distinguish between blinded EMR “exposed” and “unexposed” settings (Rubin, Das Munshi, & Wessely, 2005). Major flaws in the designs have been recognized and reviewed by others (Leszczynski, 2015; Schooneveld & Kuiper, 2007); for instance, studies assume that the details of exposure and time course do not need to be individualized, which is contrary to the evidence. But there are further problems. The most fundamental is the assumption that in ES, symptoms serve as a meter. This is invalid. Consider the analogy of sunburn: a form of radiation injury mediated by oxidative stress that affects some but not others at usual exposure levels. Those who are affected “believe” sun exposure is responsible. They would be unlikely to discern when they are being exposed versus not to ultraviolet radiation. (It is their failure to know when significant injury is occurring or has occurred that leaves them in the sun long enough to receive injury.) What is discerned is the inflammation that follows the oxidative stress that may emerge only late in exposure or after the sun exposure has been “withdrawn.” A blinded sham-exposed study would likely also produce inability to discern sham from active treatment.

Table 4: Continued.

People do not sense the EMR, but the effects produced by it and studies show that those with ES respond to different EMR sources. In RF/MW-affected persons, as in diplomats, the effects can arise after hours of exposure or hours after a short exposure—oxidative stress can cause apoptosis and can then trigger inflammation (Reutelingsperger & van Heerde, 1997) or can cause blood-brain barrier damage allowing brain swelling (see above). Progression of these mechanisms may not peak for hours or, in some cases, even a couple of days. Recovery from effects can take still longer.

For such a study to have a chance to succeed, it would be essential to pretest and individualize both the control/negative exposure condition and the active/positive exposure condition (including exposure and time course) in each individual to define a condition that will be effective in that person—if such conditions can be successfully defined and if cumulative effects do not alter the condition from one trial to the next. For some people, the background EMR at the facility, or its parking lot or lobby, or the exposure during transit to the facility may obviate the ability to define a negative exposure condition for that individual. It would be better to bring the EMR exposure to a place where the affected party is stable and asymptomatic. And the specific EMR and timing must be individualized to produce a positive condition in a suitable time course. To be valid, such a study must also protect against the possibility of physiological conditioning effects. These are distinct from nocebo effects and arise because the true stimulus produces actual physiological harm. It is known, for instance, that chemotherapy patients may vomit when they enter the room in which they have received chemotherapy. (Chemotherapy agents like EMR also cause toxicity via oxidative stress (Abraham, Kolli, & Rabi, 2010; Brea-Calvo, Rodriguez-Hernandez, Fernandez-Ayala, Navas, & Sanchez-Alcazar, 2006; Husain, Whitworth, Somani, & Rybak, 2001; Shokrzadeh et al., 2014) and mitochondrial injury (Nicolson & Conklin, 2008). The fact that symptoms also occur with expectation of chemotherapy does not mean that the chemotherapy itself lacks toxicity (or that perceived adverse effects are due to a nocebo effect); rather, expectation produces symptoms because the exposure *is* toxic. Expectation of the noxious exposure may, via conditioning processes, produce symptoms ordinarily produced by the noxious exposure. (This is potentially evolutionarily adaptive, serving to encourage persons to avoid settings in which the toxic exposure is expected.) To ensure against conditioned effects arising with expectation, a set of negative exposure visits at the test site before (and between) each positive exposure visit may be required to ensure extinction of physiologically conditioned expectation effects. In essence, the setting that optimizes prospects to identify a real effect, if present, is that in which the participant believes there will not be an active exposure.

Table 4: Continued.

<i>N</i>-of-1 studies that focus on physiological effects of EMR have proven somewhat more able to identify EMR effects in those with ES, or subsets of them for which that physiological marker is affected. Just as symptoms vary, so physiological changes may do so, so outcomes suited to one person may not apply for all. Physiological markers changed with blinded EMR exposure in a published study of a female physician with ES. She could not discern when the exposure was present or not, but measurable changes occurred and symptoms arose with the positive condition (McCarty et al., 2011). Symptoms were significantly more intense with pulsed (but not continuous) radiation than sham exposure (McCarty et al., 2011). An <i>N</i>-of-1 test was reportedly conducted in a former Miami organized crime prosecutor who developed ES and chemical intolerance, with seizures an important part of his clinical profile, following a significant chemical exposure. An EEG was undertaken, turning on and off a TV, with the party blinded to the stimulus (blinded and with headphones to prevent him hearing when the TV was turned on or off). When the TV was shielded, no effect on the EEG was seen. With an unshielded television, EEG changes including seizure activity occurred when the television was turned on, and he experienced physical twitching (Bell, 2017). (This particular marker is unlikely to be generally useful, as seizure activity is not a usual part of the clinical profile in those affected by RF/MW.) A provocation study focused in a group of individuals showed changes in heart rate variability (Havas et al., 2010), an index of autonomic function that is tied to hard outcomes like sudden death and coronary artery disease (Hayano, 1990; Singer, Martin, Magid, & et al, 1988). Moreover, three of the four participants who characterized their ES as “intense” (though only persons in this group) exhibited a striking heart rate increase of between 45 and 90 beats per minute virtually immediately with the microwave exposure, associated with marked increase in sympathetic response. Declines in parasympathetic response with RF/MW exposure were seen for 23 of 25 tested people, in all groups, including, though less so, those with no ES. In general, assessments of objectively measurable quantities of relevance, including both differences in affected vs unaffected persons irrespective of current exposure (Belpomme et al., 2015; De Luca et al., 2014), and changes occurring with exposure (Havas et al., 2010), provide a more promising approach than real-time assessments of subjective outcomes for understanding this condition.
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Table 4: Continued.

Financial conflict of interest is a major source of apparent disparities in results	One key source of disparities in study results is financial conflicts of interest. When present, financial conflicts strongly predict that study results will conform to the financial interests of authors or funders (Barnes & Bero, 1998; Bero, Oostvogel, Bacchetti, & Lee, 2007; Friedman & Richter, 2004; Golomb, 2008; Heres et al., 2006; Smith, 2005, 2006). An analysis examined why some review articles on passive smoking concluded it was harmful while others concluded it was not. The only identified factor that predicted which conclusion was industry conflict by authors—which was often undisclosed (Barnes & Bero, 1998). Financial conflicts have been a concern specifically in relation to RF/MW, for both studies and regulatory decisions (Adlkofer & Richter, 2011; Alster, 2015; Hardell, 2017; Huss et al., 2007; Leszczynski, 2015). In an analysis of studies looking at cell phone effects as a function of funding source, “Studies funded exclusively by industry reported the largest number of outcomes, but were least likely to report a statistically significant result” (So, they report everything that wasn’t affected?) “The odds ratio was 0.11 (95% confidence interval, 0.02–0.78), compared with studies funded by public agencies or charities.” Analogous to findings for a relation of industry funding to failure to find tobacco-related problems (Barnes & Bero, 1998), “the finding was not materially altered in analyses adjusted for the number of outcomes reported, study quality, and other factors” (Huss et al., 2007). It has been generally assumed that the disproportionately product-favorable results from industry-funded studies (including less evidence of product harm) arise by virtue of choices, selecting study design, exposure specifics, subjects, and outcomes to support the desired result. (These can in fact influence outcomes. See below.) But where harms of lucrative products are concerned, there is precedent for industry-funded studies going beyond those factors to hide even large and lethal harms, even for prespecified or primary outcomes—via means that have the appearance, at least, of fraud (“Did GSK trial data mask Paxil suicide risk?” 2008; Harris, 2010). Special circumstances led the apparent shenanigans in those cases to be uncovered. Whether frank manipulation of data to hide harms of lucrative products is the rule or the exception in industry-funded studies is simply not known. Because a robust body of evidence documents a strong relation of industry conflicts to outcomes, deliberations and standards should be based exclusively on studies in which such conflicts of interest are absent. (Industry-funded studies can be used for hypothesis generation.) This obviates one major source of apparent inconsistency in studies, but it eliminates inconsistencies due to this factor only as far as it is possible to discern when financial conflicts are operating.
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Table 4: Continued.

Study outcomes may appear different without "inconsistency"; Details matter, to see an effect	Design features can influence outcomes and may be selected to do so. Details of RF/MW exposure that may influence outcomes include the following (some relevant features have doubtless been missed): • Radiation frequency or frequencies (Belyaev, Sheheglow, Alipov, & Ushakov, 2000; Chen, Yang, Tao, & Yang, 2006; Gupta, Mesharam, & Krishnamurthy, 2018), • Radiation intensity (Adams & Williams, 1976) • Radiation waveform (Adams & Williams, 1976) • Polarization (Belyaev et al., 2000; Pall, 2018; Panagopoulos, Johansson, & Carlo, 2015), • Pulsed versus continuous radiation (Lai, Horita, Chou, & Guy, 1987; Pall, 2018) • Pulse width (Bonnafous et al., 1999) • Time between pulses (Belyaev et al., 2006)/repetition rate (1988) • Pulse waveform (Bolen, 1988; Wood, Armstrong, Sait, Devine, & Martin, 1998), • Pulse intensity (Elder & Chou, 2003), • Exposure duration (Lai & Singh, 1995; Robison, Pendleton, Monson, Murray, & O'Neill, 2002) • Exposure intermittency (Ivancsits, Diem, Pilger, Rudiger, & Jahn, 2002) on every timescale • Environmental conditions: temperature, humidity, air currents (Adams & Williams, 1976; Laszlo et al., 2006) • Concurrent (or preceding) exposures to other radiation (Adams & Williams, 1976; Bua et al., 2018; Kostoff & Lau, 2017), which can cause synergistic effects (Adams & Williams, 1976) • Concurrent (or preceding) chemical exposures or environment (Bua et al., 2018; Kostoff & Lau, 2017) • State of health of the animal or subject (Adams & Williams, 1976) • Species (Adams & Williams, 1976) • Size of the subject relative to wavelength (Adams & Williams, 1976) • Genetics of the animal (Belyaev et al., 2000; De Luca et al., 2014) • Antioxidant/nutrient status of the animal or subject (Ceyhan et al., 2012; Gajski & Garaj-Vrhovac, 2009; Guney et al., 2007; Gurler, Bilgici, Akar, Tomak, & Bedir, 2014; Koyu et al., 2009; Li et al., 2014; Oksay et al., 2012; Oktem et al., 2005; Oral et al., 2006; Sokolovic et al., 2013; Zhang et al., 2011; Zhang et al., 2014) • Orientation of the animal or subject relative to the radiation source (Adams & Williams, 1976) • Portion of the body irradiated (Adams & Williams, 1976) • Time between exposure and assessment of effect (Belyaev et al., 2000) • Effect measured • Metric used to measure effect
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Table 4: Continued.

Radiation that is pulsed (i.e., polarized), is applied intermittently, is more intense, and is applied for a longer time may be more likely to produce problems, for instance. Even for studies nominally examining the "same" RF/MW exposure, different choices may be made. A range of choices are illustrated in this text: "There are 124 different channels/frequencies that are used in GSM900 mobile communication. They differ by 0.2 MHz in the frequency range between 890 and 915 MHz. The test mobile phone was programmed to use channel 124 with the frequency of 915 MHz. The signal included all standard GSM modulations. No voice modulation was applied. A GSM signal is produced as 577 μs pulses (time slots), with an interpulse waiting time of 4039 μs (seven time slots). The test phone was programmed to regulate output power in the pulses in the range of 0.02–2 W (13–33 dBm). This power was kept constant during exposure at 33 dBm, as monitored online using a power meter (Bird 43, USA)" (Belyaev et al., 2006). Studies that examine symptoms as a function of distance from cell towers and base stations suggest that in important real-world settings, more intense RF/MW exposure is generally a greater problem (Altpeter et al., 1995; Navarro, Sanchez Del Pino, Gomez, Peralta, & Boveris, 2002; Oberfeld et al., 2004; Santini et al., 2002), though there may be an intensity range below which this ceases to be the case. In some conditions, nonmonotonic effects of radiation have been reported (Chiang et al., 1989; Pall, 2018), and they are arguably expected for agents in the antioxidant-prooxidant spectrum (high-dose antioxidants are often prooxidant; low-dose prooxidants, via oxidative preconditioning, may be antioxidant). Opposite-direction effects on a critical mechanism can produce opposite-direction effects in a resulting outcome. Thus, lower doses of vitamin E fluidize, and higher concentrations stabilize membranes (Packer & Fuchs, 1993); low vitamin E benefits and higher vitamin E harm vasodilatory function in cholesterol-fed rabbits (Keaney et al., 1994); "low tocopherol concentrations have stronger antiinflammatory effects in PUVA-induced erythema than higher concentrations" (Fuchs & Packer, 1993); low doses are tied to lower mortality in people, higher doses to higher all-cause mortality (Miller et al., 2005). For statins, an agent class that can produce prooxidant or antioxidant effects, bidirectional effects have been shown on many outcomes (Golomb et al., 2015). Such bidirectional effects have been shown for many outcomes with RF/MW (Bergman, 1965). It is common that where a lower amount of something may be favorable (or neutral), a higher amount may be the adverse, with a transition zone in which subject characteristics and covariables matter a lot in determining the direction. There are instances in which this directionality is flipped (Au, Cantelli-Forti, Hrelia, & Legator, 1990); for instance, sometimes a sufficient concentration leads an adaptive protection to be triggered.
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Table 4: Continued.

Beyond characteristics of the radiation, the subject may be exposed to it differently; for example, in animal studies, there may be whole-body radiation (Bilgici, Akar, Avci, & Tuncel, 2013) or head-only exposure (Burdelya et al., 2012; de Gannes et al., 2009), triggering a different spectrum of responses. And with in vitro exposure, even fewer of the variables that might contribute to effects are present. The environment in which exposure occurs may differ in ways that influence toxicity of radiation—for instance, differences in temperature may produce different effects (Laszlo et al., 2006), or concurrent or background electromagnetic exposure (Bua et al., 2018) or chemical exposures (Del Vecchio et al., 2009; Kostoff & Lau, 2017). Amphetamine use represents one exposure that has been reported to magnify problems with RF/MW (Bohen, 1988). Characteristics of the “subjects” may differ. In animal and in vitro studies, they may differ in species, strain, genetic features, cell type cell preparation, and cell density, for instance (Belyaev, Sheheglov, Alipor, & Ushakov, 2000; Del Vecchio et al., 2009). As above, “effect modification” refers to the phenomenon by which effects, including adverse effects, are not equal in all subgroups. This is a major issue in biology, particularly for exposures mediated by oxidative stress and cell energy impairment. Findings with statin cholesterol-lowering drugs illustrate how massive the disparity may be as a function of participant group. Like RF/MW, these agents have the potential for toxicity through prooxidant and mitochondrial adverse mechanisms (Golomb & Evans, 2008; Sinzinger et al., 2001). RF/MW disproportionately affects sleep and hearing (through its special extra features), but muscle and tendon problems are sometimes reported (Aschermann, 2009; Lamech, 2014; Schooneveld & Kuiper, 2007). Fluoroquinolones disproportionately affect tendons through their extra mechanisms. Statins can do so too, though more rarely (Esenkaya & Unay, 2011; Hoffman, Kraus, Dimbil, & Golomb, 2012; Marie & Noblet, 2009; “Tendon disorders due to statins,” 2010). Statins disproportionately affect muscle. The most feared muscle complication is rhabdomyolysis, massive breakdown of muscle that can overwhelm the kidneys and lead to kidney failure and death, which is also reported with fluoroquinolones though more rarely (Eisele, Garbe, Zeitz, Schneider, & Somasundaram, 2009; George, Das, Pawar, & Badyal, 2008; Gupta, Guron, Harris, & Bell, 2012; Hsiao et al., 2005; Khammassi, Abdelhedi, Mohsen, Ben Sassi, & Cherif, 2012; Korzets, Gafter, Dicker, Herman, & Ori, 2006; Petitjeans et al., 2003; Qian, Nasr, Akogyeram, & Sethi, 2012; Sanjith, Raodeo, Clerk, Pandit, & Kamad, 2012).
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Table 4: Continued.

	Statins were commonly hailed as so safe they should be put in the water supply (Brown, 2001; Dales, 2000; Haney, 1999; Roberts, 2004). But analysis of insurance claims data show that (focusing on the one adverse effect) while the rate of rhabdomyolysis was rare overall, it was common in identifiable vulnerable subgroups. Hospitalized rhabdomyolysis, per year of treatment, occurred in fewer than 1 in 22,000 on statin monotherapy. However, the rate was far higher for older persons with diabetes also on a fibrate (a second class of cholesterol-lowering drug); if they were on the statin agent whose clearance was most affected by fibrates, rhabdomyolysis occurred in about 1 in 10 per year of treatment (Graham et al., 2004). So depending on characteristics of the exposure, co-exposures, and the subject, rates of a problem—and ability for science to show the problem—can vary widely. (The particular statin agent that caused the worst problems was pulled from the market, but the conceptual point stands.) Risks of harm with exposures are not distributed equally. A problem that appears very rare overall or in one test group, often apparently not increased relative to unexposed, can be frankly common in another. If the groups most at risk are not studied or their presence is seriously diluted, serious harms can be missed. Studies that fail to detect a harm do not invalidate those that show one—and are not of equal importance where a purpose is to establish that harms can occur. Though a minority of embassy personnel were reportedly affected (Stone, 2018), the fraction is not small (Golden & Rotella, 2018). The fraction of U.S. diplomats in Cuba (and now China) reporting effects is higher than the fraction of civilians citing similar severity problems with RF/MW exposure, though in neither group can the exposure of those affected be presumed to have been typical. Table 3 suggests that once persons are symptomatic, the profile of symptoms is similar. The reportedly high prevalence of Frey-compatible effects and what seem a comparatively large number of diplomats in Cuba affected suggest exposures of a more intense or more damaging character considering that intensity, frequency, pulse waveform, pulse duration, duration, polarization, intercurrent exposures, and many other factors influence injury from RF/MW (Belyaev et al., 2000).
Rates of problems	Both diplomats (Associated Press in Washington, 2017) and RF/MW-affected individuals (Conrad & Friedman, 2013; Schooneveld & Kuiper, 2007) have shown variable time course to onset of symptoms after apparent inciting exposure and variable time course and completeness of recovery with time away from the exposure. Doctors submitting the Bamberg Appeal to the Prime Minister of Germany noted, “The symptoms occur in temporal and spatial relationship to exposure. . . . Some of the health disturbance disappears immediately the exposure ceases (removal of DECT telephone, temporary moving away from home, permanently moving away, using shielding)” (Waldman-Selsam, 2004). An intervention study from Japan, involving the “intervention” of removing a cellular phone base station on a condominium, affirms
Natural history	

Table 4: Continued.

improvement with removal of the exposure. One hundred seven of 122 inhabitants were interviewed and had medical examinations at two time points while the base station was in operation and three months after it was removed. "The health of these inhabitants was shown to improve after the removal of the antennas, and the researchers could identify no other factors that could explain this health improvement. . . . The results of these examinations and interviews indicate a connection between adverse health effects and electromagnetic radiation from mobile phone base stations" (Shinjiyo & Shinjiyo, 2014). Studies in Russia of occupationally affected persons report that even with treatments that target mechanism of RF/MW injury, for those at least moderately affected, placing them back in the setting of exposure leads to a progressive course (Sadchikova & Glotova, 1973). Natural history could differ for diplomats who may have been exposed to a more intense stimulus or one with more injurious characteristics—suggested by what appear to be a comparatively high number affected and a high prevalence of Frey effects. With a powerful exposure, depressed defenses are not equally required to produce injury. There is not a basis to know if affected diplomats will have heightened vulnerability to "usual" RF/MW exposures going forward, though this bears assessing.

^aAn illustration from a common drug, and a common food: "Grapefruit juice increased the mean peak serum concentration (Cmax) of unchanged simvastatin about 9-fold (range, 5.1-fold to 31.4-fold; $P < .01$) and the mean area under the serum simvastatin concentration-time curve [AUC (0-infinity)] 16-fold (range, 9.0-fold to 37.7-fold; $P < .05$)" (Lilja, Kivisto, & Neuvonen, 1998). Thus, just one comparatively innocuous interacting factor, grapefruit juice (which inhibits an enzyme involved in simvastatin metabolism), led some to have a 38-fold greater blood "amount" of a drug, than that same person would have had without the juice. Potential differences are magnified comparing different persons with or without juice, and more so factoring in impact of other exposures. Other risk-multiplying factors are tied to the individual. The same serum level can produce a radically different impact from person to person; relevant factors include genetic differences in muscle and factors that reduce energy supply or increase energy demand to muscle (Golomb & Evans, 2008; Golomb & Koperski, 2013; Oh, Ban, Miskie, Pollex, & Hegele, 2007; Sinzinger & O'Grady, 2004; Vladutiu et al., 2006). Thus, what is the "same" exposure before it hits two people can become a radically different exposure once it interacts with individuals' biology.

exposure-related illnesses, genetic influences on phase I or phase 2 detoxification, as well as factors that inhibit or compete for detoxification systems, play a documented role in who develops health effects (Cherry et al., 2002; Ishikawa et al., 2004; Molden, Skovlund, & Braathen, 2008; Page & Yee, 2014; Rowan et al., 2009; Steele, Lockridge, Gerkovich, Cook, & Sastre, 2015). (Phase II detoxification encompasses protections against oxidative damage.)

Table 5 briefly addresses the range of RF/MW sources that have been presumptively tied to problems. It observes that RF/MW/microwave radiation is known to have been used on the U.S. embassy in Moscow; there is precedent for use on diplomats (Gwertzman, 1976; Schumaker, 2013). That instance, though with presumably differing details of exposure, led to (disputed) reports of health effects in embassy staff and shielding efforts by the United States. Since the exposing device can be outside the building—and typically has been, for persons affected by RF/MW-emitting utility meters (Lamech, 2014)—failure of the FBI to find devices in sweeps of diplomats' rooms remains compatible with this explanation.

4 Discussion

4.1 Recap of Findings. Health effects reported by U.S. and Canadian diplomats (and family members) in Cuba and China, and the circumstances surrounding inciting episodes, are consistent with effects of RF/MW. Reports of perceived sounds fit known characteristics reported for the Frey effect (microwave auditory effect). Sounds were heard by some but not other diplomats during inciting episodes; sounds differed in character from person to person; sounds included chirping, ringing, and grinding; and sounds were heard predominantly at night. Sounds were localized with laserlike specificity in some of the cases and, within that localization, seemed to follow people. Prominence of auditory symptoms, including hearing loss, tinnitus, and ear pain in diplomat reports, typify reports of injury from pulsed RF/MW. Presence of variable additional symptoms of protean character that differ markedly from person to person, with a relative emphasis on sleep disturbance, headaches, and cognitive problems, plus presence in smaller subsets of vision, balance, and speech problems, are also characteristic. Affected persons in both groups report sensory symptoms of pressure and vibrations. Persons in both groups show evidence of brain injury. Reports in both indicate that some persons had prior head injury, and brain injury may be a predisposing factor for as well as a consequence of RF/MW injury (Heuser & Heuser, 2017; Stone, 2018). Both show varying rates of symptom persistence. How subsequent natural history will compare, for diplomat symptoms that *might* follow more intense discrete exposure (a more intense exposure may produce problems in persons who need not have relative vulnerability), versus follow repeated less intense ones (producing symptoms, evidence suggests, selectively in

Table 5: RF/MW Source Considerations.

What kinds of RF/MW sources affect civilians?	In the UCSD survey, smart meters were the dominant inciting trigger (about 50% of those 70% or so who recognized a triggering episode), with cell phones, Wi-Fi introduction or new routers, medical radiation, and other factors also reported (Golomb, 2015a). The range of apparent triggers has been vast, with RF/MW, and particularly pulsed RF/MW, commonly implicated. Considering those who have communicated with us, a couple from Scotland became affected several decades ago, after they moved to a rural area but across from a radar factory. Though they moved away, both remain "electrosensitive" decades later. Others became affected when a cell tower was placed next to their home. Gro Harlem Brundtland reports becoming sensitized following exposure to a malfunctioning microwave oven in an episode that also reportedly blinded her for a year (Woolston, 2010; www.es-uk, 2012). An Australian veteran reports that he became affected during his military service, working with radiofrequency radiation (radar workers in the military were among the first groups in whom such problems were recognized many decades ago). One who communicated with us became sensitized in association with a job placing radio collars on wildlife. An architect who contacted us was sensitized after several months working closely with Bluetooth-enabled lighting devices. Parents reported to us the onset of ES in their children with Wi-Fi introduced to the school; accommodations were denied, forcing parents to remove their children from school and move elsewhere and forcing some teachers from their job ("Math teacher asks school to protect children from Wi-Fi," 2015; "Math teacher raises concerns about WIFI comparing the effects to a concussion," 2014). In Sweden and the United Kingdom, a controversial radio system, TETRA, reportedly caused health problems in some police officers, severe insomnia in a Swedish officer resolved when the officer's managers noted the connection and placed the officer in a room without the exposure (www.es-uk, 2012). Some U.S. firefighters were affected after municipalities placed cell towers on roofs of fire stations (International Association of Fire Fighters Division of Occupational Health Safety and Medicine, 2006): "Symptoms experienced by the firefighters have included neurological impairment including severe headache, confusion, inability to focus, lethargy, inability to sleep, and inability to wake up for 911 emergency calls. Firefighters have reported getting lost on 911 calls in the same community they grew up in, and one veteran medic forgot where he was in the midst of basic CPR on a cardiac victim and couldn't recall how to start the procedure over again. Prior to the installation of the tower on his station, this medic had reportedly not made a single mistake in 20 years" (Foster, 2017). The International Association of Fire Fighters Division of Occupational Health, Safety and Medicine crafted a position paper (International Association of Fire Fighters Division of Occupational Health Safety and Medicine, 2006), and firefighters were exempted in the recent proposed California bill, SB-649 (Foster, 2017; "State of California Senate Bill 649 (SB-649). Wireless Telecommunications Bill," 2017), that sought to bypass local control in placing of 5G cell towers (Foster, 2017).
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Table 5: Continued.

Past RF/MW use and diplomats	These were not placebo effects; many developed symptoms prior to identifying the source of the problem or, in some cases, even being aware that the exposure existed at that time. Many had no idea the exposure had the potential to produce problems. They were blindsided by the onset of new problems. The causes were identified by their spatial and temporal relationship to onset, worsening, and abatement. Reports of problems from commercial sources of RF/MW have emerged from many nations including Russia (Sadchikova & Glotova, 1973), Korea (Cho et al., 2016), Japan (Kato & Johansson, 2012), Taiwan (Tseng, Lin, & Cheng, 2011), Turkey (Durusoy et al., 2017), Israel (Tachover, 2013), Australia (Lamech, 2014), New Zealand (www.esnztrust), France (Belpomme et al., 2015), England (Bergqvist et al., 1997; Eliti et al., 2007), Ireland (Bergqvist et al., 1997; IDEA, www.iervn.com), Spain (Bigorra, 2016; Navarro et al., 2003; Oberfeld et al., 2004), Italy (Bergqvist et al., 1997; De Luca et al., 2014), the Netherlands (Schooneveld & Kuiper, 2007), Switzerland (Alpeter et al., 1995; Schreier et al., 2006), Austria (Bergqvist et al., 1997; Hutter, Moshhammer, Wallner, & Kundi, 2006; Leitegeb, 1998; Schröttner & Leitegeb, 2008), Germany (Bergqvist et al., 1997; Hensinger & Wilke, 2016), Denmark (Bergqvist et al., 1997; EHS Foreningen, 2018), Sweden (Gruber et al., 2018; Johansson, 2015) where Ericsson designer Per Segerbäck was seriously affected (Nordström, 2004), Norway (www.felo.no) afflicting three-time Prime Minister Gro Harlem Brundtland; Finland (Hagstrom et al., 2013) reportedly affecting former Nokia chief technology officer Matti Niemela (Nikka, 2014), the United States (Carpenter, 2014; Heuser & Heuser, 2017; Levallois et al., 2002; Woolston, 2010), where affected former Silicon Valley techies Peter Sullivan (Harkinson, 2017) and Jeremy Johnson (Johnson, n.d.) strive to bring attention to the problem; and Canada, where Frank Clegg, formerly President of Microsoft Canada, Inc, now CEO of Canadians for Safe Technology—spearheads the effort toward recognition (Clegg, 2013). Exposure of diplomats to RF/MW is not a new phenomenon. The U.S. embassy in Moscow was reportedly radiated with microwaves from 1953 to 1988 (other sources give earlier or later end dates), spawning U.S. efforts to shield the embassy (Gwertzman, 1976; Schumaker, 2013). The Soviets claimed the purpose was to jam U.S. listening devices (Gwertzman, 1976). Based on reports of past embassy staff, a number of personnel and their offspring developed health effects, some developed white blood cell count elevations, and a couple developed hematological malignancies (Schumaker, 2013). Elevated white blood cell counts (Aschermann, 2009), as well as depressed ones (Adams & Williams, 1976), have elsewhere been reported in association with RF/MW, as have hematological malignancies (Dolk et al., 1997; Hocking & Gordon, 2003), including a recent report of an occupational relationship of RF/MW to “hemolymphatic” malignancies in the military setting: “The PF [percentage frequency] of HL [hemolymphatic]
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Table 5: Continued.

Current RF/MW source possibilities in diplomats	cancers in the case series was very high, at 40% with only 23% expected for the series age and gender profile, confidence interval CI95%: 26–56%, $p < 0.01$, 19 out of 47 patients had HL cancers. We also found high PF for multiple primaries. As for the three other cohort studies, in the Polish military sector, the PF of HL cancers was 36% in the exposed population as compared to 12% in the unexposed population, $p < 0.001$. In a small group of employees exposed to RF/MW in Israeli defense industry, the PF of HL cancers was 60% versus 17% expected for the group age and gender profile, $p < 0.05$. In Belgian radar battalions the HL PF was 8.3% versus 1.4% in the control battalions as shown in a causes of deaths study and HL cancer mortality rate ratio was 7.2 and statistically significant. Similar findings were reported on radio amateurs and Korean war technicians. Elevated risk ratios were previously reported in most of the above studies” (Peleg, Nativ, & Richter, 2018). There was also a news report of a “blood disorder” in a Cuban diplomat, but its character was unspecified (Robles & Semple, 2017a). A controversial Johns Hopkins study was commissioned to assess the health of Moscow embassy personnel but was never published in peer-reviewed literature. Staff from other Eastern European embassies were used as controls (Elwood, 2012), a problematic control group as these are the embassies most likely to have been subjected to similar exposures. Indeed a Freedom of Information Act request reportedly yielded claims of exposure from employees at other embassies (Elwood, 2012). A reanalysis asserted that Russian and Eastern European diplomats, if combined, exhibited a significant increase, relative to expectation from the general US population, in three cancer types (Elwood, 2012; Goldsmith, 1995) that have each been associated with RF/MW exposure in other studies: hematological malignancy (Peleg et al., 2018), brain cancer (Hardell & Carlberg, 2013, 2015; Hardell, Carlberg, & Hansson Mild, 2011; Hardell, Carlberg, Soderqvist, & Mild, 2013), and breast cancer (Balekrouzou et al., 2017; West et al., 2013). Some complaints, such as vision problems, concentration problems, memory loss, depression, and “other symptoms” were greater in the Moscow than the comparator group, in either men or women or, for vision and concentration problems, in each men and women. A reanalysis concluded that the Lilientfeld evidence in the context of other literature “support the RF sickness syndrome as a medical entity” (Johnson Liakouris, 1998). The source of proposed EMR/RF/MW (probably pulsed) affecting diplomats is not a principal focus of this article. For the diplomats in Cuba, causative RF/MW could in principle emanate from monitoring and surveillance devices, as has been speculated for microwaving of the U.S. embassy in Moscow (Gwertzman, 1976); from efforts to jam our listening devices, as claimed by the Soviets (Gwertzman, 1976); or from electronic weaponry, or conceivably from innocent communications sources of the type that affect some civilians (but presumably of higher typical pulse intensity, or shorter pulse duration, or in the setting of other exposures that amplify oxidative stress, or with some other feature that amplifies the fraction affected).
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Table 5: Continued.

Room sweep by FBI yielded no devices. (Lederman, Weissenstein, & Lee, 2017)	Weaponry or surveillance would seem perhaps the most likely, given the apparent preferential involvement of CIA operatives under diplomatic cover (Golden & Rotella, 2018). The source of the historical microwave exposure on the U.S. embassy in Moscow was also outside the embassy building. It reportedly originated from the building next door and later from the building across the street (Gwertzman, 1976). Smart meters (or banks of them), outside the room, were the number one reported instigating cause of symptoms in the UCSD survey, with other causes, including base stations or cell towers outside the home. Pulsed RF /MW-producing devices, including so-called “Through the wall” (TTW) surveillance technology, need not be in the room. The exposure can be short term or intermittent; it need not be continuous. For this reason, devices in whatever their location need not remain present after health effects have been produced.
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persons more vulnerable to free radical injury from RF/MW, at a level to which they will likely have subsequent exposure), is not known.

4.2 Fit with Literature. Evidence for health effects of RF/MW is not new (Adams & Williams, 1976; Bergman, 1965; Bolen, 1988; Raines, 1981). A 1971–1972 naval report bearing over 2300 citations, many from Russia and eastern Europe, already documented health effects of microwave/RF/MW, emphasizing “non-ionizing radiation at these frequencies” (Glaser, 1972). Contrary to claims by industry-affiliated parties, copious evidence documents that radiation that is not “ionizing” can also cause health effects. Entire sections of the 1971–1972 report were devoted to each of a number of the symptoms that diplomats are now reporting, including insomnia, headache, fatigue, cognitive problems, and dizziness (Glaser, 1972). Injury from nonionizing radiation occurs also without measurable heating: nonthermal radiation (Avendano, Mata, Sanchez Sarmiento, & Doncel, 2012; Leszczynski, Joenvaara, Reivinen, & Kuokka, 2002; Markova, Hillert, Malmgren, Persson, & Belyaev, 2005). Indeed, oxidative stress, which mediates nonthermal effects, also mediates thermal effects, and melatonin, which defends against oxidative RF/MW injury, also defends against so-called thermal injury (Bekyarova, Tancheva, & Hristova, 2009; Maldonado et al., 2007; Sener, Sehirli, Satiroglu, Keyer-Uysal, & Yegen, 2002a, 2002b; Tunalı, Sener, Yarat, & Emekli, 2005). Moreover, other sources of heat do not produce the same so-called thermal damage that RF/MW does (Bolen, 1988): what are deemed thermal effects may be among the manifestations of oxidative injury. While a low percentage of individuals experience overt symptoms from usual RF/MW, the absolute number may be vast: the fraction with electrosensitivity/electromagnetic illness has been estimated at between 1% and 5%, and is apparently rising (Hillert, Berglind, Arnetz, & Bellander, 2002; Johansson, 2006; Levallois, Neutra, Lee, & Hristova, 2002; Schreier, Huss, & Roosli, 2006; Schröttner & Leitgeb, 2008).

4.3 Limitations. Features of diplomats' experiences rely on media reports and one published neurological evaluation. We did not examine diplomats; however, in conditions with highly distinctive characteristics, the history is often the most important factor in the diagnosis, and diplomats' reports bear highly distinctive characteristics. The close matching of these distinctive characteristics to those of persons with health problems arising in apparent relation to pulsed RF/MW provides a basis for concern that RF/MW exposures may underlie diplomats' symptoms and health conditions.

A tremendous number of physicians and scientists and entities and scientific studies and government reports, in many nations and over many decades, have identified that RF/MW causes symptoms consistent with the spectrum now described for diplomats. Scientific skepticism about RF/MW health effects is well represented in the literature but is of the

industry-fueled stripe (think tobacco): effects of conflicts of interest on research results (as well as on funding, regulatory agencies, legislation and academics) regarding RF/MW, have been repeatedly documented and decried (Alster, 2015; Hardell, 2017; Huss, Egger, Hug, Huwiler-Müntener, & Rösli, 2007; Kostoff & Lau, 2017; Leszczynski, 2015), and evidence of this influence parallels evidence of the potent impact of conflict of interest in medicine more generally (Golomb, 2008). In one illustrative analysis, studies of health effects of cell phones that were funded exclusively by industry were least likely to report a significant effect. Relative to studies funded exclusively by public agencies or charities, the odds ratio was 0.11 (95% CI 0.02–0.78) (Huss et al., 2007)—that is, the odds were about a tenth as great for a significant finding in a study in purely industry-funded studies. The finding was not materially altered when analysis was adjusted for factors like study quality.

Richard Smith, then editor in chief of the *British Medical Journal*, penned an article “Conflicts of Interest: How Money Clouds Objectivity.” Responding to evidence tying study results on a different lucrative product (tobacco) to conflicts of interest (often undisclosed), he suggested, “far from conflict of interest being unimportant in the objective and pure world of science where method and the quality of data is everything, it is the main factor determining the result of studies” (Smith, 2006).

5 Conclusion and Implications

Numerous highly specific features of diplomats’ experiences and symptoms fit the hypothesis of RF/MW injury. If doubts remain, earplugs could be issued to diplomats for use in candidate episodes (e.g. strange noise plus ear pain); these should mute perceived noise from sonic sources (caveat: a sound like crickets chirping may in fact be crickets chirping), but not microwave ones—which may even be intensified. Monitoring for culpable radiation sources must sensitively capture pulsed RF/MW, including that which may be used only on an intermittent basis. It should encompass the 2.4 to 10,000 MHz range in which the Frey effect has been reported. Perhaps attention to diplomats’ plight can ignite awareness of the many others affected by similar problems. Meanwhile, research documenting compatible health effects of RF/MW in a subgroup may inform those caring for diplomats and those in pursuit of causative devices.

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References

- Abdel-Rassoul, G., Abou El-Fateh, O., Abou Salem, M., Michael, A., Farahat, F., El-Batanouny, M., & Salem, E. (2007). Neurobehavioral effects among inhabitants around mobile phone base stations. *NeuroToxicology*, 28, 434–440.
- Abdel-Wahab, M. H., Arafa, H. M., El-Mahdy, M. A., & Abdel-Naim, A. B. (2002). Potential protective effect of melatonin against dibromoacetonitrile-induced oxidative stress in mouse stomach. *Pharmacol Res.*, 46(3), 287–293.
- Abdel Moneim, A. E., Ortiz, F., Leonardo-Mendonca, R. C., Vergano-Villodres, R., Guerrero-Martinez, J. A., Lopez, L. C., Acuna-Castroviejo, et al. (2015). Protective effects of melatonin against oxidative damage induced by Egyptian cobra (*Naja haje*) crude venom in rats. *Acta Trop.*, 143, 58–65.
- Abraham, P., Kolli, V. K., & Rabi, S. (2010). Melatonin attenuates methotrexate-induced oxidative stress and renal damage in rats. *Cell Biochem. Funct.*, 28(5), 426–433.
- Adair, J. C., Baldwin, N., Kornfeld, M., & Rosenberg, G. A. (1999). Radiation-induced blood-brain barrier damage in astrocytoma: Relation to elevated gelatinase B and urokinase. *J Neurooncol*, 44(3), 283–289.
- Adamczyk-Sowa, M., Pierzchala, K., Sowa, P., Mucha, S., Sadowska-Bartosch, I., Adamczyk, J., & Hartel, M. (2014). Melatonin acts as antioxidant and improves sleep in MS patients. *Neurochem. Res.*, 39(8), 1585–1593.
- Adams, R. L., & Williams, R. A. (1976). *Biological effects of electromagnetic radiation (radio waves and microwaves): Eurasian Communist countries*. Washington, DC: Defense Intelligence Agency.
- Adlkofer, F., & Richter, K. (2011). *Radiation protection in conflict with science*. Saarbrücken: Competence Initiative for the Protection of Humanity, Environment and Democracy.
- Agrawal, N., Ray, R. S., Farooq, M., Pant, A. B., & Hans, R. K. (2007). Photosensitizing potential of ciprofloxacin at ambient level of UV radiation. *Photochem. Photobiol.*, 83(5), 1226–1236.
- Ahsan, H., Ali, A., & Ali, R. (2003). Oxygen free radicals and systemic autoimmunity. *Clin. Exp. Immunol.*, 131(3), 398–404.
- Aitken, R. J., Bennetts, L. E., Sawyer, D., Wiklendt, A. M., & King, B. V. (2005). Impact of radio frequency electromagnetic radiation on DNA integrity in the male germline. *Int. J. Androl.*, 28(3), 171–179.
- Akpınar, D., Ozturk, N., Ozen, S., Agar, A., & Yargicoglu, P. (2012). The effect of different strengths of extremely low-frequency electric fields on antioxidant status, lipid peroxidation, and visual evoked potentials. *Electromagn. Biol. Med.*, 31(4), 436–448.
- Akter, U., Niwa, M., Nose, T., Kaida, T., Matsuno, H., Kozawa, O., & Uematsu, T. (1998). Effects of several agents on UVB- and UVA plus systemic fluoroquinolone-induced erythema of guinea pig skin evaluated by reflectance colorimetry. *Free Radic. Biol. Med.*, 24(7–8), 1113–1119.
- Al Ahmad, A., Gassmann, M., & Ogunshola, O. O. (2012). Involvement of oxidative stress in hypoxia-induced blood-brain barrier breakdown. *Microvasc. Res.*, 84(2), 222–225.

- Alsanosi, A. A., Al-Momani, M. O., Hagr, A. A., Almomani, F. M., Shami, I. M., & Al-Habeeb, S. F. (2013). The acute auditory effects of exposure for 60 minutes to mobile's electromagnetic field. *Saudi Med. J.*, 34(2), 142–146.
- Alster, N. (2015). *Captured agency: How the Federal Communications Commission is dominated by the industries it presumably regulates*. Harvard University, Edmond J. Safra Center for Ethics. www.harvard.ethics.edu (<https://creativecommons.org/licenses/by/4.0/>)
- Altpeter, E. S., Krebs, T., Pfluger, D. H., von Kanel, J., Blattman, R., et al. (1995). *Study on health effects of the shortwave transmitter station of Schwarzenburg, Berne, Switzerland* (Study 55). Berne, Switzerland: Federal Office of Energy.
- Anstey, A. V. (1999). Photosensitivity in the Smith-Lemli-Opitz syndrome. *Photodermatol. Photoimmunol. Photomed.*, 15(6), 217–218.
- Anstey, A. (2001). Photomedicine: Lessons from the Smith-Lemli-Opitz syndrome. *J. Photochem. Photobiol. B.*, 62(3), 123–127.
- Anstey, A. (2006). School in photodermatology: Smith-Lemli-Opitz syndrome. *Photodermatol. Photoimmunol. Photomed.*, 22(4), 200–204.
- Anstey, A. V., Azurdia, R. M., Rhodes, L. E., Pearse, A. D., & Bowden, P. E. (2005). Photosensitive Smith-Lemli-Opitz syndrome is not caused by a single gene mutation: Analysis of the gene encoding 7-dehydrocholesterol reductase in five U.K. families. *Br. J. Dermatol.*, 153(4), 774–779.
- Anstey, A. V., Ryan, A., Rhodes, L. E., Charman, C. R., Arlett, C. F., Tyrrell, R. M., Taylor, C., et al. (1999). Characterization of photosensitivity in the Smith-Lemli-Opitz syndrome: A new congenital photosensitivity syndrome. *Br. J. Dermatol.*, 141(3), 406–414.
- Anstey, A. V., & Taylor, C. R. (1999). Photosensitivity in the Smith-Lemli-Opitz syndrome: The US experience of a new congenital photosensitivity syndrome. *J. Am. Acad. Dermatol.*, 41(1), 121–123.
- Antunes Wilhelm, E., Ricardo Jesse, C., Folharini Bortolatto, C., & Wayne Nogueira, C. (2013). Correlations between behavioural and oxidative parameters in a rat quinolinic acid model of Huntington's disease: Protective effect of melatonin. *Eur. J. Pharmacol.*, 701(1–3), 65–72.
- Aoki, M., Nata, T., Morishita, R., Matsushita, H., Nakagami, H., Yamamoto, K., Yamakazi, K., et al. (2001). Endothelial apoptosis induced by oxidative stress through activation of NF-kappaB: Antiapoptotic effect of antioxidant agents on endothelial cells. *Hypertension*, 38(1), 48–55.
- Aranda, M., Albendea, C. D., Lostale, F., Lopez-Pingarron, L., Fuentes-Broto, L., Martinez-Ballarín, E., Reiter, R. J., et al. (2010). In vivo hepatic oxidative stress because of carbon tetrachloride toxicity: Protection by melatonin and pinoline. *J. Pineal. Res.*, 49(1), 78–85.
- Argun, M., Tok, L., Uguz, A. C., Celik, O., Tok, O. Y., & Naziroglu, M. (2014). Melatonin and amfenac modulate calcium entry, apoptosis, and oxidative stress in ARPE-19 cell culture exposed to blue light irradiation (405 nm). *Eye (Lond.)*, 28(6), 752–760.
- Aschermann, C. (2009). *Observations from a psychotherapy practice on Mobile telecommunications and DECT telephones* (rev. and ext.). Trans. Margaret E. White.
- Associated Press. (2017a). Dangerous sound? What Americans heard in Cuba attacks. October 12.

- Associated Press. (2017b). Tillerson says diplomats in Havana suffered "health attacks." *Los Angeles Times*, August 12.
- Associated Press. (2017c). Bizarre Cuba mystery: Did sonic weapon cause U.S. diplomats brain injury? *Mercury News*, September 14.
- Associated Press. (2017d). US Cuban diplomats to discuss health incidents, September 18.
- Associated Press in Washington. (2017). Mystery of sonic weapon attacks at US embassy in Cuba deepens. *Guardian*, September 14.
- Au, W. W., Cantelli-Forti, G., Hrelia, P., & Legator, M. S. (1990). Cytogenetic assays in genotoxic studies: Somatic cell effects of benzene and germinal cell effects of dibromochloropropane. *Teratogen. Carcinogen Mutagen*, 10, 125–134.
- Avendano, C., Mata, A., Sanchez Sarmiento, C. A., & Doncel, G. F. (2012). Use of laptop computers connected to Internet through Wi-Fi decreases human sperm motility and increases sperm DNA fragmentation. *Fertil. Steril.*, 97(1), 39–45 e32.
- Ayata, A., Mollaoglu, H., Yilmaz, H. R., Akturk, O., Ozguner, F., & Altuntas, I. (2004). Oxidative stress-mediated skin damage in an experimental mobile phone model can be prevented by melatonin. *J. Dermatol.*, 31(11), 878–883.
- Aynali, G., Naziroglu, M., Celik, O., Dogan, M., Yariktas, M., & Yasan, H. (2013). Modulation of wireless (2.45 GHz)-induced oxidative toxicity in laryngotracheal mucosa of rat by melatonin. *Eur. Arch. Otorhinolaryngol.*, 270(5), 1695–1700.
- Azam, S., Hadi, N., Khan, N. U., & Hadi, S. M. (2003). Antioxidant and prooxidant properties of caffeine, theobromine and xanthine. *Med. Sci. Monit.*, 9(9), BR325–BR330.
- Azurdia, R. M., Anstey, A. V., & Rhodes, L. E. (2001). Cholesterol supplementation objectively reduces photosensitivity in the Smith-Lemli-Opitz syndrome. *Br. J. Dermatol.*, 144(1), 143–145.
- Back, S. A., Luo, N. L., Mallinson, R. A., O'Malley, J. P., Wallen, L. D., Frei, B., Morrow, J. D. (2005). Selective vulnerability of preterm white matter to oxidative damage defined by F2-isoprostanes. *Ann. Neurol.*, 58(1), 108–120.
- Bagchi, M., Balmoori, J., Ye, X., Bagchi, D., Ray, S. D., & Stohs, S. J. (2001). Protective effect of melatonin on naphthalene-induced oxidative stress and DNA damage in cultured macrophage J774A.1 cells. *Mol. Cell Biochem.*, 221(1–2), 49–55.
- Bahreymi Toossi, M. H., Sadeghnia, H. R., Mohammad Mahdizadeh Feyzabadi, M., Hosseini, M., Hedayati, M., Mosallanejad, R., Beheshti, F., et al. (2017). Exposure to mobile phone (900–1800 MHz) during pregnancy: Tissue oxidative stress after childbirth. *J. Matern.-Fetal Neonatal Med.*, 31, 1298–1303. 1–6.
- Baiomy, A. A., Attia, H. F., Soliman, M. M., & Makrum, O. (2015). Protective effect of ginger and zinc chloride mixture on the liver and kidney alterations induced by malathion toxicity. *Int. J. Immunopathol. Pharmacol.*, 28(1), 122–128.
- Balekouzou, A., Yin, P., Afewerky, H. K., Bekolo, C., Pamatika, C. M., Nambei, S. W., Djeintote, M., et al. (2017). Behavioral risk factors of breast cancer in Bangui of Central African Republic: A retrospective case-control study. *PLoS One*, 12(2), e0171154.
- Bandyopadhyay, D., Ghosh, G., Bandyopadhyay, A., & Reiter, R. J. (2004). Melatonin protects against piroxicam-induced gastric ulceration. *J. Pineal Res.*, 36(3), 195–203.

- Bardak, Y., Ozerturk, Y., Ozguner, F., Durmus, M., & Delibas, N. (2000). Effect of melatonin against oxidative stress in ultraviolet-B exposed rat lens. *Curr. Eye Res.*, 20(3), 225–230.
- Barichello, T., Lemos, J. C., Generoso, J. S., Cipriano, A. L., Milioli, G. L., Marcelino, D. M., Vuolo, F., et al. (2011). Oxidative stress, cytokine/chemokine and disruption of blood-brain barrier in neonate rats after meningitis by *Streptococcus agalactiae*. *Neurochem Res.*, 36, 1922–1930.
- Barnes, D. E., & Bero, L. A. (1998). Why review articles on the health effects of passive smoking reach different conclusions. *JAMA*, 279(19), 1566–1570.
- Barnes, F. S., & Greenebaum, B. (2015). The effects of weak magnetic fields on radical pairs. *Bioelectromagnetics*, 36(1), 45–54.
- Barnes, F., & Greenebaum, B. (2016). Some effects of weak magnetic fields on biological systems: RF fields can change radical concentrations and cancer cell growth rates. *IEEE Power Electronics Magazine*, 3(1), 60–68.
- Baxi, D. B., Singh, P. K., Vachhrajani, K. D., & Ramachandran, A. V. (2013). Melatonin supplementation in rat ameliorates ovariectomy-induced oxidative stress. *Climacteric*, 16(2), 274–283.
- Beatty, S., Koh, H., Phil, M., Henson, D., & Boulton, M. (2000). The role of oxidative stress in the pathogenesis of age-related macular degeneration. *Surv. Ophthalmol.*, 45(2), 115–134.
- Behan, W. M., McDonald, M., Darlington, L. G., & Stone, T. W. (1999). Oxidative stress as a mechanism for quinolinic acid-induced hippocampal damage: Protection by melatonin and deprenyl. *Br. J. Pharmacol.*, 128(8), 1754–1760.
- Bekyarova, G., Tancheva, S., & Hristova, M. (2009). Protective effect of melatonin against oxidative hepatic injury after experimental thermal trauma. *Methods Find. Exp. Clin. Pharmacol.*, 31(1), 11–14.
- Bell, A. (2017). *Poisoned: How a crime-busting prosecutor turned his medical mystery into a crusade for environmental victims*. New York: Skyhorse Publishing.
- Belpomme, D., Campagnac, C., & Irigaray, P. (2015). Reliable disease biomarkers characterizing and identifying electrohypersensitivity and multiple chemical sensitivity as two etiopathogenic aspects of a unique pathological disorder. *Rev. Environ. Health*, 30(4), 251–271.
- Belyaev, I. Y., Koch, C. B., Terenius, O., Roxstrom-Lindquist, K., Malmgren, L. O., W, H. S., Salford, L. G., et al. (2006). Exposure of rat brain to 915 MHz GSM microwaves induces changes in gene expression but not double stranded DNA breaks or effects on chromatin conformation. *Bioelectromagnetics*, 27(4), 295–306.
- Belyaev, I. Y., Sheheglov, V. S., Alipov, E. D., & Ushakov, V. D. (2000). Non-thermal effects of extremely high-frequency microwaves on chromatin conformation in cells in vitro: Dependence on physical, physiological, and genetic factors. *IEEE Transactions on Microwave Theory and Techniques*, 48(11), 2172–2179.
- Benderitter, M., Vincent-Genod, L., Pouget, J. P., & Voisin, P. (2003). The cell membrane as a biosensor of oxidative stress induced by radiation exposure: A multi-parameter investigation. *Radiat. Res.*, 159(4), 471–483.
- Bergman, W. (1965). *The effect of microwaves on the central nervous system*. Trans. Technical Library Research Service, Ford Motor Company.

- Bergqvist, U., Vogel, E., Aringer, L., Cunningham, J., Gobba, F., Leitgeb, N., Miro, L., et al. (Eds.). (1997). *Possible health implications of subjective symptoms and electromagnetic fields: A report prepared by a European group of experts for the European Commission, DG V*. Solna, Sweden: European Commission Directorate General V. Employment, Industrial Relations and Social Affairs, National Institute for Working Life, Sweden.
- Bero, L., Oostvogel, F., Bacchetti, P., & Lee, K. (2007). Factors associated with findings of published trials of drug-drug Comparisons: Why some statins appear more efficacious than others. *PLoS Med.*, 4(6), e184.
- Berr, C., Balansard, B., Arnaud, J., Roussel, A. M., & Alperovitch, A. (2000). Cognitive decline is associated with systemic oxidative stress: The EVA study. *J. Am. Geriatr. Soc.*, 48(10), 1285–1291.
- Bhatia, A. L., & Manda, K. (2004). Study on pre-treatment of melatonin against radiation-induced oxidative stress in mice. *Environ. Toxicol. Pharmacol.*, 18(1), 13–20.
- Bigorra, D. (2016). *Electromagnetic hypersensitivity is on the rise*. <http://mieuxprevenir.blogspot.com/2017/01/electrohypersensitivity-is-on-rise.html>
- Bilgili, B., Akar, A., Avci, B., & Tuncel, O. K. (2013). Effect of 900 MHz radiofrequency radiation on oxidative stress in rat brain and serum. *Electromagn. Biol. Med.*, 32(1), 20–29.
- Bilski, P., Martinez, L. J., Koker, E. B., & Chignell, C. F. (1996). Photosensitization by norfloxacin is a function of pH. *Photochem. Photobiol.*, 64(3), 496–500.
- Birenbaum, L., Grosz, G. M., Rosenthal, S. W. Z., & Zaret, M. M. (1969). Effect of microwaves on the eye. *IEEE Transactions on Biomedical Engineering*, 16(1), 7–14.
- Blasig, I. E., Mertsch, K., & Haseloff, R. F. (2002). Nitronyl nitroxides, a novel group of protective agents against oxidative stress in endothelial cells forming the blood-brain barrier. *Neuropharmacology*, 43(6), 1006–1014.
- Board, T. E. (2017). Cuba and the mystery of sonic weapons. *New York Times*, October 6.
- Bocumini, L. E., Fowler, C. L., Campbell, T. A., Puertolas, L. F., & Kaidbey, K. H. (2000). Photoreaction potential of orally administered levofloxacin in healthy subjects. *Ann. Pharmacother.*, 34(4), 453–458.
- Bolen, S. M. (1988). *Radiofrequency/microwave radiation biological effects and safety standards: A review*. (No. RL-TR-94-53). New York: Rome Laboratory, Air Force Materiel Command, Griffiss Air Force Base.
- Bonnafous, P., Vernhes, M.-C., Teissie, J., & Gabriel, B. (1999). The generation of reactive-oxygen species associated with long-lasting pulse-induced electroporation of mammalian cells is based on a non-destructive alteration of the plasma membrane. *Biochimica et Biophysica Acta—Biomembranes*, 1461(1), 123–134.
- Bonne, C., & Muller, A. (2000). [Role of oxidative stress in age-related macular degeneration]. *J. Fr. Ophthalmol.*, 23(8), 835–840.
- Bowry, V. W., Mohr, D., Cleary, J., & Stocker, R. (1995). Prevention of tocopherol-mediated peroxidation in ubiquinol-10-free human low density lipoprotein. *J. Biol. Chem.*, 270(11), 5756–5763.
- Bravard, A., Ageron-Blanc, A., Alvarez, S., Drane, P., Le Rhun, Y., Paris, F., Luccion, C., et al. (2002). Correlation between antioxidant status, tumorigenicity and radiosensitivity in sister rat cell lines. *Carcinogenesis*, 23(5), 705–711.

- Brea-Calvo, G., Rodriguez-Hernandez, A., Fernandez-Ayala, D. J., Navas, P., & Sanchez-Alcazar, J. A. (2006). Chemotherapy induces an increase in coenzyme Q10 levels in cancer cell lines. *Free Radic. Biol. Med.*, 40(8), 1293–1302.
- Bresgen, N., Karlhuber, G., Krizbai, I., Bauer, H., Bauer, H. C., & Eckl, P. M. (2003). Oxidative stress in cultured cerebral endothelial cells induces chromosomal aberrations, micronuclei, and apoptosis. *J. Neurosci. Res.*, 72(3), 327–333.
- Brinchman, S. (2011). Living nightmare: How SDG&E led to headaches, hearing loss. lamesa.patch.come/blog_posts, August 14.
- Brown, D. (2001). Heart drug far surpasses expectations. *Washington Post*, May 19, p. A1.
- Brubaker, J. W., Mohny, B. G., & Pulido, J. S. (2009). Cystoid macular edema in a patient with chronic progressive external ophthalmoplegia with mitochondrial myopathy. *Ophthalmic Genet.*, 30(1), 50–53.
- Bruck, R., Aeed, H., Avni, Y., Shirin, H., Matas, Z., Shahmurov, M., Avinoach, I., et al. (2004). Melatonin inhibits nuclear factor kappa B activation and oxidative stress and protects against thioacetamide induced liver damage in rats. *J. Hepatol.*, 40(1), 86–93.
- Bua, L., Tibaldi, E., Falcioni, L., Lauriola, M., De Angelis, L., Gnudi, F., Manservigi, M., et al. (2018). Results of lifespan exposure to continuous and intermittent extremely low frequency electromagnetic fields (ELFEMF) administered alone to Sprague Dawley rats. *Environ. Res.*, 164, 271–279.
- Buckley, C., & Harris, G. (2018). First Cuba, now China? An American falls ill after “abnormal” sounds. *New York Times*, May 23.
- Burch, J. B., Reif, J. S., & Yost, M. G. (1999). Geomagnetic disturbances are associated with reduced nocturnal excretion of a melatonin metabolite in humans. *Neurosci. Lett.*, 266(3), 209–212.
- Burch, J. B., Reif, J. S., & Yost, M. G. (2008). Geomagnetic activity and human melatonin metabolite excretion. *Neurosci. Lett.*, 438(1), 76–79.
- Burdelya, L. G., Gleiberman, A. S., Toshkov, I., Aygun-Sunar, S., Bapardekar, M., Manderscheid-Kern, P., Bellnier, D., et al. (2012). Toll-like receptor 5 agonist protects mice from dermatitis and oral mucositis caused by local radiation: Implications for head-and-neck cancer radiotherapy. *Int. J. Radiat. Oncol. Biol. Phys.*, 83(1), 228–234.
- Burdge, D. R., Nakielna, E. M., & Rabin, H. R. (1995). Photosensitivity associated with ciprofloxacin use in adult patients with cystic fibrosis. *Antimicrob. Agents Chemother.*, 39(3), 793.
- Cadenas, S., & Barja, G. (1999). Resveratrol, melatonin, vitamin E, and PBN protect against renal oxidative DNA damage induced by the kidney carcinogen KBrO₃. *Free Radic. Biol. Med.*, 26(11–12), 1531–1537.
- Cain, C. A., & Rissmann, W. J. (1978). Mammalian auditory responses to 3.0 GHz microwave pulses. *IEEE Trans. Biomed. Eng.*, 25, 288–293.
- Calderon-Margalit, R., Adler, B., Abramson, J. H., Gofin, J., & Kark, J. D. (2006). Butyrylcholinesterase activity, cardiovascular risk factors, and mortality in middle-aged and elderly men and women in Jerusalem. *Clin. Chem.*, 52(5), 845–852.
- Carelli, V., Ross-Cisneros, F. N., & Sadun, A. A. (2002). Optic nerve degeneration and mitochondrial dysfunction: Genetic and acquired optic neuropathies. *Neurochem. Int.*, 40(6), 573–584.

- Carpenter, D. O. (2014). Excessive exposure to radiofrequency electromagnetic fields may cause the development of electrohypersensitivity. *Altern. Ther. Health Med.*, 20(6), 40–42.
- Carrillo-Vico, A., Lardone, P. J., Naji, L., Fernandez-Santos, J. M., Martin-Lacave, I., Guerrero, J. M., Calvo, J. R., et al. (2005). Beneficial pleiotropic actions of melatonin in an experimental model of septic shock in mice: Regulation of pro-/anti-inflammatory cytokine network, protection against oxidative damage and anti-apoptotic effects. *J. Pineal Res.*, 39(4), 400–408.
- Casta, A., Quackebush, E. J., Houck, C. S., & Korson, M. S. (1997). Perioperative white matter degeneration and death in a patient with a defect in mitochondrial oxidative phosphorylation. *Anesthesiology*, 87(2), 420–425.
- Celiker, M., Özgür, A., Tümkaya, L., Terzi, S., Yılmaz, M., Kalkan, Y., Erdogan, E., et al. (2016). Effects of exposure to 2100MHz GSM-like radiofrequency electromagnetic field on auditory system of rats. *Braz. J. Otorhinolaryngol.*, November 5. doi:30210.31016/j.bjorl.32016.30210.30004.
- Ceyhan, A. M., Akkaya, V. B., Gulecol, S. C., Ceyhan, B. M., Ozguner, F., & Chen, W. (2012). Protective effects of beta-glucan against oxidative injury induced by 2.45-GHz electromagnetic radiation in the skin tissue of rats. *Arch. Dermatol. Res.*, 304(7), 521–527.
- Chabra, A., Shokrzadeh, M., Naghshvar, F., Salehi, F., & Ahmadi, A. (2014). Melatonin ameliorates oxidative stress and reproductive toxicity induced by cyclophosphamide in male mice. *Hum. Exp. Toxicol.*, 33(2), 185–195.
- Charman, C. R., Ryan, A., Tyrrell, R. M., Pearse, A. D., Arlett, C. F., Kurwa, H. A., Shortland, G., et al. (1998). Photosensitivity associated with the Smith-Lemli-Opitz syndrome. *Br. J. Dermatol.*, 138(5), 885–888.
- Chen, K. B., Lin, A. M., & Chiu, T. H. (2003). Oxidative injury to the locus coeruleus of rat brain: Neuroprotection by melatonin. *J. Pineal. Res.*, 35(2), 109–117.
- Chen, L. J., Gao, Y. Q., Li, X. J., Shen, D. H., & Sun, F. Y. (2005). Melatonin protects against MPTP/MPP+–induced mitochondrial DNA oxidative damage in vivo and in vitro. *J. Pineal Res.*, 39(1), 34–42.
- Chen, P., Yang, Y. Q., Tao, H. H., & Yang, H. C. (2006). [Effects of electromagnetic fields of different frequencies on proliferation and DNA damage of gallbladder cancer cells]. *Nan Fang Yi Ke Da Xue Xue Bao*, 26(3), 328–330.
- Chen, Q., Li, X., Wu, L., Qi, Y., & Wu, X. (1998). Mitochondrial gene defect in patients with chronic progressive external ophthalmoplegia. *Chin. Med. J.*, 111(6), 500–503.
- Chen, Y., Meyer, J. N., Hill, H. Z., Lange, G., Condon, M. R., Klein, J. C., Ndirangu, D., et al. (2017). Role of mitochondrial DNA damage and dysfunction in veterans with Gulf War illness. *PLoS One*, 12(9), e0184832.
- Chen, Y. P. (2006). Microwave treatment of eight seconds protects cells of *Isatis indigotica* from enhanced UVB radiation lesions. *Photochem. Photobiol.*, 82(2), 503–507.
- Cherry, N., Mackness, M., Durrington, P., Povey, A., Dippnall, M., Smith, T., Mackness, B., et al. (2002). Paraoxonase (PON1) polymorphisms in farmers attributing ill health to sheep dip. *Lancet*, 359(9308), 763–764.
- Chetelat, A. A., Albertini, S., & Gocke, E. (1996). The photomutagenicity of fluoroquinolones in tests for gene mutation, chromosomal aberration, gene conversion and DNA breakage (Comet assay). *Mutagenesis*, 11(5), 497–504.

- Chiang, H., Yao, G. D., Fang, Q. S., Wang, K. Q., Lu, D. Z., & Zhou, Y. K. (1989). Health effects of environmental electromagnetic fields. *J. Bioelectricity*, 8(1), 127–131.
- Chignell, C. F., Kukielszak, B. M., Sik, R. H., Bilski, P. J., & He, Y. Y. (2006). Ultraviolet A sensitivity in Smith-Lemli-Opitz syndrome: Possible involvement of cholesta-5,7,9(11)-trien-3 beta-ol. *Free Radic. Biol. Med.*, 41(2), 339–346.
- Cho, S., Breedlove, J. J., & Gunning, S. T. (2008). Radiation recall reaction induced by levofloxacin. *J. Drugs Dermatol.*, 7(1), 64–67.
- Cho, Y. M., Lim, H. J., Jang, H., Kim, K., Choi, J. W., Shin, C., Lee, S. K., et al. (2016). A follow-up study of the association between mobile phone use and symptoms of ill health. *Environ. Health Toxicol.*, 32, e2017001.
- Clarke, T. K., Lupton, M. K., Fernandez-Pujals, A. M., Starr, J., Davies, G., Cox, S., Pattie, A., et al. (2015). Common polygenic risk for autism spectrum disorder (ASD) is associated with cognitive ability in the general population. *Mol. Psychiatry*, 21, 419–425.
- Cleary, S. F. (1980). Microwave cataractogenesis. *Proceedings IEEE*, 68, 49–55.
- Clegg, F. (2013). Electrohypersensitivity Is real. *Huffington Post*, June 12. huffingtonpost.ca/frank-clegg/post_5393_b_3745157.html
- Cochrane, E. (2017). Mysterious health issues drove diplomats from Cuba. *New York Times*, August 10.
- Conklin, S. M., Harris, J. I., Manuck, S. B., Yao, J. K., Hibbeln, J. R., & Muldoon, M. F. (2007). Serum omega-3 fatty acids are associated with variation in mood, personality and behavior in hypercholesterolemic community volunteers. *Psychiatry Res.*, 152(1), 1–10.
- Conrad, R., & Friedman, E. (2013). *Smart meter health effects survey and report, exhibit D*. ME Public Utilities Commission, Docket 2011-00262 (Item 210). <http://www.mainecoalitiontostopsmartmeters.org/wp-content/uploads/2013/2001/Exhibit-2010-Smart-Meter-Health-Effects-Report-Survey2012.pdf>
- Counter, S. A. (1993). Electromagnetic stimulation of the auditory system: Effects and side-effects. *Scand. Audiol. Suppl.*, 37, 1–3.
- Crews, F. (2017). *Freud: The making of an illusion*. New York: Holt.
- Cruz, A., Padillo, F. J., Granados, J., Tunez, I., Munoz, M. C., Briceno, J., Pera-Madrado, et al. (2003). Effect of melatonin on cholestatic oxidative stress under constant light exposure. *Cell Biochem. Funct.*, 21(4), 377–380.
- Cuba's sonic attacks. (2017). *Wall Street Journal*, September 26, A16.
- Cui, J., Zhong, R., Chu, E., Zhang, X. F., Zhang, W. G., Fang, C. F., Dong, Q., et al. (2012). Correlation between oxidative stress and L-type calcium channel expression in the ventricular myocardia of selenium-deficient mice. *J. Int. Med. Res.*, 40(5), 1677–1687.
- Cutz, A. (1989). Effects of microwave radiation on the eye: The occupational health perspective. *Lens and Eye Toxicity Research*, 6(1&2), 379–386.
- Dabbeni-Sala, F., Floreani, M., Franceschini, D., Skaper, S. D., & Giusti, P. (2001). Kainic acid induces selective mitochondrial oxidative phosphorylation enzyme dysfunction in cerebellar granule neurons: Protective effects of melatonin and GSH ethyl ester. *FASEB J*, 15(10), 1786–1788.
- Daily, L., Wakim, K. G., Herrick, J. F., Parkhill, E. M., & Benedict, W. L., et al. (1952). The effects of microwave diathermy on the eye. *Am. J. Ophthalm.*, 35, 1001.
- Dales, M. J. M. (2000). Statination. *Internal Medicine News*, February 1, 55.

- Das, A., Belagodu, A., Reiter, R. J., Ray, S. K., & Banik, N. L. (2008). Cytoprotective effects of melatonin on C6 astroglial cells exposed to glutamate excitotoxicity and oxidative stress. *J. Pineal Res.*, 45(2), 117–124.
- Dawson, G. A., Brown, S. I., & Tellefsen, L. (2009). A drug-related phototoxic reaction and its possible relationship to a radiation-induced skin reaction. *Oncologist*, 14(3), 303–306.
- de Gannes, F. P., Billaudel, B., Taxile, M., Haro, E., Ruffie, G., Leveque, P., Veyret, B., et al. (2009). Effects of head-only exposure of rats to GSM-900 on blood-brain barrier permeability and neuronal degeneration. *Radiat. Res.*, 172(3), 359–367.
- De Luca, C., Chung Sheun Thai, J., Raskovic, D., Cesareo, E., Caccamo, D., Trukhanov, A., & Korkina, L., et al. (2014). Metabolic and genetic screening of electromagnetic hypersensitive subjects as a feasible tool for diagnostics and intervention. *Mediators Inflamm.*, 2014, 924184.
- Dehghan, F., Khaksari Hadad, M., Asadikram, G., Najafipour, H., & Shahrokhi, N. (2013). Effect of melatonin on intracranial pressure and brain edema following traumatic brain injury: Role of oxidative stresses. *Arch. Med. Res.*, 44(4), 251–258.
- Del Vecchio, G., Giuliani, A., Fernandez, M., Mesirca, P., Bersani, F., Pinto, R., Ardoino, L., et al. (2009). Effect of radiofrequency electromagnetic field exposure on in vitro models of neurodegenerative disease. *Bioelectromagnetics*, 30(7), 564–572.
- Dethmers, J. K., & Meister, A. (1981). Glutathione export by human lymphoid cells: Depletion of glutathione by inhibition of its synthesis decreases export and increases sensitivity to irradiation. *Proc. Natl. Acad. Sci. USA*, 78(12), 7492–7496.
- Did GSK trial data mask Paxil suicide risk? (2008). *New Scientist*, February 8, 12.
- Ding, K., Wang, H., Xu, J., Li, T., Zhang, L., Ding, Y., Zhu, L., et al. (2014). Melatonin stimulates antioxidant enzymes and reduces oxidative stress in experimental traumatic brain injury: The Nrf2-ARE signaling pathway as a potential mechanism. *Free Radic. Biol. Med.*, 73, 1–11.
- Dodson, R. F., Patten, B. M., Hyman, B. M., & Chu, L. W. (1976). Mitochondrial abnormalities in progressive ophthalmoplegia. *Cytobios.*, 15(57), 57–60.
- Dogan, M. S., Yavaş, M. C., Günay, A., Yavuz, A. Z., Deveci, E., Akkuş, Z., Tanik, A., et al. (2017). The protective effect of melatonin and *Ganoderma lucidum* against the negative effects of extremely low frequency electric and magnetic fields on pulp structure in rat teeth. *Biotechnology and Biotechnological Equipment*, 31(5), 979–988.
- Dolk, H., Shaddick, G., Walls, P., Grundy, C., Thakrar, B., Kleinschmidt, I., & Elliott, P. (1997). Cancer incidence near radio and television transmitters in Great Britain. I. Sutton Coldfield transmitter. *Am. J. Epidemiol.*, 145(1), 1–9.
- Duan, Y., Wang, Z., Zhang, H., He, Y., Lu, R., Zhang, R., Sun, G., et al. (2013). The preventive effect of lotus seedpod procyanidins on cognitive impairment and oxidative damage induced by extremely low frequency electromagnetic field exposure. *Food Funct.*, 4(8), 1252–1262.
- Durusoy, R., Hassoy, H., Ozkurt, A., & Karababa, A. O. (2017). Mobile phone use, school electromagnetic field levels and related symptoms: A cross-sectional survey among 2150 high school students in Izmir. *Environ. Health*, 16(1), 51.
- Dutta, A., Chakraborty, A., Saha, A., Ray, S., & Chatterjee, A. (2005). Interaction of radiation- and bleomycin-induced lesions and influence of glutathione level on the interaction. *Mutagenesis*, 20(5), 329–335.

- Eapen, B. R. (2007). Photosensitivity in Smith-Lemli-Opitz syndrome: A flux balance analysis of altered metabolism. *Bioinformation*, 2(2), 78–82.
- Ebaid, H., Bashandy, S. A., Alhazza, I. M., Rady, A., & El-Shehry, S. (2013). Folic acid and melatonin ameliorate carbon tetrachloride-induced hepatic injury, oxidative stress and inflammation in rats. *Nutr. Metab.*, 10(1), 20.
- Eger, H., & Jahn, M. (2010). Specific health symptoms and cell phone radiation in Selbitz (Bavaria, Germany) = Evidence of a dose-response relationship. *Umwelt-Medizin-Gesellschaft*, 23.
- EHS Foreningen (EHS Association). (2018). Hearing at the Danish Parliament on wireless radiation puts pressure on the National Board of Health. Press release, April 10. <https://via.ritzau.dk/pressemeddelelse?publisherId=12609765&releaseId=12609776>
- Eisele, S., Garbe, E., Zeitz, M., Schneider, T., & Somasundaram, R. (2009). Ciprofloxacin-related acute severe myalgia necessitating emergency care treatment: A case report and review of the literature. *Int. J. Clin. Pharmacol. Ther.*, 47(3), 165–168.
- El-Helaly, M., & Abu-Hashem, E. (2010). Oxidative stress, melatonin level, and sleep insufficiency among electronic equipment repairers. *Indian J. Occup. Environ. Med.*, 14(3), 66–70.
- El-Missiry, M. A., Fayed, T. A., El-Sawy, M. R., & El-Sayed, A. A. (2007). Ameliorative effect of melatonin against gamma-irradiation-induced oxidative stress and tissue injury. *Ecotoxicol. Environ. Saf.*, 66(2), 278–286.
- El-Missiry, M. A., Othman, A. I., Al-Abdan, M. A., & El-Sayed, A. A. (2014). Melatonin ameliorates oxidative stress, modulates death receptor pathway proteins, and protects the rat cerebrum against bisphenol-A-induced apoptosis. *J. Neurol. Sci.*, 347(1–2), 251–256.
- El-Sokkary, G. H. (2000). Melatonin protects against oxidative stress induced by the kidney carcinogen KBrO(3). *Neuro. Endocrinol. Lett.*, 21(6), 461–468.
- El-Sokkary, G. H., Nafady, A. A., & Shabash, E. H. (2010). Melatonin administration ameliorates cadmium-induced oxidative stress and morphological changes in the liver of rat. *Ecotoxicol. Environ. Saf.*, 73(3), 456–463.
- Elder, J. A., & Chou, C. K. (2003). Auditory response to pulsed radiofrequency energy. *Bioelectromagnetics (Suppl. 6)*, S162–S173.
- Elmorsy, E., Elzalabany, L. M., Elsheikha, H. M., & Smith P. A. (2014). Adverse effects of anti psychotics on micro-vascular endothelial cells of the human blood-brain barrier. *Brain Res.*, 1583, 255–268.
- Eltiti, S., Wallace, D., Zougkou, K., Russo, R., Joseph, S., Rasor, P., & Fox, E. (2007). Development and evaluation of the electromagnetic hypersensitivity questionnaire. *Bioelectromagnetics*, 28(2), 137–151.
- Elwood, J. M. (2012). Microwaves in the cold war: The Moscow embassy study and its interpretation: Review of a retrospective cohort study. *Environmental Health*, 11, 85.
- Enciu, A. M., Gherghiceanu, M., & Popescu, B. O. (2013). Triggers and effectors of oxidative stress at blood-brain barrier level: Relevance for brain ageing and neurodegeneration. *Oxid. Med. Cell Longev.*, 2013, 297512.
- Engin, A. B., Sepici-Dincel, A., Gonul, I. I., & Engin, A. (2012). Oxidative stress-induced endothelial cell damage in thyroidectomized rat. *Exp. Toxicol. Pathol.*, 64(5), 481–485.

- Epperly, M. W., Kagan, V. E., Sikora, C. A., Gretton, J. E., Defilippi, S. J., Bar-Sagi, D., & Greenberger, S., et al. (2001). Manganese superoxide dismutase-plasmid/liposome (MnSOD-PL) administration protects mice from esophagitis associated with fractionated radiation. *Int. J. Cancer*, 96(4), 221–231.
- Esenkaya, I., & Unay, K. (2011). Tendon, tendon healing, hyperlipidemia and statins. *Muscles Ligaments Tendons J.*, 1(4), 169–171.
- Esmekaya, M. A., Ozer, C., & Seyhan, N. (2011). 900 MHz pulse-modulated radiofrequency radiation induces oxidative stress on heart, lung, testis and liver tissues. *Gen. Physiol. Biophys.*, 30(1), 84–89.
- Espino, J., Bejarano, I., Ortiz, A., Lozano, G. M., Garcia, J. F., Pariente, J. A., & Rodriguez, A. B., et al. (2010). Melatonin as a potential tool against oxidative damage and apoptosis in ejaculated human spermatozoa. *Fertil. Steril.*, 94(5), 1915–1917.
- Esrefoglu, M., Gul, M., Ates, B., & Selimoglu, M. A. (2006). Ultrastructural clues for the protective effect of melatonin against oxidative damage in cerulein-induced pancreatitis. *J. Pineal. Res.*, 40(1), 92–97.
- Esrefoglu, M., Gul, M., Emre, M. H., Polat, A., & Selimoglu, M. A. (2005). Protective effect of low dose of melatonin against cholestatic oxidative stress after common bile duct ligation in rats. *World J. Gastroenterol.*, 11(13), 1951–1956.
- Evans, J. W., Taylor, Y. C., & Brown, J. M. (1984). The role of glutathione and DNA strand break repair in determining the shoulder of the radiation survival curve. *Br. J. Cancer Suppl.*, 6, 49–53.
- Fagundes, D. S., Gonzalo, S., Arruebo, M. P., Plaza, M. A., & Murillo, M. D. (2010). Melatonin and Trolox ameliorate duodenal LPS-induced disturbances and oxidative stress. *Dig. Liver Dis.*, 42(1), 40–44.
- Feher, J., Kovacs, B., Kovacs, I., Schveoller, M., Papale, A., & Balacco Gabrieli, C. (2005). Improvement of visual functions and fundus alterations in early age-related macular degeneration treated with a combination of acetyl-L-carnitine, n-3 fatty acids, and coenzyme Q10. *Ophthalmologica*, 219(3), 154–166.
- Feher, J., Papale, A., Mannino, G., Gualdi, L., & Balacco Gabrieli, C. (2003). Mitotropic compounds for the treatment of age-related macular degeneration: The metabolic approach and a pilot study. *Ophthalmologica*, 217(5), 351–357.
- Feng, Z., Liu, Z., Li, X., Jia, H., Sun, L., Tian, C., Jia, L., et al. (2010). Alpha-tocopherol is an effective phase II enzyme inducer: Protective effects on acrolein-induced oxidative stress and mitochondrial dysfunction in human retinal pigment epithelial cells. *J. Nutr. Biochem.*, 21(12), 1222–1231.
- Ferguson, J., & Johnson, B. E. (1990). Ciprofloxacin-induced photosensitivity: In vitro and in vivo studies. *Br. J. Dermatol.*, 123(1), 9–20.
- Ferguson, J., & Johnson, B. E. (1993). Clinical and laboratory studies of the photosensitizing potential of norfloxacin, a 4-quinolone broad-spectrum antibiotic. *Br. J. Dermatol.*, 128(3), 285–295.
- Fernie, K. J., Bird, D. M., & Petitclerc, D. (1999). Effects of electromagnetic fields on photophasic circulating melatonin levels in American kestrels. *Environ. Health Perspect.*, 107(11), 901–904.
- Ferrer, I. (2009). Altered mitochondria, energy metabolism, voltage-dependent anion channel, and lipid rafts converge to exhaust neurons in Alzheimer's disease. *J. Bioenerg. Biomembr.*, 41(5), 425–431.

- Fetoni, A. R., De Bartolo, P., Eramo, S. L. M., Rolesi, R., Paciello, F., Bergamini, C., Fato, R., et al. (2013). Noise-induced hearing loss (NIHL) as a target of oxidative stress-mediated damage: Cochlear and cortical responses after an increase in antioxidant defense. *Journal of Neuroscience*, 33(9), 4011–4023.
- Filomeni, G., Cardaci, S., Da Costa Ferreira, A. M., Rotilio, G., & Ciriolo, M. R. (2011). Metabolic oxidative stress elicited by the copper(II) complex [Cu(isaepy)2] triggers apoptosis in SH-SY5Y cells through the induction of the AMP-activated protein kinase/p38MAPK/p53 signalling axis: Evidence for a combined use with 3-bromopyruvate in neuroblastoma treatment. *Biochem. J.*, 437(3), 443–453.
- Finnie, J. W., Blumbergs, P. C., Cai, Z., Manavis, J., & Kuchel, T. R. (2006). Effect of mobile telephony on blood-brain barrier permeability in the fetal mouse brain. *Pathology*, 38(1), 63–65.
- Finnie, J. W., Blumbergs, P. C., Manavis, J., Utteridge, T. D., Gebiski, V., Davies, R. A., Vernon-Roberts, B., et al. (2002). Effect of long-term mobile communication microwave exposure on vascular permeability in mouse brain. *Pathology*, 34(4), 344–347.
- Finsterer, J. (2008). Cognitive decline as a manifestation of mitochondrial disorders (mitochondrial dementia). *J. Neurol. Sci.*, 272(1–2), 20–33.
- Fiorini, A., Koudriavtseva, T., Bucaj, E., Coccia, R., Foppoli, C., Giorgi, A., Schinina, M. E., et al. (2013). Involvement of oxidative stress in occurrence of relapses in multiple sclerosis: The spectrum of oxidatively modified serum proteins detected by proteomics and redox proteomics analysis. *PLoS One*, 8(6), e65184.
- Foster, S. (2017). *Health exemption for firefighters sends a message to the world*. <http://betweenrockandhardplace.wordpress.com>
- France-Lanord, V., Brugg, B., Michel, P. P., Agid, Y., & Ruberg, M. (1997). Mitochondrial free radical signal in ceramide-dependent apoptosis: A putative mechanism for neuronal death in Parkinson's disease. *J. Neurochem.*, 69(4), 1612–1621.
- Franke, H., Ringelstein, E. B., & Stogbauer, F. (2005). Electromagnetic fields (GSM 1800) do not alter blood-brain barrier permeability to sucrose in models in vitro with high barrier tightness. *Bioelectromagnetics*, 26(7), 529–535.
- Franke, H., Streckert, J., Bitz, A., Goeke, J., Hansen, V., Ringelstein, E. B., Nattkamper, W., et al. (2005). Effects of universal mobile telecommunications system (UMTS) electromagnetic fields on the blood-brain barrier in vitro. *Radiat. Res.*, 164(3), 258–269.
- Freeman, L. R., & Keller, J. N. (2012). Oxidative stress and cerebral endothelial cells: Regulation of the blood-brain-barrier and antioxidant based interventions. *Biochim. Biophys. Acta*, 1822(5), 822–829.
- Frei, M., Jauchem, J., & Heinmets, F. (1988). Physiological effects of 2.8 GHz radio-frequency radiation: A comparison of pulsed and continuous-wave radiation. *J. Microw. Power Electromagn. Energy*, 23(2), 85–93.
- Fry, A. H. (1961). Auditory system response to radio frequency energy. *Aerosp. Med.*, 32, 1140–1142.
- Friedman, L. S., & Richter, E. D. (2004). Relationship between conflicts of interest and research results. *J. Gen. Intern. Med.*, 19(1), 51–56.
- Fritze, K., Sommer, C., Schmitz, B., Mies, G., Hossmann, K. A., Kiessling, M., & Wiessner, C. (1997). Effect of global system for mobile communication (GSM)

- microwave exposure on blood-brain barrier permeability in rat. *Acta Neuropathol.*, 94(5), 465–470.
- Frye, R. E., Delatorre, R., Taylor, H., Slattery, J., Melnyk, S., Chowdhury, N., & James, R. J. (2013). Redox metabolism abnormalities in autistic children associated with mitochondrial disease. *Transl. Psychiatry*, 3, e273.
- Frye, R. E., Melnyk, S., & Macfabe, D. F. (2013). Unique acyl-carnitine profiles are potential biomarkers for acquired mitochondrial disease in autism spectrum disorder. *Transl. Psychiatry*, 3, e220.
- Frye, R. E., & Rossignol, D. A. (2011). Mitochondrial dysfunction can connect the diverse medical symptoms associated with autism spectrum disorders. *Pediatr. Res.*, 69(5 Pt. 2), 41R–47R.
- Fuchs, J., & Packer, L. (1993). Vitamin E in dermatological therapy. In L. Packer & J. Fuchs (Eds.), *Vitamin E in health and disease*. New York: Dekker.
- Fuentes-Broto, L., Miana-Mena, F. J., Piedrafita, E., Berzosa, C., Martinez-Ballarín, E., García-Gil, F. A., Reiter, R. J., et al. (2010). Melatonin protects against taurolithocholic-induced oxidative stress in rat liver. *J. Cell. Biochem.*, 110(5), 1219–1225.
- Fujita, H., & Matsuo, I. (1994). In vitro phototoxic activities of new quinolone antibacterial agents: Lipid peroxidative potentials. *Photodermatol. Photoimmunol. Photomed.*, 10(5), 202–205.
- Fukui, K., Omoi, N. O., Hayasaka, T., Shinnkai, T., Suzuki, S., Abe, K., & Urano, S. (2002). Cognitive impairment of rats caused by oxidative stress and aging, and its prevention by vitamin E. *Ann. NY Acad. Sci.*, 959, 275–284.
- Gajski, G., & Garaj-Vrhovac, V. (2009). Radioprotective effects of honeybee venom (*Apis mellifera*) against 915-MHz microwave radiation-induced DNA damage in Wistar rat lymphocytes: In vitro study. *Int. J. Toxicol.*, 28(2), 88–98.
- Gammack, J. K. (2008). Light therapy for insomnia in older adults. *Clin. Geriatr. Med.*, 24(1), 139–149, viii.
- Gao, X. H., Hu, H. R., Ma, X. L., Chen, J., & Zhang, G. H. (2016). [Cell phone electromagnetic radiation damages the testicular ultrastructure of male rats]. *Zhonghua Nan Ke Xue*, 22(6), 491–495.
- García-Rubio, L., Matas, P., & Miguez, M. P. (2005). Protective effect of melatonin on paraquat-induced cytotoxicity in isolated rat hepatocytes. *Hum. Exp. Toxicol.*, 24(9), 475–480.
- Gasche, Y., Copin, J. C., Sugawara, T., Fujimura, M., & Chan, P. H. (2001). Matrix metalloproteinase inhibition prevents oxidative stress-associated blood-brain barrier disruption after transient focal cerebral ischemia. *J. Cereb. Blood Flow Metab.*, 21(12), 1393–1400.
- Gazi, S., Altun, A., & Erdogan, O. (2006). Contrast-induced nephropathy: Preventive and protective effects of melatonin. *J. Pineal Res.*, 41(1), 53–57.
- Gearan, A. (2017). State Department reports new instances of American diplomats harmed in Cuba. *Washington Post*, September 1.
- Gelderman, K. A., Hultqvist, M., Olsson, L. M., Bauer, K., Pizzolla, A., Olofsson, P., & Holmdel, R., et al. (2007). Rheumatoid arthritis: The role of reactive oxygen species in disease development and therapeutic strategies. *Antioxid. Redox. Signal.*, 9(10), 1541–1567.
- Genuis, S. J., & Lipp, C. T. (2012). Electromagnetic hypersensitivity: Fact or fiction? *Sci. Total Environ.*, 414, 103–112.

- George, P., Das, J., Pawar, B., & Badyal, D. (2008). Gatifloxacin-induced rhabdomyolysis. *J. Postgrad. Med.*, 54(3), 233–234.
- Gerster, H. (1999). High-dose vitamin C: A risk for persons with high iron stores? *Int. J. Vitam. Nutr. Res.*, 69(2), 67–82.
- Gilgun-Sherki, Y., Melamed, E., & Offen, D. (2004). The role of oxidative stress in the pathogenesis of multiple sclerosis: The need for effective antioxidant therapy. *J. Neurol.*, 251(3), 261–268.
- Glaser, Z. R. (1972). *Bibliography of reported biological phenomena ("effects") and clinical manifestations attributed to microwave and radiofrequency radiation* (Research Report, 2nd Printing, with Revisions, Corrections, and Additions. 20 No. AD750271 MF12.524.015-0004B. Supersedes AD No 734391). Bethesda, MD: Naval Medical Research Institute.
- Glickman, G., Byrne, B., Pineda, C., Hauck, W. W., & Brainard, G. C. (2006). Light therapy for seasonal affective disorder with blue narrow-band light-emitting diodes (LEDs). *Biol. Psychiatry*, 59(6), 502–507.
- Golden, T., & Rotella, S. (2018). The sound and the fury: Inside the mystery of the Havana embassy. *ProPublica*, February 14.
- Goldsmith, J. R. (1995). Where the trail leads . . . Ethical problems arising when the trail of professional work lead to evidence of cover-up of serious risk and misrepresentation of scientific judgement concerning human exposures to radar. *Eubios. J. Asian Int. Bioeth.*, 5, 92–94.
- Golomb, B. A. (2008). *Conflict of interest in medicine*. Sponsored by the Science Network, Salk Institute. La Jolla, CA. October 5. <http://thesciencenetwork.org/programs/beyond-belief-candles-in-the-dark/beatrice-golomb>
- Golomb, B. A. (2014). Statins and activity: Proceed with caution. *JAMA Intern Med*, 174(8), 1270–1272.
- Golomb, B. A. (2015a). Electrosensitivity: A “current” and future problem. Presented at Cell Phones and Wireless Technologies: Should Safety Guidelines Be Strengthened to Protect Adults, Children and Vulnerable Populations? Commonwealth Club, San Francisco, June 22.
- Golomb, B. A. (2015b). Misinterpretation of trial evidence on statin adverse effects may harm patients. *Eur. J. Prev. Cardiol*, 22(4), 492–493.
- Golomb, B. A. (2015c). Psychogenic illness. In John Brockman (Ed.), *This idea must die: Scientific theories that are blocking progress* (pp. 511–514). New York: Harper.
- Golomb, B. A. (2018). Effect modification. In J. Brockman (Ed.), *This idea is brilliant: Lost, overlooked, and underappreciated scientific concepts everyone should know* (pp. 440–443). New York: Harper.
- Golomb, B. A., Allison, M., Koperski, S., Koslik, H. J., Devaraj, S., & Ritchie, J. B. (2014). Coenzyme Q10 benefits symptoms in Gulf War veterans: Results of a randomized double-blind study. *Neural Comput.*, 26(11), 2594–2651.
- Golomb, B. A., & Evans, M. A. (2008). Statin adverse effects: A review of the literature and evidence for a mitochondrial mechanism. *Am. J. Cardiovasc. Drugs*, 8(6), 373–418.
- Golomb, B. A., & Koperski, S. (2013). Who becomes weak on statins? Effect modification exposed in a RCT by risk factor compounding. *Circulation*, 127, AP072.
- Golomb, B. A., Koslik, H. J., & Redd, A. J. (2015). Fluoroquinolone-induced serious, persistent, multisymptom adverse effects. *BMJ Case Rep.*, 2015.

- Goswami, S., & Haldar, C. (2014a). Melatonin improves ultraviolet B-induced oxidative damage and inflammatory conditions in cutaneous tissue of a diurnal Indian palm squirrel *Funambulus pennanti*. *Br. J. Dermatol.*, 171(5), 1147–1155.
- Goswami, S., & Haldar, C. (2014b). UVB irradiation severely induces systemic tissue injury by augmenting oxidative load in a tropical rodent: efficacy of melatonin as an antioxidant. *J. Photochem. Photobiol. B*, 141, 84–92.
- Goswami, S., Sharma, S., & Haldar, C. (2013). The oxidative damages caused by ultraviolet radiation type C (UVC) to a tropical rodent *Funambulus pennanti*: Role of melatonin. *J. Photochem. Photobiol. B*, 125, 19–25.
- Goto, Y., Koga, Y., Horai, S., & Nonaka, I. (1990). Chronic progressive external ophthalmoplegia: A correlative study of mitochondrial DNA deletions and their phenotypic expression in muscle biopsies. *J. Neurol. Sci.*, 100(1–2), 63–69.
- Graham, D. J., Staffa, J. A., Shatin, D., Andrade, S. E., Schech, S. D., La Grenade, L., Gurwitz, J. H., et al. (2004). Incidence of hospitalized rhabdomyolysis in patients treated with lipid-lowering drugs. *JAMA*, 292(21), 2585–2590.
- Granowitz, E. V. (1989). Photosensitivity rash in a patient being treated with ciprofloxacin. *J. Infect. Dis.*, 160(5), 910–911.
- Griefahn, B., Kunemund, C., Blaszkewicz, M., Lerchl, A., & Degen, G. H. (2002). Effects of electromagnetic radiation (bright light, extremely low-frequency magnetic fields, infrared radiation) on the circadian rhythm of melatonin synthesis, rectal temperature, and heart rate. *Ind. Health*, 40(4), 320–327.
- Gruber, J., Schaffer, S., & Halliwell, B. (2008). The mitochondrial free radical theory of ageing—where do we stand? *Front. Biosci.*, 13, 6554–6579.
- Gruber, M. J., Palmquist, E., & Nordin, S. (2018). Characteristics of perceived electromagnetic hypersensitivity in the general population. *Scand J. Psychol.*, 59, 422–427.
- Gul, A., Rahman, M. A., Hasnain, S. N., Salim, A., & Simjee, S. U. (2008). Could oxidative stress associate with age products in cataractogenesis? *Curr. Eye Res.*, 33(8), 669–675.
- Guney, M., Ozguner, F., Oral, B., Karahan, N., & Mungan, T. (2007). 900 MHz radiofrequency-induced histopathologic changes and oxidative stress in rat endometrium: Protection by vitamins E and C. *Toxicol. Ind. Health*, 23(7), 411–420.
- Guney, Y., Hicsonmez, A., Uluoglu, C., Guney, H. Z., Ozel Turkcu, U., Take, G., Yucel, B., et al. (2007). Melatonin prevents inflammation and oxidative stress caused by abdominopelvic and total body irradiation of rat small intestine. *Braz. J. Med. Biol. Res.*, 40(10), 1305–1314.
- Gupta, A., Guron, N., Harris, M., & Bell, R. (2012). Levofloxacin-induced rhabdomyolysis in a hemodialysis patient. *Hemodial Int.*, 16(1), 101–103.
- Gupta, S. K., Mesharam, M. K., & Krishnamurthy, S. (2018). Electromagnetic radiation 2450 MHz exposure causes cognition deficit with mitochondrial dysfunction and activation of intrinsic pathway of apoptosis in rats. *Journal of Biosciences*, 43(2), 263–276.
- Gupta, Y. K., Gupta, M., & Kohli, K. (2003). Neuroprotective role of melatonin in oxidative stress vulnerable brain. *Indian J. Physiol. Pharmacol.*, 47(4), 373–386.
- Gurler, H. S., Bilgici, B., Akar, A. K., Tomak, L., & Bedir, A. (2014). Increased DNA oxidation (8-OHdG) and protein oxidation (AOPP) by low level electromagnetic field (2.45 GHz) in rat brain and protective effect of garlic. *Int. J. Radiat. Biol.*, 90(10), 892–896.

- Gwertzman, B. (1976). Moscow rays linked to U.S. bugging. *New York Times*, February 26.
- Hagstrom, M., Auranen, J., & Ekman, R. (2013). Electromagnetic hypersensitive Finns: Symptoms, perceived sources and treatments, a questionnaire study. *Pathophysiology*, 20(2), 117–122.
- Halgamuge, M. N. (2013). Critical time delay of the pineal melatonin rhythm in humans due to weak electromagnetic exposure. *Indian J. Biochem. Biophys.*, 50(4), 259–265.
- Halliday, G. M. (2005). Inflammation, gene mutation and photoimmunosuppression in response to UVR-induced oxidative damage contributes to photocarcinogenesis. *Mutat. Res.*, 571(1–2), 107–120.
- Halteman, E. (2011). *Wireless utility meter safety impacts survey: Final results summary*. September 13. <http://emfsafetynetwork.org/wp-content/uploads/2011/09/Wireless-Utility-Meter-Safety-Impacts-Survey-Results-Final.pdf>
- Hanada, K., Gange, R. W., & Connor, M. J. (1990). Effect of glutathione depletion on sunburn cell formation in the hairless mouse. *Journal of Investigative Dermatology*, 96(6), 838–840.
- Haney, D. Q. (1999). Cholesterol drug is very secret weapon. *San Diego Union Tribune*, August 23, p. E2.
- Haorah, J., Knipe, B., Leibhart, J., Ghorpade, A., & Persidsky, Y. (2005). Alcohol-induced oxidative stress in brain endothelial cells causes blood-brain barrier dysfunction. *J. Leukoc. Biol.*, 78(6), 1223–1232.
- Haorah, J., Ramirez, S. H., Schall, K., Smith, D., Pandya, R., & Persidsky, Y. (2007). Oxidative stress activates protein tyrosine kinase and matrix metalloproteinases leading to blood-brain barrier dysfunction. *J. Neurochem.*, 101(2), 566–576.
- Hara, M., Yoshida, M., Nishijima, H., Yokosuka, M., Iigo, M., Ohtani-Kaneko, R., Shimeda, A., et al. (2001). Melatonin, a pineal secretory product with antioxidant properties, protects against cisplatin-induced nephrotoxicity in rats. *J. Pineal. Res.*, 30(3), 129–138.
- Hardell, L. (2017). World Health Organization, radiofrequency radiation and health: A hard nut to crack (Review). *International Journal of Oncology*, June 21.
- Hardell, L., & Carlberg, M. (2013). Using the Hill viewpoints from 1965 for evaluating strengths of evidence of the risk for brain tumors associated with use of mobile and cordless phones. *Rev. Environ. Health*, 28(2–3), 97–106.
- Hardell, L., & Carlberg, M. (2015). Mobile phone and cordless phone use and the risk for glioma: Analysis of pooled case-control studies in Sweden, 1997–2003 and 2007–2009. *Pathophysiology*, 22(1), 1–13.
- Hardell, L., Carlberg, M., & Hansson Mild, K. (2011). Pooled analysis of case-control studies on malignant brain tumours and the use of mobile and cordless phones including living and deceased subjects. *Int. J. Oncol.*, 38(5), 1465–1474.
- Hardell, L., Carlberg, M., Soderqvist, F., Hardell, K., Bjornfoth, H., van Bavel, B., & Lindstrom, D., et al. (2008). Increased concentrations of certain persistent organic pollutants in subjects with self-reported electromagnetic hypersensitivity—a pilot study. *Electromagn Biol. Med.*, 27(2), 197–203.
- Hardell, L., Carlberg, M., Soderqvist, F., & Mild, K. H. (2013). Pooled analysis of case-control studies on acoustic neuroma diagnosed 1997–2003 and 2007–2009 and use of mobile and cordless phones. *Int. J. Oncol.*, 43(4), 1036–1044.

- Harkinson, J. (2017). This former techie owes his fortune to electronic devices. Now he thinks they're dangerous. *Mother Jones*, January 28.
- Harris, G. (2010). Caustic government report deals blow to diabetes drug. *New York Times*, July 9.
- Harris, G. (2017a). 16 Americans sickened after attack on embassy staff in Havana. *New York Times*, August 24.
- Harris, G. (2017b). Tillerson says U.S. may close Cuba embassy over mystery ailments. *New York Times*, September 17.
- Harris, G. (2018a). Pompeo says mysterious sickness among diplomats in Cuba has spread to China. *New York Times*, May 23.
- Harris, G. (2018b). U.S. to open formal inquiry on Americans sickened in Cuba. *New York Times*, January 9.
- Harris, G. (2018c). U.S. to open inquiry over 24 Americans sickened in Cuba. *New York Times*, January 10.
- Harris, G., & Goldman, A. (2017a). Illnesses at U.S. embassy in Havana prompt evacuation of more diplomats. *New York Times*, September 29.
- Harris, G., & Goldman, A. (2017b). U.S. pares embassy in Cuba over mystery attack. *New York Times*, September 30.
- Hassig, M., Jud, F., & Spiess, B. (2012). [Increased occurrence of nuclear cataract in the calf after erection of a mobile phone base station]. *Schweiz. Arch. Tierheilkd*, 154(2), 82–86.
- Havas, M., Marrongelle, J., Pollner, B., Kelley, E., Rees, C. R. G., & Tully, L. (2010). Provocation study using heart rate variability shows microwave radiation from 2.4 GHz cordless phone affects autonomic nervous system. *Eur. J. Oncol. Library*, 5, 273–300.
- Hayano, J. (1990). Decreased magnitude of heart rate spectral components in coronary artery disease. *Circulation*, 81, 1217–1224.
- Hensinger, P., & Wilke, I. (2016). Wireless communication technologies: New study findings confirm risks of nonionizing radiation. (Trans. Katharina Gustavs). *Umwelt-medizin-gesellschaft*, March. www.mobilfunkstudien.org
- Herbert, M. R., & Sage, C. (2013a). Autism and EMF? Plausibility of a pathophysiological link—part I. *Pathophysiology*, 20(3), 191–209.
- Herbert, M. R., & Sage, C. (2013b). Autism and EMF? Plausibility of a pathophysiological link—part II. *Pathophysiology*, 20(3), 211–234.
- Heres, S., Davis, J., Maino, K., Jetzinger, E., Kissling, W., & Leucht, S. (2006). Why olanzapine beats risperidone, risperidone beats quetiapine, and quetiapine beats olanzapine: An exploratory analysis of head-to-head comparison studies of second-generation antipsychotics. *Am. J. Psychiatry*, 163(2), 185–194.
- Herrera, F., Sainz, R. M., Mayo, J. C., Martin, V., Antolin, I., & Rodriguez, C. (2001). Glutamate induces oxidative stress not mediated by glutamate receptors or cystine transporters: Protective effect of melatonin and other antioxidants. *J. Pineal Res.*, 31(4), 356–362.
- Heuser, G., & Heuser, S. A. (2017). Functional brain MRI in patients complaining of electrohypersensitivity after long term exposure to electromagnetic fields. *Rev. Environ. Health*, July 5.
- Hillert, L., Berglind, N., Arnetz, B. B., & Bellander, T. (2002). Prevalence of self-reported hypersensitivity to electric or magnetic fields in a population-based questionnaire survey. *Scand. J. Work Environ. Health*, 28(1), 33–41.

- Hiramoto, K., Ohkawa, T., Oikawa, N., & Kikugawa, K. (2003). Is nitric oxide (NO) an antioxidant or a prooxidant for lipid peroxidation? *Chem. Pharm. Bull. (Tokyo)*, 51(9), 1046–1050.
- Hocking, B., & Gordon, I. (2003). Decreased survival for childhood leukemia in proximity to television towers. *Arch. Environ. Health*, 58(9), 560–564.
- Hodgkiss, R. J., Stratford, M. R., & Wafra, R. R. (1989). The effect of alpha-tocopherol and alpha-tocopheryl quinone on the radiosensitivity of thiol-depleted mammalian cells. *Int. J. Radiat. Oncol. Biol. Phys.*, 16(5), 1297–1300.
- Hoffman, K. B., Kraus, C., Dimbil, M., & Golomb, B. A. (2012). A survey of the FDA's AERS database regarding muscle and tendon adverse events linked to the statin drug class. *PLoS One*, 7(8), e42866.
- Holmboe, G., & Johansson, O. (2005). Symptombeskrivning samt förekomst av IgE och positiv Phadiatop Combi hos personer med funktionsnedsättningen elöverkänslighet. [Description of symptoms as well as occurrence of IgE and positive *Phadiatop combi* in persons with the physical impairment electrohypersensitivity. *Medicinsk Access*, 1, 58–63.
- Holt, J. A. (1995). Some characteristics of the glutathione cycle revealed by ionising and non-ionising electromagnetic radiation. *Med. Hypotheses*, 45(4), 345–368.
- Hoshino, S., Tamaoka, A., Ohkoshi, N., Shoji, S., & Goto, Y. (1997). [A case of mitochondrial encephalomyopathy showing ophthalmoplegia, diabetes mellitus and hearing loss associated with the A3243G mutation of mitochondrial DNA]. *Rinsho Shinkeigaku*, 37(4), 326–330.
- Houston, B. J., Nixon, B., King, B. V., De Iuliis, G. N., & Aitken, R. J. (2016). The effects of radiofrequency electromagnetic radiation on sperm function. *Reproduction*, 152(6), R263–R276.
- Hsiao, S. H., Chang, C. M., Tsao, C. J., Lee, Y. Y., Hsu, M. Y., & Wu, T. J. (2005). Acute rhabdomyolysis associated with ofloxacin/levofloxacin therapy. *Ann. Pharmacother.*, 39(1), 146–149.
- Hu, M. L., Chen, Y. K., & Lin, Y. F. (1995). The antioxidant and prooxidant activity of some B vitamins and vitamin-like compounds. *Chem. Biol. Interact.*, 97(1), 63–73.
- Hu, S., Yin, S., Jiang, X., Huang, D., & Shen, G. (2009). Melatonin protects against alcoholic liver injury by attenuating oxidative stress, inflammatory response, and apoptosis. *Eur. J. Pharmacol.*, 616(1–3), 287–292.
- Hurst, R. D., Heales, S. J., Dobbie, M. S., Barker, J. E., & Clark, J. B. (1998). Decreased endothelial cell glutathione and increased sensitivity to oxidative stress in an in vitro blood-brain barrier model system. *Brain Res.*, 802(1–2), 232–240.
- Husain, K., Whitworth, C., Somani, S. M., & Rybak, L. P. (2001). Carboplatin-induced oxidative stress in rat cochlea. *Hear. Res.*, 159(1–2), 14–22.
- Huss, A., Egger, M., Hug, K., Huwiler-Müntener, K., & Rösli, M. (2007). Source of funding and results of studies of health effects of mobile phone use: Systematic review of experimental studies. *Environ Health Perspect.*, 115, 1–4.
- Hutter, H. P., Moshhammer, H., Wallner, P., & Kundi, M. (2006). Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations. *Occup. Environ. Med.*, 63(5), 307–313.
- Hyman, B. N., Patten, B. M., & Dodson, R. F. (1977). Mitochondrial abnormalities in progressive external ophthalmoplegia. *Am. J. Ophthalmol.*, 83(3), 362–371.

- Iborra, A., Palacio, J. R., & Martinez, P. (2005). Oxidative stress and autoimmune response in the infertile woman. *Chem. Immunol. Allergy*, 88, 150–162.
- IDEA (Irish Doctors' Environmental Association). (2004). *IDEA position on electromagnetic radiation*. <http://www.ideaireland.org/emr.htm>
- Ikeda-Douglas, C. J., Zicker, S. C., Estrada, J., Jewell, D. E., & Milgram, N. W. (2004). Prior experience, antioxidants, and mitochondrial cofactors improve cognitive function in aged beagles. *Vet. Ther.*, 5(1), 5–16.
- Ikeda, T., Choi, B. H., Yee, S., Murata, Y., & Quilligan, E. J. (1999). Oxidative stress, brain white matter damage and intrauterine asphyxia in fetal lambs. *Int. J. Dev. Neurosci.*, 17(1), 1–14.
- Ilhan, A., Gurel, A., Armutcu, F., Kamisli, S., Iraz, M., Akyol, O., & Ozen, S., et al. (2004). Ginkgo biloba prevents mobile phone-induced oxidative stress in rat brain. *Clin. Chim. Acta*, 340(1–2), 153–162.
- Imaida, K., Hagiwara, A., Yoshino, H., Tamano, S., Sano, M., Futakuchi, M., Ogawa, K., et al. (2000). Inhibitory effects of low doses of melatonin on induction of pre-neoplastic liver lesions in a medium-term liver bioassay in F344 rats: Relation to the influence of electromagnetic near field exposure. *Cancer Lett.*, 155(1), 105–114.
- Indik, J. H., Goldman, S., & Gaballa, M. A. (2001). Oxidative stress contributes to vascular endothelial dysfunction in heart failure. *Am. J. Physiol. Heart Circ. Physiol.*, 281(4), H1767–H1770.
- Ingalls, C. E. (1967). Sensation of hearing in electromagnetic fields. *NY State J. Med.*, 67, 2992–2997.
- Insel, K. C., Moore, I. M., Vidrine, A. N., & Montgomery, D. W. (2012). Biomarkers for cognitive aging, part II: Oxidative stress, cognitive assessments, and medication adherence. *Biol. Res. Nurs.*, 14(2), 133–138.
- International Association of Fire Fighters Division of Occupational Health Safety and Medicine. (2006). Position on the health effects from radio frequency/microwave (RF/MW) radiation in fire department facilities from base stations for antennas and towers for the conduction of cell phone transmissions. <http://www.iaff.org/hs/resi/celltowerfinal.htm>
- Irmak, M. K., Fadillioglu, E., Gulec, M., Erdogan, H., Yagmurca, M., & Akyol, O. (2002). Effects of electromagnetic radiation from a cellular telephone on the oxidant and antioxidant levels in rabbits. *Cell Biochem. Funct.*, 20(4), 279–283.
- Ishikawa, C., Ozaki, H., Nakajima, T., Ishii, T., Kanai, S., Anjo, S., Shirai, K., et al. (2004). A frameshift variant of CYP2C8 was identified in a patient who suffered from rhabdomyolysis after administration of cerivastatin. *J. Hum. Genet.*, 49(10), 582–585.
- Iuchi, Y., Kibe, N., Tsunoda, S., Suzuki, S., Mikami, T., Okada, F., Uchida, K., et al. (2010). Implication of oxidative stress as a cause of autoimmune hemolytic anemia in NZB mice. *Free Radic. Biol. Med.*, 48(7), 935–944.
- Ivancsits, S., Diem, E., Pilger, A., Rudiger, H. W., & Jahn, O. (2002). Induction of DNA strand breaks by intermittent exposure to extremely-low-frequency electromagnetic fields in human diploid fibroblasts. *Mutat. Res.*, 519(1–2), 1–13.
- Jaffe, A., & Bush, A. (1999). If you can't stand the rash, get out of the kitchen: An unusual adverse reaction to ciprofloxacin. *Pediatr. Pulmonol.*, 28(6), 449–450.

- Jagetia, G. C., & Baliga, M. S. (2003). Treatment of mice with a herbal preparation (Mentat) protects against radiation-induced mortality. *Phytother. Res.*, 17(8), 876–881.
- Jagetia, G. C., Venkatesh, P., & Baliga, M. S. (2004). Fruit extract of *Aegle marmelos* protects mice against radiation-induced lethality. *Integr. Cancer Ther.*, 3(4), 323–332.
- Jain, S., Agarwal, J., Laskar, S., Gupta, T., & Shrivastava, S. (2008). Radiation recall dermatitis with gatifloxacin: A review of literature. *J. Med. Imaging Radiat. Oncol.*, 52(2), 191–193.
- Jang, S. S., Kim, H. G., Lee, J. S., Han, J. M., Park, H. J., Huh, G. J., & Son, C. G., et al. (2013). Melatonin reduces X-ray radiation-induced lung injury in mice by modulating oxidative stress and cytokine expression. *Int. J. Radiat. Biol.*, 89(2), 97–105.
- Jarasuniene, D., & Simaitis, A. (2003). [Oxidative stress and endothelial dysfunction]. *Medicina (Kaunas)*, 39(12), 1151–1157.
- Javaheri, M., Khurana, R. N., O'Hearn T. M., Lai, M. M., & Sadun, A. A. (2007). Linezolid-induced optic neuropathy: A mitochondrial disorder? *Br. J. Ophthalmol.*, 91(1), 111–115.
- Jensen, G. L., & Meister, A. (1983). Radioprotection of human lymphoid cells by exogenously supplied glutathione is mediated by gamma-glutamyl transpeptidase. *Proc. Natl. Acad. Sci. USA*, 80(15), 4714–4717.
- Jeyakumar, A., Williamson, M. E., Brickman, T. M., Krakovitz, P., & Parikh, S. (2009). Otolaryngologic manifestations of mitochondrial cytopathies. *Am. J. Otolaryngol.*, 30(3), 162–165.
- Jindal, M., Garg, G. R., Mediratta, P. K., & Fahim, M. (2011). Protective role of melatonin in myocardial oxidative damage induced by mercury in murine model. *Hum. Exp. Toxicol.*, 30(10), 1489–1500.
- Johansson, O. (2006). Electrohypersensitivity: State-of-the-art of a functional impairment. *Electromagn. Biol. Med.*, 25(4), 245–258.
- Johansson, O. (2015). Electrohypersensitivity: A functional impairment due to an inaccessible environment. *Rev. Environ Health*, 30(4), 311–321.
- Johnson, J. (n.d.). Protect your family from EMF Pollution. Retrieved August 20, 2018, from <https://www.emfanalysis.com/about/>
- Johnson Liakouris, A. G. (1998). Radiofrequency (RF) sickness in the Lilienfeld study: An effect of modulated microwaves? *Arch. Environ. Health*, 53, 236–238.
- Kacmaz, A., User, E. Y., Sehirli, A. O., Tilki, M., Ozkan, S., & Sener, G. (2005). Protective effect of melatonin against ischemia/reperfusion-induced oxidative remote organ injury in the rat. *Surg. Today*, 35(9), 744–750.
- Kalluri, R., Cantley, L. G., Kerjaschki, D., & Neilson, E. G. (2000). Reactive oxygen species expose cryptic epitopes associated with autoimmune goodpasture syndrome. *J. Biol. Chem.*, 275(26), 20027–20032.
- Kao, K. P. (1994). Mitochondrial disease with chronic progressive external ophthalmoplegia: Clinical analysis of 19 cases. *Zhonghua Yi Xue Za Zhi (Taipei)*, 53(2), 95–100.
- Karaer, I., Simsek, G., Gul, M., Bahar, L., Gurocak, S., Parlakpinar, H., & Nuransoy, A., et al. (2015). Melatonin protects inner ear against radiation damage in rats. *Laryngoscope*, 125, E345–E349.

- Karbownik, M., & Reiter, R. J. (2002). Melatonin protects against oxidative stress caused by delta-aminolevulinic acid: Implications for cancer reduction. *Cancer Invest.*, 20(2), 276–286.
- Karslioglu, I., Ertekin, M. V., Taysi, S., Kocer, I., Sezen, O., Gepdiremen, A., Koe, M., et al. (2005). Radioprotective effects of melatonin on radiation-induced cataract. *J. Radiat. Res. (Tokyo)*, 46(2), 277–282.
- Kato, Y., & Johansson, O. (2012). Reported functional impairments of electrohypersensitive Japanese: A questionnaire survey. *Pathophysiology*, 19(2), 95–100.
- Katsu, M., Niizuma, K., Yoshioka, H., Okami, N., Sakata, H., & Chan, P. H. (2010). Hemoglobin-induced oxidative stress contributes to matrix metalloproteinase activation and blood-brain barrier dysfunction in vivo. *J. Cereb. Blood Flow Metab.*, 30(12), 1939–1950.
- Keaney, J. J., Gaziano, J., Xu, A., Frei, B., Curran-Celentano, J., Shwaery, G., Loscalzo, J., et al. (1994). Low-dose alpha-tocopherol improves and high-dose alpha-tocopherol worsens endothelial vasodilator function in cholesterol-fed rabbits. *Journal of Clinical Investigation*, 93(2), 844–845.
- Kerman, M., Cirak, B., Ozguner, M. F., Dagtekin, A., Sutcu, R., Altuntas, I., & Delibas, N., et al. (2005). Does melatonin protect or treat brain damage from traumatic oxidative stress? *Exp. Brain Res.*, 163(3), 406–410.
- Khammassi, N., Abdelhedi, H., Mohsen, D., Ben Sassi, M., & Cherif, O. (2012). [Rhabdomyolysis and acute renal failure secondary to ciprofloxacin therapy]. *Therapie*, 67(1), 67–68.
- Kilic, A., Selek, S., Erel, O., & Aksoy, N. (2008). Protective effects of melatonin on oxidative-antioxidative balance and cataract formation in rats. *Ann. Ophthalmol. (Skokie)*, 40(1), 22–27.
- Kim, B. C., Shon, B. S., Ryoo, Y. W., Kim, S. P., & Lee, K. S. (2001). Melatonin reduces X-ray irradiation-induced oxidative damages in cultured human skin fibroblasts. *J. Dermatol. Sci.*, 26(3), 194–200.
- Kimura, M., Kawada, A., Kobayashi, T., Hiruma, M., & Ishibashi, A. (1996). Photosensitivity induced by fleroxacin. *Clin. Exp. Dermatol.*, 21(1), 46–47.
- King, A., Gottlieb, E., Brooks, D. G., Murphy, M. P., & Dunaief, J. L. (2004). Mitochondria-derived reactive oxygen species mediate blue light-induced death of retinal pigment epithelial cells. *Photochem. Photobiol.*, 79(5), 470–475.
- Kirkham, P. A., Caramori, G., Casolari, P., Papi, A. A., Edwards, M., Shamji, B., & Triantapyllopoulos, K., et al. (2011). Oxidative stress-induced antibodies to carbonyl-modified protein correlate with severity of chronic obstructive pulmonary disease. *Am. J. Respir. Crit. Care Med.*, 184(7), 796–802.
- Koc, M., Taysi, S., Buyukokuroglu, M. E., & Bakan, N. (2003a). Melatonin protects rat liver against irradiation-induced oxidative injury. *J. Radiat. Res.*, 44(3), 211–215.
- Koc, M., Taysi, S., Buyukokuroglu, M. E., & Bakan, N. (2003b). The effect of melatonin against oxidative damage during total-body irradiation in rats. *Radiat. Res.*, 160(2), 251–255.
- Koch, C. J., & Skov, K. A. (1994). Enhanced radiation-sensitivity by preincubation with nitroimidazoles: Effect of glutathione depletion. *Int. J. Radiat. Oncol. Biol. Phys.*, 29(2), 345–349.
- Koga, Y., & Nataliya, P. (2005). [Migraine headache and mitochondrial DNA abnormality]. *Nihon Rinsho*, 63(10), 1720–1726.

- Koillinen, H., Jaaskelainen, S., & Koski, K. (2009). [Mitochondrial disorder underlying headache symptoms]. *Duodecim.*, 125(3), 297–300.
- Kontush, A., Finckh, B., Karten, B., Kohlschutter, A., & Beisiegel, U. (1996). Antioxidant and prooxidant activity of alpha-tocopherol in human plasma and low density lipoprotein. *J. Lipid Res.*, 37(7), 1436–1448.
- Korkmaz, G. G., Uzun, H., Cakatay, U., & Aydin, S. (2012). Melatonin ameliorates oxidative damage in hyperglycemia-induced liver injury. *Clin. Invest. Med.*, 35(6), E370–E377.
- Korzets, A., Gafter, U., Dicker, D., Herman, M., & Ori, Y. (2006). Levofloxacin and rhabdomyolysis in a renal transplant patient. *Nephrol. Dial. Transplant*, 21(11), 3304–3305.
- Koslik, H. J., Hamilton, G., & Golomb, B. A. (2014). Mitochondrial dysfunction in Gulf War illness revealed by 31phosphorus magnetic resonance spectroscopy: A case-control study. *PLoS One*, 9(3), e92887.
- Kostoff, R. N., & Lau, C. G. Y. (2017). Chapter 4. Modified health effects of non-ionizing electromagnetic radiation combined with other agents reported in the biomedical literature. In C. D. Geddes (Ed.), *Microwave effects on DNA and proteins*. New York: Springer.
- Kowald, A. (2001). The mitochondrial theory of aging. *Biol. Signals Recept.*, 10(3–4), 162–175.
- Koyu, A., Ozguner, F., Yilmaz, H., Uz, E., Cesur, G., & Ozelik, N. (2009). The protective effect of caffeic acid phenethyl ester (CAPE) on oxidative stress in rat liver exposed to the 900 MHz electromagnetic field. *Toxicol. Ind. Health*, 25(6), 429–434.
- Koylu, H., Mollaoglu, H., Ozguner, F., Naziroglu, M., & Delibas, N. (2006). Melatonin modulates 900 Mhz microwave-induced lipid peroxidation changes in rat brain. *Toxicol. Ind. Health*, 22(5), 211–216.
- Kumagai, S., Jikimoto, T., & Saegusa, J. (2003). [Pathological roles of oxidative stress in autoimmune diseases]. *Rinsho Byori*, 51(2), 126–132.
- Kuruppu, D. K., & Matthews, B. R. (2013). Young-onset dementia. *Semin. Neurol.*, 33(4), 365–385.
- Lai, H., Horita, A., Chou, C. K., & Guy, A. W. (1987). Low-level microwave irradiations affect central cholinergic activity in the rat. *J. Neurochem.*, 48(1), 40–45.
- Lai, H., & Singh, N. P. (1995). Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. *Bioelectromagnetics*, 16(3), 207–210.
- Lai, H., & Singh, N. P. (1997). Melatonin and a spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand breaks in rat brain cells. *Bioelectromagnetics*, 18(6), 446–454.
- Lamech, F. (2014). Self-reporting of symptom development from exposure to radiofrequency fields of wireless smart meters in Victoria, Australia: A case series. *Altern. Ther. Health Med.*, 20(6), 28–39.
- Lan, C. T., Hsu, J. C., & Ling, E. A. (2001). Influence of sleep deprivation coupled with administration of melatonin on the ultrastructure of rat pineal gland. *Brain Res.*, 910(1–2), 1–11.
- Land, J. M., Hockaday, J. M., Hughes, J. T., & Ross, B. D. (1981). Childhood mitochondrial myopathy with ophthalmoplegia. *J. Neurol. Sci.*, 51(3), 371–382.
- Lankoff, A., Banasik, A., & Nowak, M. (2002). Protective effect of melatonin against nodularin-induced oxidative stress. *Arch. Toxicol.*, 76(3), 158–165.

- Laothong, U., Pinlaor, P., Hiraku, Y., Boonsiri, P., Prakobwong, S., Khoontawad, J., & Pinlaoro, et al. (2010). Protective effect of melatonin against *Opisthorchis viverrini*-induced oxidative and nitrosative DNA damage and liver injury in hamsters. *J. Pineal Res.*, 49(3), 271–282.
- Laszlo, A., Davidson, T., Harvey, A., Sim, J. E., Malyapa, R. S., Spitz, D. R., & Roti Roti, J. L. (2006). Alterations in heat-induced radiosensitization accompanied by nuclear structure alterations in Chinese hamster cells. *Int. J. Hyperthermia*, 22(1), 43–60.
- Lederman, J. (2017a). 19 American diplomats in Cuba suffering health problems after “attacks” blamed on secret sonic weapon. *Independent*. September 2.
- Lederman, J. (2017b). Trump: Cuba “is responsible” for attacks on US personnel. Associated Press, October 16.
- Lederman, J. (2018). US stands by claim workers attacked in Cuba, maybe by virus. Associated Press International, January 10.
- Lederman, J., & Lee, M. (2017). Cuba tells Tillerson: No culpability. Associated Press, September 27.
- Lederman, J., & Weissenstein, J. M. (2017). Dangerous sound? What Americans heard in Cuba attacks. *Associated Press News*, October 13.
- Lederman, J., Weissenstein, M., & Lee, M. (2017). Cuba mystery grows: New details on what befell US diplomats. Associated Press News, September 16.
- Lederman, J., Weissenstein, M., Lee, M., & Associated Press. (2017). Bizarre Cuba mystery: Did sonic weapon cause U.S. diplomats’ brain injuries? *Mercury News*, September 14.
- Lee, H. C., & Wei, Y. H. (1997). Role of mitochondria in human aging. *J. Biomed. Sci.*, 4(6), 319–326.
- Lee, J. C., Kim, J., Park, J. K., Chung, G. H., & Jang, Y. S. (2003). The antioxidant, rather than prooxidant, activities of quercetin on normal cells: Quercetin protects mouse thymocytes from glucose oxidase-mediated apoptosis. *Exp. Cell Res.*, 291(2), 386–397.
- Leitgeb, N. (1998). *Electromagnetic hypersensitivity*. Paper presented at the International Workshop on Electromagnetic Fields and Non-specific Health Symptoms. European Cooperation in the Field of Science and Technical Research, Graz, Austria.
- Leszczynski, D. (2015). *Science and conflict of interest in bioelectromagnetics*. Keynote speech at Swiss Association Gigahertz, March 7. <http://bit.ly/1CMWkHq>
- Leszczynski, D., Joenvaara, S., Reivinen, J., & Kuokka, R. (2002). Non-thermal activation of the hsp27/p38MAPK stress pathway by mobile phone radiation in human endothelial cells: Molecular mechanism for cancer- and blood-brain barrier-related effects. *Differentiation*, 70(2–3), 120–129.
- Levallois, P., Neutra, R., Lee, G., & Hristova, L. (2002). Study of self-reported hypersensitivity to electromagnetic fields in California. *Environ. Health Perspect*, 110 (Suppl. 4), 619–623.
- Li, J., Meng, Z., Zhang, G., Xing, Y., Feng, L., Fan, S., Fan, L., et al. (2015). N-acetylcysteine relieves oxidative stress and protects hippocampus of rat from radiation-induced apoptosis by inhibiting caspase-3. *Biomed. Pharmacother*, 70, 1–6.

- Li, W. H., Li, Y. Z., Song, D. D., Wang, X. R., Liu, M., Wu, X. D., et al. (2014). Calreticulin protects rat microvascular endothelial cells against microwave radiation-induced injury by attenuating endoplasmic reticulum stress. *Microcirculation*, 21(6), 506–515.
- Li, W., Lidebjer, C., Yuan, X. M., Szymanowski, A., Backteman, K., Ernerudh, J., Leanderson, P., et al. (2008). NK cell apoptosis in coronary artery disease: Relation to oxidative stress. *Atherosclerosis*, 199(1), 65–72.
- Liang, F. Q., & Godley, B. F. (2003). Oxidative stress-induced mitochondrial DNA damage in human retinal pigment epithelial cells: A possible mechanism for RPE aging and age-related macular degeneration. *Exp. Eye Res.*, 76(4), 397–403.
- Liang, F. Q., Green, L., Wang, C., Alssadi, R., & Godley, B. F. (2004). Melatonin protects human retinal pigment epithelial (RPE) cells against oxidative stress. *Exp. Eye Res.*, 78(6), 1069–1075.
- Lilja, J. J., Kivisto, K. T., & Neuvonen, P. J. (1998). Grapefruit juice–simvastatin interaction: Effect on serum concentrations of simvastatin, simvastatin acid, and HMG-CoA reductase inhibitors. *Clin. Pharmacol. Ther.*, 64(5), 477–483.
- Lin, J. C. (1980). The microwave auditory phenomenon. *Proceedings of the IEEE*, 68(1), 67–73.
- Lin'kova, N. S., Poliakova, V. O., Kvetnoi, I. M., Trofimov, A. V., & Sevost'ianova, N. N. (2011). [Characteristics of the pineal gland and thymus relationship in aging]. *Adv. Gerontol.*, 24(1), 38–42.
- Liu, D. D., Ren, Z., Yang, G., Zhao, Q. R., & Mei, Y. A. (2014). Melatonin protects rat cerebellar granule cells against electromagnetic field-induced increases in Na(+) currents through intracellular Ca(2+) release. *J. Cell. Mol. Med.*, 18(6), 1060–1070.
- Liu, Y., Zhu, B., Wang, X., Luo, L., Li, P., Paty, D. W., & Cynader, M. S., et al. (2003). Bilirubin as a potent antioxidant suppresses experimental autoimmune encephalomyelitis: Implications for the role of oxidative stress in the development of multiple sclerosis. *J. Neuroimmunol.*, 139(1–2), 27–35.
- Lochhead, J. J., McCaffrey, G., Quigley, C. E., Finch, J., DeMarco, K. M., Nametz, N., & Davis, T. P., et al. (2010). Oxidative stress increases blood-brain barrier permeability and induces alterations in occludin during hypoxia-reoxygenation. *J. Cereb. Blood Flow Metab.*, 30(9), 1625–1636.
- Loscalzo, J. (2002). Oxidative stress in endothelial cell dysfunction and thrombosis. *Pathophysiol. Haemost. Thromb.*, 32(5–6), 359–360.
- Maes, M., Kubera, M., Mihaylova, I., Geffard, M., Galecki, P., Leunis, J. C., & Berk, M., et al. (2013). Increased autoimmune responses against auto-epitopes modified by oxidative and nitrosative damage in depression: Implications for the pathways to chronic depression and neuroprogression. *J. Affect. Disord.*, 149(1–3), 23–29.
- Maisch, D. (2012). Smart meter health concerns: Just a Nocebo effect, or an emerging public health nightmare? *Australasian Coll. Nutr. Environ. Med. J.*, 31(2), 15–19.
- Maldonado, M. D., Murillo-Cabezas, F., Calvo, J. R., Lardone, P. J., Tan, D. X., Guerrero, J. M., & Reiter, R. J., et al. (2007). Melatonin as pharmacologic support in burn patients: A proposed solution to thermal injury-related lymphocytopenia and oxidative damage. *Crit. Care Med.*, 35(4), 1177–1185.
- Man, I., Murphy, J., & Ferguson, J. (1999). Fluoroquinolone phototoxicity: A comparison of moxifloxacin and lomefloxacin in normal volunteers. *J. Antimicrob. Chemother.*, 43 (Suppl. B), 77–82.

- Mancuso, M., Coppede, F., Migliore, L., Siciliano, G., & Murri, L. (2006). Mitochondrial dysfunction, oxidative stress and neurodegeneration. *J. Alzheimers, Dis.*, 10(1), 59–73.
- Manda, K., Anzai, K., Kumari, S., & Bhatia, A. L. (2007). Melatonin attenuates radiation-induced learning deficit and brain oxidative stress in mice. *Acta Neurobiol. Exp. (Wars)*, 67(1), 63–70.
- Manda, K., & Reiter, R. J. (2010). Melatonin maintains adult hippocampal neurogenesis and cognitive functions after irradiation. *Prog. Neurobiol.*, 90(1), 60–68.
- Manda, K., Ueno, M., & Anzai, K. (2007). AFMK, a melatonin metabolite, attenuates X-ray-induced oxidative damage to DNA, proteins and lipids in mice. *J. Pineal Res.*, 42(4), 386–393.
- Manda, K., Ueno, M., & Anzai, K. (2008). Melatonin mitigates oxidative damage and apoptosis in mouse cerebellum induced by high-LET 56Fe particle irradiation. *J. Pineal Res.*, 44(2), 189–196.
- Manwaring, N., Jones, M. M., Wang, J. J., Rochtchina, E., Howard, C., Newall, P., Mitchell, P., et al. (2007). Mitochondrial DNA haplogroups and age-related hearing loss. *Arch. Otolaryngol. Head Neck Surg.*, 133(9), 929–933.
- Marie, I., & Noblet, C. (2009). [Drug-associated tendon disorders: After fluoroquinolones . . . here are statins!]. *Rev. Med. Interne*, 30(4), 307–310.
- Markova, E., Hillert, L., Malmgren, L., Persson, B. R., & Belyaev, I. Y. (2005). Microwaves from GSM mobile telephones affect 53BP1 and gamma-H2AX foci in human lymphocytes from hypersensitive and healthy persons. *Environ. Health Perspect.*, 113(9), 1172–1177.
- Martin, J. A., Taylor, C., Trehan, M., Baron, E. D., & Anstey, A. V. (2006). Phototesting in patients with Smith-Lemli-Opitz syndrome confirms sensitivity to UV-A. *Arch. Dermatol.*, 142(5), 647–648.
- Martin, V., Sainz, R. M., Antolin, I., Mayo, J. C., Herrera, F., & Rodriguez, C. (2002). Several antioxidant pathways are involved in astrocyte protection by melatonin. *J. Pineal Res.*, 33(4), 204–212.
- Martinez-Samano, J., Torres-Duran, P. V., Juarez-Oropeza, M. A., Elias-Vinas, D., & Verdugo-Diaz, L. (2010). Effects of acute electromagnetic field exposure and movement restraint on antioxidant system in liver, heart, kidney and plasma of Wistar rats: A preliminary report. *Int. J. Radiat. Biol.*, 86(12), 1088–1094.
- Massin, P., Guillausseau, P. J., Vialettes, B., Paquis, V., Orsini, F., Grimaldi, A. D., & Gaudric, A., et al. (1995). Macular pattern dystrophy associated with a mutation of mitochondrial DNA. *Am. J. Ophthalmol.*, 120(2), 247–248.
- Math teacher asks school to protect children from Wi-Fi. (2015). North Kingston School Committee Meeting, Rhode Island USA, February 10. <https://www.youtube.com/watch?v=UqrW4ZJb5Uc>
- Math teacher raises concerns about Wi-Fi comparing the effects to a concussion. (2014). North Kingston School Committee Meeting, Rhode Island, May 13. <https://www.youtube.com/watch?v=QbgIdyhAXfM>
- Mayo, J. C., Tan, D. X., Sainz, R. M., Lopez-Burillo, S., & Reiter, R. J. (2003). Oxidative damage to catalase induced by peroxyl radicals: functional protection by melatonin and other antioxidants. *Free Radic. Res.*, 37(5), 543–553.
- Mayo, J. C., Tan, D. X., Sainz, R. M., Natarajan, M., Lopez-Burillo, S., & Reiter, R. J. (2003). Protection against oxidative protein damage induced by metal-catalyzed

- reaction or alkylperoxyl radicals: Comparative effects of melatonin and other antioxidants. *Biochim. Biophys. Acta*, 1620(1–3), 139–150.
- McCally, R. L., Farrell, R. A., Barger, C. B., Kues, H. A., & Hochheimer, B. F. (1986). Nonionizing radiation damage in the eye. *Johns Hopkins APL Technologies Digest*, 7, 73–91.
- McCarty, D. E., Carrubba, S., Chesson, A. L., Frilot, C., Gonzalez-Toledo, E., & Marino, A. A. (2011). Electromagnetic hypersensitivity: Evidence for a novel neurological syndrome. *Int. J. Neurosci.*, 121(12), 670–676.
- McQuade, J. M., Merritt, J. H., Miller, S. A., Scholin, T., Cook, M. C., Salazar, A., et al. (2009). Radiofrequency-radiation exposure does not induce detectable leakage of albumin across the blood-brain barrier. *Radiat. Res.*, 171(5), 615–621.
- McRee, D. I. (1980). Soviet and Eastern European research on biological effects of microwave radiation. *Proc. IEEE*, 68, 84–91.
- Meena, R., Kumari, K., Kumar, J., Rajamani, P., Verma, H. N., & Kesari, K. K. (2014). Therapeutic approaches of melatonin in microwave radiations-induced oxidative stress-mediated toxicity on male fertility pattern of Wistar rats. *Electromagn. Biol. Med.*, 33(2), 81–91.
- Megha, K., Deshmukh, P. S., Banerjee, B. D., Tripathi, A. K., Ahmed, R., & Abegaonkar, M. P. (2015). Low intensity microwave radiation induced oxidative stress, inflammatory response and DNA damage in rat brain. *NeuroToxicology*, 51, 158–165.
- Mehta, K. D., Mehta, A. K., Halder, S., Khanna, N., Tripathi, A. K., & Sharma, K. K. (2014). Protective effect of melatonin on propoxur-induced impairment of memory and oxidative stress in rats. *Environ. Toxicol.*, 29(6), 705–713.
- Melchiorri, D., Reiter, R. J., Attia, A. M., Hara, M., Burgos, A., & Nistico, G. (1995). Potent protective effect of melatonin on in vivo paraquat-induced oxidative damage in rats. *Life Sci.*, 56(2), 83–89.
- Miller, E. R., III, Pastor-Barriuso, R., Dalal, D., Riemersma, R. A., Appel, L. J., & Guallar, E. (2005). Meta-analysis: High-dosage vitamin E supplementation may increase all-cause mortality. *Ann. Intern. Med.*, 142(1), 37–46.
- Miller, V. M., Lawrence, D. A., Mondal, T. K., & Seegal, R. F. (2009). Reduced glutathione is highly expressed in white matter and neurons in the unperturbed mouse brain: Implications for oxidative stress associated with neurodegeneration. *Brain Res.*, 1276, 22–30.
- Mitchell, J. B., & Russo, A. (1987). The role of glutathione in radiation and drug induced cytotoxicity. *Br. J. Cancer Suppl.*, 8, 96–104.
- Miyamoto, N., Maki, T., Pham, L. D., Hayakawa, K., Seo, J. H., Mandeville, E. T., Mandeville, J. B., et al. (2013). Oxidative stress interferes with white matter renewal after prolonged cerebral hypoperfusion in mice. *Stroke*, 44(12), 3516–3521.
- Modi, G., Heckman, J. M., & Saffer, D. (1992). Vitelliform macular degeneration associated with mitochondrial myopathy. *Br. J. Ophthalmol.*, 76(1), 58–60.
- Molden, E., Skovlund, E., & Braathen, P. (2008). Risk management of simvastatin or atorvastatin interactions with CYP3A4 inhibitors. *Drug Saf.*, 31(7), 587–596.
- Montilla, P. L., Tunez, I. F., Munoz de Agueda, C., Gascon, F. L., & Soria, J. V. (1998). Protective role of melatonin and retinol palmitate in oxidative stress and hyperlipidemic nephropathy induced by adriamycin in rats. *J. Pineal Res.*, 25(2), 86–93.

- Montilla, P. L., Vargas, J. F., Tunez, I. F., Munoz de Agueda, M. C., Valdelvira, M. E., & Cabrera, E. S. (1998). Oxidative stress in diabetic rats induced by streptozotocin: Protective effects of melatonin. *J. Pineal Res.*, 25(2), 94–100.
- Mor, M., Spadoni, G., Diamantini, G., Bedini, A., Tarzia, G., Silva, C., Vacondio, F., et al. (2003). Antioxidant and cytoprotective activity of indole derivatives related to melatonin. *Adv. Exp. Med. Biol.*, 527, 567–575.
- Morimoto, K., Kawada, A., Hiruma, M., Ishibashi, A., & Banba, H. (1995). Photosensitivity to simvastatin with an unusual response to photopatch and photo tests. *Contact Dermatitis*, 33(4), 274.
- Morishima, I., Matsui, H., Mukawa, H., Hayashi, K., Toki, Y., Okumura, K., Ito, T., et al. (1998). Melatonin, a pineal hormone with antioxidant property, protects against adriamycin cardiomyopathy in rats. *Life Sci.*, 63(7), 511–521.
- Morishima, I., Okumura, K., Matsui, H., Kaneko, S., Numaguchi, Y., Kawakami, K., Mokuno, S., et al. (1999). Zinc accumulation in adriamycin-induced cardiomyopathy in rats: Effects of melatonin, a cardioprotective antioxidant. *J. Pineal Res.*, 26(4), 204–210.
- Munoz-Cortes, M., Cabre, C., Villa, D., Vives, J. P., Arruche, M., Soler, J., Compte, M. T., et al. (2013). Oxidative stress and other risk factors for white matter lesions in chronic hemodialysis patients. *Clin. Nephrol.*, 80(3), 187–197.
- Murphy, G. M. (2001). Diseases associated with photosensitivity. *J. Photochem. Photobiol. B*, 64(2–3), 93–98.
- Myers, S. L. (2018). More Americans evacuated from China over mysterious ailments. *New York Times*, June 30.
- Navara, K. J., & Nelson, R. J. (2007). The dark side of light at night: Physiological, epidemiological, and ecological consequences. *J. Pineal Res.*, 43(3), 215–224.
- Navarro, A., Sanchez Del Pino, M. J., Gomez, C., Peralta, J. L., & Boveris, A. (2002). Behavioral dysfunction, brain oxidative stress, and impaired mitochondrial electron transfer in aging mice. *Am. J. Physiol. Regul. Integr. Comp. Physiol.*, 282(4), R985–R992.
- Navarro, A., Segura, J., Portoles, M., & Gomez-Perretta, C. (2003). The microwave syndrome: A preliminary study in Spain. *Electromagnetic Biology and Medicine*, 22(2&3), 161–169.
- Naziroglu, M., Celik, O., Ozgul, C., Cig, B., Dogan, S., Bal, R., & Gumral, N., et al. (2012). Melatonin modulates wireless (2.45 GHz)-induced oxidative injury through TRPM2 and voltage gated Ca(2+) channels in brain and dorsal root ganglion in rat. *Physiol. Behav.*, 105(3), 683–692.
- Naziroglu, M., Tokat, S., & Demirci, S. (2012). Role of melatonin on electromagnetic radiation-induced oxidative stress and Ca2+ signaling molecular pathways in breast cancer. *J. Recept. Signal Transduct. Res.*, 32(6), 290–297.
- Nedorost, S. T., Dijkstra, J. W., & Handel, D. W. (1989). Drug-induced photosensitivity reaction. *Arch Dermatol.*, 125(3), 433–434.
- Neri, S., Signorelli, S., Pulvirenti, D., Mauceri, B., Cilio, D., Bordonaro, F., & Abate, G., et al. (2006). Oxidative stress, nitric oxide, endothelial dysfunction and tinnitus. *Free Radic. Res.*, 40(6), 615–618.
- [A new congenital photosensitivity syndrome. Smith-Lemili-Opitz syndrome]. (1999). *Hautarzt*, 50(2), 159.

- Nicolson, G. L., & Conklin, K. A. (2008). Reversing mitochondrial dysfunction, fatigue and the adverse effects of chemotherapy of metastatic disease by molecular replacement therapy. *Clin. Exp. Metastasis*, 25(2), 161–169.
- Nikka, A. (2014). Former Nokia boss: Mobile-phones wrecked my health. (Transl. Henrik Eriksson). *Satakunnan Kansa*. <http://betweenrockandhardplace.wordpress.com/2014/10/18/former-nokia-technology-chief-mobile-phones-wrecked-my-health>
- Nishiyama, A., Nakano, D., & Hitomi, H. (2010). [Calcium antagonists: Current and future applications based on new evidence. Effects of calcium channel blockers on oxidative stress]. *Clin. Calcium*, 20(1), 38–44.
- Nittby, H., Brun, A., Eberhardt, J., Malmgren, L., Persson, B. R., & Salford, L. G. (2009). Increased blood-brain barrier permeability in mammalian brain 7 days after exposure to the radiation from a GSM-900 mobile phone. *Pathophysiology*, 16(2–3), 103–112.
- Nittby, H., Grafstrom, G., Eberhardt, J. L., Malmgren, L., Brun, A., Persson, B. R., & Salford, L. G., et al. (2008). Radiofrequency and extremely low-frequency electromagnetic field effects on the blood-brain barrier. *Electromagn. Biol. Med.*, 27(2), 103–126.
- Nordström, G. (2004). *The invisible disease*. New York: O Books.
- Oberfeld, G., Navarro, A. E., Portoles, M., Maestu, C., & Gomez-Perretta, C. (2004). *The microwave syndrome: Further aspects of a Spanish study*. Paper presented at the WHO 3rd International Workshop on Biological Effects of Electromagnetic Fields, Kos, Greece, October.
- Ochoa, J. J., Diaz-Castro, J., Kajarabille, N., Garcia, C., Guisado, I. M., De Teresa, C., & Guisado, R., et al. (2011). Melatonin supplementation ameliorates oxidative stress and inflammatory signaling induced by strenuous exercise in adult human males. *J. Pineal Res.*, 51(4), 373–380.
- Oh, J., Ban, M. R., Miskie, B. A., Pollex, R. L., & Hegele, R. A. (2007). Genetic determinants of statin intolerance. *Lipids Health Dis.*, 6, 7.
- Oksay, T., Naziroglu, M., Dogan, S., Guzel, A., Gumral, N., & Kosar, P. A. (2012). Protective effects of melatonin against oxidative injury in rat testis induced by wireless (2.45 GHz) devices. *Andrologia*, 46, 65–72.
- Oktem, F., Ozguner, F., Mollaoglu, H., Koyu, A., & Uz, E. (2005). Oxidative damage in the kidney induced by 900-MHz-emitted mobile phone: Protection by melatonin. *Arch. Med. Res.*, 36(4), 350–355.
- Oliinyk, E. V., & Meshchyshe, I. F. (2004). [Effect of melatonin and radiation on pro- and antioxidant state of the liver and blood of rats]. *Ukr. Biokhim. Zh.* (1999), 76(5), 144–147.
- Oliveira, H. S., Goncalo, M., & Figueiredo, A. C. (2000). Photosensitivity to lomefloxacin: A clinical and photobiological study. *Photodermatol. Photoimmunol. Photomed.*, 16(3), 116–120.
- Omurtag, G. Z., Tozan, A., Sehirli, A. O., & Sener, G. (2008). Melatonin protects against endosulfan-induced oxidative tissue damage in rats. *J. Pineal Res.*, 44(4), 432–438.
- Oral, B., Guney, M., Ozguner, F., Karahan, N., Mungan, T., Comlekci, S., & Cesur, G., (2006). Endometrial apoptosis induced by a 900-MHz mobile phone: Preventive effects of vitamins E and C. *Adv. Ther.*, 23(6), 957–973.

- Ortega-Gutierrez, S., Garcia, J. J., Martinez-Ballarín, E., Reiter, R. J., Millan-Plano, S., Robinson, M., & Acuna-Castroviejo, D. (2002). Melatonin improves deferoxamine antioxidant activity in protecting against lipid peroxidation caused by hydrogen peroxide in rat brain homogenates. *Neurosci. Lett.*, 323(1), 55–59.
- Ortiz, F., Acuna-Castroviejo, D., Doerrier, C., Dayoub, J. C., Lopez, L. C., Venegas, C., Garcia, J. A., et al. (2015). Melatonin blunts the mitochondrial/NLRP3 connection and protects against radiation-induced oral mucositis. *J. Pineal Res.*, 58(1), 34–49.
- Othman, A. I., Edrees, G. M., El-Missiry, M. A., Ali, D. A., Aboel-Nour, M., & Dabdoub, B. R. (2014). Melatonin controlled apoptosis and protected the testes and sperm quality against bisphenol A-induced oxidative toxicity. *Toxicol. Ind. Health*, 32, 1537–1549.
- Othman, A. I., El-Missiry, M. A., & Amer, M. A. (2001). The protective action of melatonin on indomethacin-induced gastric and testicular oxidative stress in rats. *Redox. Rep.*, 6(3), 173–177.
- Otonello, S., Foroni, C., Carta, A., Petrucco, S., & Maraini, G. (2000). Oxidative stress and age-related cataract. *Ophthalmologica*, 214(1), 78–85.
- Ozacak, V. H., Barut, F., & Ozacak, H. S. (2009). Melatonin provides neuroprotection by reducing oxidative stress and HSP70 expression during chronic cerebral hypoperfusion in ovariectomized rats. *J. Pineal Res.*, 47(2), 156–163.
- Ozacak, V. H., Sayan, H., Arslan, S. O., Altaner, S., & Aktas, R. G. (2005). Protective effect of melatonin on contractile activity and oxidative injury induced by ischemia and reperfusion of rat ileum. *Life Sci.*, 76(14), 1575–1588.
- Ozcelik, N., Soyoz, M., & Kilinc, I. (2004). Effects of ochratoxin A on oxidative damage in rat kidney: Protective role of melatonin. *J. Appl. Toxicol.*, 24(3), 211–215.
- Ozdemir, D., Uysal, N., Gonenc, S., Acikgoz, O., Sonmez, A., Topcu, A., Ozdemir, N., et al. (2005). Effect of melatonin on brain oxidative damage induced by traumatic brain injury in immature rats. *Physiol Res*, 54(6), 631–637.
- Ozguner, F., Altinbas, A., Ozaydin, M., Dogan, A., Vural, H., Kisioglu, A. N., & Cesur, G., et al. (2005). Mobile phone-induced myocardial oxidative stress: Protection by a novel antioxidant agent caffeic acid phenethyl ester. *Toxicol. Ind. Health*, 21(9), 223–230.
- Ozguner, F., Bardak, Y., & Comlekci, S. (2006). Protective effects of melatonin and caffeic acid phenethyl ester against retinal oxidative stress in long-term use of mobile phone: A comparative study. *Mol. Cell. Biochem.*, 282(1–2), 83–88.
- Ozguner, F., Oktem, F., Armagan, A., Yilmaz, R., Koyu, A., Demirel, R., Vural, H., et al. (2005). Comparative analysis of the protective effects of melatonin and caffeic acid phenethyl ester (CAPE) on mobile phone-induced renal impairment in rat. *Mol. Cell Biochem.*, 276(1–2), 31–37.
- Ozguner, F., Oktem, F., Ayata, A., Koyu, A., & Yilmaz, H. R. (2005). A novel antioxidant agent caffeic acid phenethyl ester prevents long-term mobile phone exposure-induced renal impairment in rat. Prognostic value of malondialdehyde, N-acetyl-beta-D-glucosaminidase and nitric oxide determination. *Mol. Cell Biochem.*, 277(1–2), 73–80.
- Pachalska, M., DiMauro, S., Forminska-Kapuscik, M., Kurzbauer, H., Talar, J., MacQueen, B. D., Pawlicka, I., et al. (2002). The course of vision disturbances in a patient with the MELAS syndrome. *Med. Sci. Monit.*, 8(2), CS11–CS20.

- Packer, L., & Fuchs, J. (Eds.). (1993). *Vitamin E in health and disease*. New York: Dekker.
- Page, S. R., & Yee, K. C. (2014). Rhabdomyolysis in association with simvastatin and dosage increment in clarithromycin. *Intern. Med. J.*, 44(7), 690–693.
- Pal, S., & Chatterjee, A. K. (2006). Possible beneficial effects of melatonin supplementation on arsenic-induced oxidative stress in Wistar rats. *Drug Chem. Toxicol.*, 29(4), 423–433.
- Pall, M. L. (2015). Scientific evidence contradicts findings and assumptions of Canadian Safety Panel 6: Microwaves act through voltage-gated calcium channel activation to induce biological impacts at non-thermal levels, supporting a paradigm shift for microwave/lower frequency electromagnetic field action. *Rev. Environ. Health*, 30(2), 99–116.
- Pall, M. (2018). Wi-Fi is an important threat to health. *Environmental Research*, 164, 405–416.
- Palozza, P., Luberto, C., Calviello, G., Ricci, P., & Bartoli, G. M. (1997). Antioxidant and prooxidant role of beta-carotene in murine normal and tumor thymocytes: Effects of oxygen partial pressure. *Free Radic. Biol. Med.*, 22(6), 1065–1073.
- Panagopoulos, D. J., Johansson, O., & Carlo, G. L. (2015). Polarization: A key difference between man-made and natural electromagnetic fields, in regard to biological activity. *Scientific Reports*, October, 1–10.
- Panetta, A. (2017). Canada won't follow U.S. in reducing Cuba staff. *Canadian Press*, September 29.
- Park, M. T., Kim, M. J., Kang, Y. H., Choi, S. Y., Lee, J. H., Choi, J. A., Kang, C. M., et al. (2005). Phytosphingosine in combination with ionizing radiation enhances apoptotic cell death in radiation-resistant cancer cells through ROS-dependent and -independent AIF release. *Blood*, 105(4), 1724–1733.
- Parry, B. L., Meliska, C. J., Sorenson, D. L., Lopez, A., Martinez, L. F., Hauger, R. L., & Elliott, J. A., et al. (2010). Increased sensitivity to light-induced melatonin suppression in premenstrual dysphoric disorder. *Chronobiol. Int.*, 27(7), 1438–1453.
- Peet, M., & Horrobin, D. F. (2002). A dose-ranging study of the effects of ethyl-eicosapentaenoate in patients with ongoing depression despite apparently adequate treatment with standard drugs. *Arch. Gen. Psychiatry*, 59(10), 913–919.
- Peleg, M., Nativ, O., & Richter, E. D. (2018). Radio frequency radiation-related cancer: Assessing causation in the occupational/military setting. *Environ. Res.*, 163, 123–133.
- Perlez, J., & Myers, L. (2018). China pledges to investigate fears of sonic attacks on U.S. diplomats. *New York Times*, June 7.
- Petitjeans, F., Nadaud, J., Perez, J. P., Debieu, B., Olive, F., Villevieille, T., & Pats, B., et al. (2003). A case of rhabdomyolysis with fatal outcome after a treatment with levofloxacin. *Eur. J. Clin. Pharmacol.*, 59(10), 779–780.
- Pineda, M., Playan-Arison, A., Alcaine-Villarroya, M. J., Vernet, A. M., Serra-Castanera, A., Solano, A., Vilaseca, M. A., et al. (2004). [Familiar chronic progressive external ophthalmoplegia of mitochondrial origin]. *Rev. Neurol.*, 38(11), 1023–1027.
- Polyakova, V. O., Linkova, N. S., Kvetnoy, I. M., & Khavinson, V. (2011). Functional unity of the thymus and pineal gland and study of the mechanisms of aging. *Bull. Exp. Biol. Med.*, 151(5), 627–630.

- Popov, S. S., Shulgin, K. K., Popova, T. N., Pashkov, A. N., Agarkov, A. A., & de Carvalho, M. A. (2015). Effects of melatonin-aided therapy on the glutathione antioxidant system activity and liver protection. *J. Biochem. Mol. Toxicol.*, 29, 449–457.
- Porto Arceo, J. A. (2003). [Special features of NSAID intolerance in children]. *Allergol. Immunopathol. (Madr.)*, 31(3), 109–125.
- Powell, R. M. (2015). Symptoms after exposure to smart meter radiation. PMID, 25478801, <https://www.scribd.com/doc/289777267/Symptoms-after-Exposure-to-Smart-Meter-Radiation>
- Princ, F. G., Maxit, A. G., Cardalda, C., Batlle, A., & Juknat, A. A. (1998). In vivo protection by melatonin against delta-aminolevulinic acid-induced oxidative damage and its antioxidant effect on the activity of haem enzymes. *J. Pineal Res.*, 24(1), 1–8.
- Prithivirajsingh, S., Story, M. D., Bergh, S. A., Geara, F. B., Ang, K. K., Ismail, S. M., Stevens, C. W., et al. (2004). Accumulation of the common mitochondrial DNA deletion induced by ionizing radiation. *FEBS Lett.*, 571(1–3), 227–232.
- Profumo, E., Buttari, B., & Rigano, R. (2011). Oxidative stress in cardiovascular inflammation: Its involvement in autoimmune responses. *Int. J. Inflamm.*, 2011, 295705.
- Qi, X., Lewin, A. S., Sun, L., Hauswirth, W. W., & Guy, J. (2007). Suppression of mitochondrial oxidative stress provides long-term neuroprotection in experimental optic neuritis. *Invest. Ophthalmol. Vis. Sci.*, 48(2), 681–691.
- Qian, Q., Nasr, S. H., Akogyeram, C. O., & Sethi, S. (2012). Myoglobin-associated acute kidney injury in the setting of ciprofloxacin administration. *Am. J. Kidney Dis.*, 59(3), 462–466.
- Qin, F., Zhang, J., Cao, H., Yi, C., Li, J. X., Nie, J., Chen, L. L., et al. (2012). Effects of 1800-MHz radiofrequency fields on circadian rhythm of plasma melatonin and testosterone in male rats. *J. Toxicol. Environ. Health A*, 75(18), 1120–1128.
- Raines, J. K. (1981). *Electromagnetic field interactions with the human body: Observed effects and theories*. (NASA CR 166661), Report prepared for: National Aeronautics and Space Administration. <https://ntrs.nasa.gov/search.jsp?R=19810017132>
- Rao, M. V., & Chhunchha, B. (2010). Protective role of melatonin against the mercury induced oxidative stress in the rat thyroid. *Food Chem. Toxicol.*, 48(1), 7–10.
- Rapoport, S. I., & Breus, T. K. (2011). [Melatonin as a most important factor of natural electromagnetic fields impacting patients with hypertensive disease and coronary heart disease. Part 1]. *Klin. Med. (MOSCOW)*, 89(3), 9–14.
- Razygraev, A. V. (2010). [Pineal gland glutathione peroxidase activity in rats and its age-associated change]. *Adv. Gerontol.*, 23(3), 392–395.
- Redmayne, M., & Johansson, O. (2014). Could myelin damage from radiofrequency electromagnetic field exposure help explain the functional impairment electrohypersensitivity? A review of the evidence. *J. Toxicol. Environ. Health B Crit. Rev.*, 17(5), 247–258.
- Reiter, R. J. (1993a). Electromagnetic fields and melatonin production. *Biomed. Pharmacother.*, 47(10), 439–444.
- Reiter, R. J. (1993b). Static and extremely low frequency electromagnetic field exposure: Reported effects on the circadian production of melatonin. *J. Cell Biochem.*, 51(4), 394–403.

- Reiter, R. J. (1994). Melatonin suppression by static and extremely low frequency electromagnetic fields: Relationship to the reported increased incidence of cancer. *Rev. Environ. Health*, 10(3–4), 171–186.
- Reutelingsperger, C. P., & van Heerde, W. L. (1997). Annexin V, the regulator of phosphatidylserine-catalyzed inflammation and coagulation during apoptosis. *Cell. Mol. Life. Sci.*, 53(6), 527–532.
- Reynolds, A., Laurie, C., Mosley, R. L., & Gendelman, H. E. (2007). Oxidative stress and the pathogenesis of neurodegenerative disorders. *Int. Rev. Neurobiol.*, 82, 297–325.
- Rezzani, R., Buffoli, B., Rodella, L., Stacchiotti, A., & Bianchi, R. (2005). Protective role of melatonin in cyclosporine A-induced oxidative stress in rat liver. *Int. Immunopharmacol.*, 5(9), 1397–1405.
- Riordan-Eva, P. (2000). Neuro-ophthalmology of mitochondrial diseases. *Curr. Opin. Ophthalmol.*, 11(6), 408–412.
- Roberts, M. (2004). Statin-fortified drinking water? *BBC News*, August 1.
- Robison, J. G., Pendleton, A. R., Monson, K. O., Murray, B. K., & O'Neill, K. L. (2002). Decreased DNA repair rates and protection from heat induced apoptosis mediated by electromagnetic field exposure. *Bioelectromagnetics*, 23(2), 106–112.
- Robles, F., & Semple, K. (2017a). “Health attacks” on U.S. diplomats in Cuba baffle both countries. *New York Times*, October 4.
- Robles, F., & Semple, K. (2017b). U.S. and Cuba baffled by “health attacks” on American envoys in Cuba. *New York Times*, August 12.
- Rogers, A. (2017). Were US diplomats in Cuba victims of a sonic attack—or something else? *Wired*, October 5.
- Röösli, M., Möser, M., Baldinini, Y., Meier, M., & Braun-Fahrlander, C. (2004). Symptoms of ill health ascribed to electromagnetic field exposure: A questionnaire survey. *Int. J. Hyg. Environ. Health*, 207, 141–150.
- Rose, S., Melnyk, S., Pavliv, O., Bai, S., Nick, T. G., Frye, R. E., James, S. J., et al. (2012). Evidence of oxidative damage and inflammation associated with low glutathione redox status in the autism brain. *Transl. Psychiatry*, 2, e134.
- Rosen, N. (2008). Headache and mitochondrial disorders. *Headache*, 48(5), 733–734.
- Rosenzweig, S., & Carmichael, S. T. (2013). Age-dependent exacerbation of white matter stroke outcomes: A role for oxidative damage and inflammatory mediators. *Stroke*, 44(9), 2579–2586.
- Rossignol, D. A., & Frye, R. E. (2012). Mitochondrial dysfunction in autism spectrum disorders: A systematic review and meta-analysis. *Mol. Psychiatry*, 17(3), 290–314.
- Rowan, C., Brinker, A. D., Nourjah, P., Chang, J., Mosholder, A., Barrett, J. S., & Avigan, M., et al. (2009). Rhabdomyolysis reports show interaction between simvastatin and CYP3A4 inhibitors. *Pharmacoevidentiol. Drug Saf.*, 18(4), 301–309.
- Rubin, G. J., Das Munshi, J., & Wessely, S. (2005). Electromagnetic hypersensitivity: A systematic review of provocation studies. *Psychosom. Med.*, 67, 224–232.
- Rucker, J. C., Hamilton, S. R., Bardenstein, D., Isada, C. M., & Lee, M. S. (2006). Linezolid-associated toxic optic neuropathy. *Neurology*, 66(4), 595–598.
- Ryan, B. J., Nissim, A., & Winyard, P. G. (2014). Oxidative post-translational modifications and their involvement in the pathogenesis of autoimmune diseases. *Redox Biol.*, 2, 715–724.

- Sadchikova, M. N., & Glotova, K. V. (1973). The clinic, pathogenesis, treatment, and outcome of radiowave sickness. (Translated from Russian.) In Z. V. Gordon (Ed.) 1974, *Biological effects of radiofrequency electromagnetic fields* (pp. 54–62). Arlington, VA: Joint Publications Research Service.
- Sadir, S., Deveci, S., Korkmaz, A., & Oter, S. (2007). Alpha-tocopherol, beta-carotene and melatonin administration protects cyclophosphamide-induced oxidative damage to bladder tissue in rats. *Cell Biochem. Funct.*, 25(5), 521–526.
- Sahna, E., Parlakpınar, H., Turkoz, Y., & Acet, A. (2005). Protective effects of melatonin on myocardial ischemia/reperfusion induced infarct size and oxidative changes. *Physiol. Res.*, 54(5), 491–495.
- Sahna, E., Parlakpınar, H., Vardi, N., Cigremis, Y., & Acet, A. (2004). Efficacy of melatonin as protectant against oxidative stress and structural changes in liver tissue in pinealectomized rats. *Acta Histochem.*, 106(5), 331–336.
- Sailer, E., Kamarachev, J., Boehler, A., Speich, R., Hofer, M., Benden, C., French, L. E., et al. (2011). Persistent photodamage following drug photosensitization in a lung-transplant recipient. *Photodermatol. Photoimmunol. Photomed.*, 27(4), 213–215.
- Sainz, R. M., Reiter, R. J., Tan, D. X., Roldan, F., Natarajan, M., Quiros, I., Hevia, D., et al. (2008). Critical role of glutathione in melatonin enhancement of tumor necrosis factor and ionizing radiation-induced apoptosis in prostate cancer cells in vitro. *J. Pineal. Res.*, 45(3), 258–270.
- Salford, L. G., Brun, A., Stureson, K., Eberhardt, J. L., & Persson, B. R. (1994). Permeability of the blood-brain barrier induced by 915 MHz electromagnetic radiation, continuous wave and modulated at 8, 16, 50, and 200 Hz. *Microsc. Res. Tech.*, 27(6), 535–542.
- Salido, G. M., & Rosado, J. A. (2009). *Apoptosis: Involvement of oxidative stress and intracellular Ca²⁺ homeostasis*. New York: Springer.
- Sandbach, J. M., Coscun, P. E., Grossniklaus, H. E., Kokoszka, J. E., Newman, N. J., & Wallace, D. C. (2001). Ocular pathology in mitochondrial superoxide dismutase (Sod2)-deficient mice. *Invest. Ophthalmol. Vis. Sci.*, 42(10), 2173–2178.
- Sanjith, S., Raodeo, A., Clerk, A., Pandit, R., & Karnad, D. R. (2012). Moxifloxacin-induced rhabdomyolysis. *Intensive Care Med.*, 38(4), 725.
- Santini, R., Santini, P., Danze, J. M., Le Ruz, P., & Seigne, M. (2002). [Investigation on the health of people living near mobile telephone relay stations: Incidence according to distance and sex]. *Pathol. Biol. (Paris)*, 50(6), 369–373.
- Saravanan, K. S., Sindhu, K. M., & Mohanakumar, K. P. (2007). Melatonin protects against rotenone-induced oxidative stress in a hemiparkinsonian rat model. *J. Pineal Res.*, 42(3), 247–253.
- Sastre, J., Pallardo, F. V., & Vina, J. (2003). The role of mitochondrial oxidative stress in aging. *Free Radic. Biol. Med.*, 35(1), 1–8.
- Savastano, M., Brescia, G., & Marioni, G. (2007). Antioxidant therapy in idiopathic tinnitus: preliminary outcomes. *Arch. Med. Res.*, 38(4), 456–459.
- Schaefer, A. M., Blakely, E. L., Griffiths, P. G., Turnbull, D. M., & Taylor, R. W. (2005). Ophthalmoplegia due to mitochondrial DNA disease: The need for genetic diagnosis. *Muscle Nerve*, 32(1), 104–107.
- Scheife, R. T., Cramer, W. R., & Decker, E. L. (1993). Photosensitizing potential of ofloxacin. *Int. J. Dermatol.*, 32(6), 413–416.

- Schooneveld, H., & Kuiper, J. (2007). Electrohypersensitivity (EHS) in the Netherlands: A questionnaire survey. Stichting EHS (Dutch EHS Foundation).
- Schreier, N., Huss, A., & Roosli, M. (2006). The prevalence of symptoms attributed to electromagnetic field exposure: A cross-sectional representative survey in Switzerland. *Soz Präventivmed.* 51(4), 202–209.
- Schröttner, J., & Leitgeb, N. (2008). Sensitivity to electricity: Temporal changes in Austria. *BMC Public Health*, 8, 310.
- Schumaker, J. (2013). *Moments in U.S. diplomatic history: Microwaving embassy Moscow—Another perspective*. Association for Diplomat Studies and Training, September. adst.org/2013/2009/microwaving-embassy-moscow-another-perspective/#.WeOG0Dtrxfg
- Seidman, M. D., Khan, M. J., Bai, U., Shirwany, N., & Quirk, W. S. (2000). Biologic activity of mitochondrial metabolites on aging and age-related hearing loss. *Am. J. Otol.*, 21(2), 161–167.
- Sener, G., Atasoy, B. M., Ersoy, Y., Arbak, S., Sengoz, M., & Yegen, B. C. (2004). Melatonin protects against ionizing radiation-induced oxidative damage in corpus cavernosum and urinary bladder in rats. *J. Pineal Res.*, 37(4), 241–246.
- Sener, G., Jahovic, N., Tosun, O., Atasoy, B. M., & Yegen, B. C. (2003). Melatonin ameliorates ionizing radiation-induced oxidative organ damage in rats. *Life Sci.*, 74(5), 563–572.
- Sener, G., Kacmaz, A., User, Y., Ozkan, S., Tilki, M., & Yegen, B. C. (2003). Melatonin ameliorates oxidative organ damage induced by acute intra-abdominal compartment syndrome in rats. *J. Pineal Res.*, 35(3), 163–168.
- Sener, G., Paskaloglu, K., Toklu, H., Kapucu, C., Ayanoglu-Dulger, G., Kacmaz, A., & Sakarcin, A., et al. (2004). Melatonin ameliorates chronic renal failure-induced oxidative organ damage in rats. *J. Pineal Res.*, 36(4), 232–241.
- Sener, G., Sehirli, A. O., & Ayanoglu-Dulger, G. (2003). Melatonin protects against mercury(II)-induced oxidative tissue damage in rats. *Pharmacol. Toxicol.*, 93(6), 290–296.
- Sener, G., Sehirli, A. O., Satiroglu, H., Keyer-Uysal, M., & Yegen, B. C. (2002a). Melatonin improves oxidative organ damage in a rat model of thermal injury. *Burns*, 28(5), 419–425.
- Sener, G., Sehirli, A. O., Satiroglu, H., Keyer-Uysal, M., & Yegen, B. C. (2002b). Melatonin prevents oxidative kidney damage in a rat model of thermal injury. *Life Sci.*, 70(25), 2977–2985.
- Senol, N., & Naziroglu, M. (2014). Melatonin reduces traumatic brain injury-induced oxidative stress in the cerebral cortex and blood of rats. *Neural Regen Res.*, 9(11), 1112–1116.
- Sepcic, J., Bucuk, M., Perkovic, O., Sepic-Grahovac, D., Troselj-Vukic, B., Poljak, I., Crnic-Martinovic, M., et al. (2010). Drug-induced aseptic meningitis, sensorineural hearing loss and vestibulopathy. *Coll. Antropol.*, 34(3), 1101–1104.
- Shafiee, H., Mohammadi, H., Rezayat, S. M., Hosseini, A., Baeri, M., Hassani, S., Mohammadirad, A., et al. (2010). Prevention of malathion-induced depletion of cardiac cells mitochondrial energy and free radical damage by a magnetic magnesium-carrying nanoparticle. *Toxicol. Mech. Methods*, 20(9), 538–543.
- Shah, A. A., & Sinha, A. A. (2013). Oxidative stress and autoimmune skin disease. *Eur. J. Dermatol.*, 23(1), 5–13.

- Sharma, A. K., Mehta, A. K., Rathor, N., Chalawadi Hanumantappa, M. K., Khanna, N., & Bhattacharya, S. K. (2013). Melatonin attenuates cognitive dysfunction and reduces neural oxidative stress induced by phosphamidon. *Fundam. Clin. Pharmacol.*, 27(2), 146–151.
- Sharma, S., & Haldar, C. (2006). Melatonin prevents X-ray irradiation induced oxidative damage in peripheral blood and spleen of the seasonally breeding rodent, *Funambulus pennanti*, during reproductively active phase. *Int. J. Radiat. Biol.*, 82(6), 411–419.
- Shea, C. R., Wimberly, J., & Hasan, T. (1986). Mitochondrial phototoxicity sensitized by doxycycline in cultured human carcinoma cells. *J. Invest. Dermatol.*, 87(3), 338–342.
- Shen, Y. X., Xu, S. Y., Wei, W., Sun, X. X., Liu, L. H., Yang, J., & Dong, C., et al. (2002). The protective effects of melatonin from oxidative damage induced by amyloid beta-peptide 25–35 in middle-aged rats. *J. Pineal Res.*, 32(2), 85–89.
- Shifow, A. A., Kumar, K. V., Naidu, M. U., & Ratnakar, K. S. (2000). Melatonin, a pineal hormone with antioxidant property, protects against gentamicin-induced nephrotoxicity in rats. *Nephron.*, 85(2), 167–174.
- Shil, P., Sanghvi, H., Vidyasagar, P. B., & Mishra, K. P. (2005). Enhancement of radiation cytotoxicity in murine cancer cells by electroporation: In vitro and in vivo studies. *J. Environ. Pathol. Toxicol. Oncol.*, 24(4), 291–298.
- Shimoda, K., Akahane, K., Nomura, M., & Kato, M. (1996). LD50 value, phototoxicity and convulsion induction test of the new quinolone antibacterial agent (S)-10-[(S)-(8-amino-6-azaspiro[3,4]octan-6-yl)]-9-fluoro-2, 3-dihydro-3-methyl-7-oxo-7H-pyrido[1,2,3-de][1,4]benzoxazine-6-carboxylic acid hemihydrate in laboratory animals. *Arzneimittelforschung*, 46(6), 625–628.
- Shinjo, T., & Shinjo, A. (2014). Significant decrease of clinical symptoms after mobile phone base station removed: An intervention study. *Umwelt medizin gesellschaft (Environmental Medicine Company)*, 27, 294–301.
- Shirazi, A., Haddadi, G. H., Asadi-Amoli, F., Sakhaee, S., Ghazi-Khansari, M., & Avand, A. (2011). Radioprotective effect of melatonin in reducing oxidative stress in rat lenses. *Cell J.*, 13(2), 79–82.
- Shirazi, A., Mihandoost, E., Mohseni, M., Ghazi-Khansari, M., & Rabie Mahdavi, S. (2013). Radio-protective effects of melatonin against irradiation-induced oxidative damage in rat peripheral blood. *Phys. Med.*, 29(1), 65–74.
- Shokrzadeh, M., Chabra, A., Naghshvar, F., Ahmadi, A., Jafarnejhad, M., & Hasani-Nourian, Y. (2015). Protective effects of melatonin against cyclophosphamide-induced oxidative lung toxicity in mice. *Drug Res. (Stuttgart)*, 65, 281–286.
- Shonai, T., Adachi, M., Sakata, K., Takekawa, M., Endo, T., Imai, K., & Hareyama, M., et al. (2002). MEK/ERK pathway protects ionizing radiation-induced loss of mitochondrial membrane potential and cell death in lymphocytic leukemia cells. *Cell Death Differ.*, 9(9), 963–971.
- Simone, G., Tamba, M., & Quintiliani, M. (1983). Role of glutathione in affecting the radiosensitivity of molecular and cellular systems. *Radiat. Environ. Biophys.*, 22(3), 215–223.
- Singer, D. H., Martin, G. J., Magid, N., Weiss, J. S., Schaad, J. W., Kehoe, R., Zheutlin, T., et al. (1988). Low heart rate variability and sudden cardiac death. *J. Electrocardiol.*, 21, S46–S55.

- Singh, S., Mani, K. V., & Kapoor, N. (2015). Effect of occupational EMF exposure from radar at two different frequency bands on plasma melatonin and serotonin levels. *Int. J. Radiat. Biol.*, 91, 426–434.
- Sinzinger, H., Lupattelli, G., & Chehne, F. (2000). Increased lipid peroxidation in a patient with CK-elevation and muscle pain during statin therapy. *Atherosclerosis*, 153(1), 255–256.
- Sinzinger, H., Lupattelli, G., Chehne, F., Oguogho, A., & Furberg, C. D. (2001). Iso-prostane 8-epi-PGF2alpha is frequently increased in patients with muscle pain and/or CK-elevation after HMG-Co-enzyme-A-reductase inhibitor therapy. *J. Clin. Pharm. Ther.*, 26(4), 303–310.
- Sinzinger, H., & O'Grady, J. (2004). Professional athletes suffering from familial hypercholesterolaemia rarely tolerate statin treatment because of muscular problems. *British Journal of Clinical Pharmacology*, 57(4), 525–528.
- Sirav, B., & Seyhan, N. (2009). Blood-brain barrier disruption by continuous-wave radio frequency radiation. *Electromagn. Biol. Med.*, 28(2), 215–222.
- Sirav, B., & Seyhan, N. (2011). Effects of radiofrequency radiation exposure on blood-brain barrier permeability in male and female rats. *Electromagn. Biol. Med.*, 30(4), 253–260.
- Skaper, S. D., Floreani, M., Ceccon, M., Facci, L., & Giusti, P. (1999). Excitotoxicity, oxidative stress, and the neuroprotective potential of melatonin. *Ann. NY Acad. Sci.*, 890, 107–118.
- Smart meters or no power at all? Nevada Energy sends armed men to disconnect power—just for opting out. (2012). <https://richardalanmiller.com/newsblog/smart-meter-or-no-power-at-all-nevada-energy-sends-armed-men-to-disconnect-power-j>
- Smith, R. (2005). Medical journals are an extension of the marketing arm of pharmaceutical companies. *PLoS Med.*, 2(5), e138.
- Smith, R. (2006). Conflicts of interest: How money clouds objectivity. *J. R. Soc. Med.*, 99(6), 292–297.
- Smits, B. W., Westeneng, H. J., van Hal, M. A., van Engelen, B. G., & Overeem, S. (2012). Sleep disturbances in chronic progressive external ophthalmoplegia. *Eur. J. Neurol.*, 19(1), 176–178.
- Snyder, R. D., & Cooper, C. S. (1999). Photogenotoxicity of fluoroquinolones in Chinese hamster V79 cells: Dependency on active topoisomerase II. *Photochem. Photobiol.*, 69(3), 288–293.
- Soderqvist, F., Carlberg, M., Hansson Mild, K., & Hardell, L. (2009). Exposure to an 890-MHz mobile phone-like signal and serum levels of S100B and transthyretin in volunteers. *Toxicol. Lett.*, 189(1), 63–66.
- Soderqvist, F., Carlberg, M., & Hardell, L. (2009). Mobile and cordless telephones, serum transthyretin and the blood-cerebrospinal fluid barrier: A cross-sectional study. *Environ. Health*, 8, 19.
- Sokolovic, D., Djindjic, B., Nikolic, J., Bjelakovic, G., Pavlovic, D., Kocic, G., Krstic, D., et al. (2008). Melatonin reduces oxidative stress induced by chronic exposure of microwave radiation from mobile phones in rat brain. *J. Radiat. Res.*, 49(6), 579–586.
- Sokolovic, D., Djordjevic, B., Kocic, G., Veljkovic, A., Marinkovic, M., Basic, J., Jevtovic-Stoimenov, T., et al. (2013). Melatonin protects rat thymus against

- oxidative stress caused by exposure to microwaves and modulates proliferation/apoptosis of thymocytes. *Gen. Physiol. Biophys.*, 32(1), 79–90.
- Someya, S., Xu, J., Kondo, K., Ding, D., Salvi, R. J., Yamasoba, T., Rabinovitch, P. S., et al. (2009). Age-related hearing loss in C57BL/6J mice is mediated by Bak-dependent mitochondrial apoptosis. *Proc. Natl. Acad. Sci. USA*, 106(46), 19432–19437.
- Sousa, S. C., & Castilho, R. F. (2005). Protective effect of melatonin on rotenone plus Ca²⁺-induced mitochondrial oxidative stress and PC12 cell death. *Antioxid. Redox. Signal*, 7(9–10), 1110–1116.
- Souza, L. C., Wilhelm, E. A., Bortolatto, C. F., Nogueira, C. W., Boeira, S. P., & Jesse, C. R. (2014). The protective effect of melatonin against brain oxidative stress and hyperlocomotion in a rat model of mania induced by ouabain. *Behav. Brain Res.*, 271, 316–324.
- Soyoz, M., Ozcelik, N., Kilinc, I., & Altuntas, I. (2004). The effects of ochratoxin A on lipid peroxidation and antioxidant enzymes: A protective role of melatonin. *Cell Biol. Toxicol.*, 20(4), 213–219.
- Spadoni, G., Diamantini, G., Bedini, A., Tarzia, G., Vacondio, F., Silva, C., Rivara, M., et al. (2006). Synthesis, antioxidant activity and structure-activity relationships for a new series of 2-(N-acylaminoethyl)indoles with melatonin-like cytoprotective activity. *J. Pineal Res.*, 40(3), 259–269.
- State of California. (2017). Wireless Telecommunications Bill. Senate Bill 649 (SB-649). Passed the Assembly September 13, 2017. Passed the Senate September 14, 2017. Vetoed by Governor Brown.
- Steele, L. (2000). Prevalence and patterns of Gulf War illness in Kansas veterans: Association of symptoms with characteristics of person, place, and time of military service. *Am. J. Epidemiol*, 152(10), 992–1002.
- Steele, L., Lockridge, O., Gerkovich, M. M., Cook, M. R., & Sastre, A. (2015). Butyrylcholinesterase genotype and enzyme activity in relation to Gulf War illness: Preliminary evidence of gene-exposure interaction from a case-control study of 1991 Gulf War veterans. *Environ. Health*, 14, 4.
- Stone, R. (2017). Stressful conditions, not “sonic weapon,” sickened U.S diplomats, Cuba asserts. *Science*, December 5. <http://www.sciencemag.org/news/2017/2012/stressful-conditions-not-sonic-weapon-sickened-us-diplomats-cuba-panel-asserts>
- Stone, R. (2018). Reports of inner-ear damage deepen diplomat controversy: As mystery symptoms reported in Cuba spread to China, some blame an attack; others see “suggestion and paranoia,” *Science*, 360(6395), 1281–1282.
- Suke, S. G., Kumar, A., Ahmed, R. S., Chakraborti, A., Tripathi, A. K., Mediratta, P. K., & Banerjee, B. D., et al. (2006). Protective effect of melatonin against propoxur-induced oxidative stress and suppression of humoral immune response in rats. *Indian J. Exp. Biol.*, 44(4), 312–315.
- Sutken, E., Aral, E., Ozdemir, F., Uslu, S., Alatas, O., & Colak, O. (2007). Protective role of melatonin and coenzyme Q10 in ochratoxin A toxicity in rat liver and kidney. *Int. J. Toxicol.*, 26(1), 81–87.
- Swanson, R. L., Hampton, S., Green-McKenzie, J., Diaz-Arrastia, R., Grady, M. S., Verma, R., Biester, R., et al. (2018). Neurological manifestations among US Government personnel reporting directional audible and sensory phenomena in Havana, Cuba. *JAMA*, February 15. doi:10.1001/jama.2018.1742

- Tachover, D. (2013). The Israeli Supreme Court ordered the Israeli government to investigate the number of children currently suffering from EHS. *EMFacts*, July 23. <https://www.emfacts.com/2013/2007/the-israeli-supreme-court-ordered-the-israeli-government-to-investigate-the-number-of-children-currently-suffering-fr>
- Takemori, K., Murakami, T., Kometani, T., & Ito, H. (2013). Possible involvement of oxidative stress as a causative factor in blood-brain barrier dysfunction in stroke-prone spontaneously hypertensive rats. *Microvasc. Res.*, 90, 169–172.
- Tan, D. X., Manchester, L. C., Qin, L., & Reiter, R. J. (2016). Melatonin: A mitochondrial targeting molecule involving mitochondrial protection and dynamics. *Int. J. Mol. Sci.*, 17(12).
- Tang, J., Zhang, Y., Yang, L., Chen, Q., Tan, L., Zuo, S., Feng, H., et al. (2015). Exposure to 900 MHz electromagnetic fields activates the mmp-1/ERK pathway and causes blood-brain barrier damage and cognitive impairment in rats. *Brain Res.*, 1601, 92–101.
- Tarwadi, K., & Agte, V. (2004). Linkages of antioxidant, micronutrient, and socioeconomic status with the degree of oxidative stress and lens opacity in indian cataract patients. *Nutrition*, 20(3), 261–267.
- Taylor, A., Jacques, P. F., & Epstein, E. M. (1995). Relations among aging, antioxidant status, and cataract. *Am. J. Clin. Nutr.*, 62(6 Suppl.), 1439S–1447S.
- Taysi, S., Koc, M., Buyukokuroglu, M. E., Altinkaynak, K., & Sahin, Y. N. (2003). Melatonin reduces lipid peroxidation and nitric oxide during irradiation-induced oxidative injury in the rat liver. *J. Pineal Res.*, 34(3), 173–177.
- Taysi, S., Memisogullari, R., Koc, M., Yazici, A. T., Aslankurt, M., Gumustekin, K., Al., B., et al. (2008). Melatonin reduces oxidative stress in the rat lens due to radiation-induced oxidative injury. *Int. J. Radiat. Biol.*, 84(10), 803–808.
- Tendon disorders due to statins. (2010). *Prescribe Int.*, 19(106), 73.
- Thomas, B., & Mohanakumar, K. P. (2004). Melatonin protects against oxidative stress caused by 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine in the mouse nigrostriatum. *J. Pineal Res.*, 36(1), 25–32.
- Thomas, S. M., Gebicki, J. M., & Dean, R. T. (1989). Radical initiated alpha-tocopherol depletion and lipid peroxidation in mitochondrial membranes. *Biochim. Biophys. Acta*, 1002(2), 189–197.
- Thual, N., Penven, K., Chevallier, J. M., Domp Martin, A., & Leroy, D. (2005). [Fluvastatin-induced dermatomyositis]. *Ann. Dermatol. Venerol.*, 132(12 Pt. 1), 996–999.
- Tiwari, V., & Chopra, K. (2013). Resveratrol abrogates alcohol-induced cognitive deficits by attenuating oxidative-nitrosative stress and inflammatory cascade in the adult rat brain. *Neurochem. Int.*, 62(6), 861–869.
- Tok, L., Naziroglu, M., Dogan, S., Kahya, M. C., & Tok, O. (2014). Effects of melatonin on Wi-Fi-induced oxidative stress in lens of rats. *Indian J. Ophthalmol.*, 62(1), 12–15.
- Tomas-Zapico, C., Martinez-Fraga, J., Rodriguez-Colunga, M. J., Tolivia, D., Hardeland, R., & Coto-Montes, A. (2002). Melatonin protects against delta-aminolevulinic acid-induced oxidative damage in male Syrian hamster Harderian glands. *Int. J. Biochem. Cell Biol.*, 34(5), 544–553.

- Totan, Y., Cekic, O., Borazan, M., Uz, E., Sogut, S., & Akyol, O. (2001). Plasma malondialdehyde and nitric oxide levels in age related macular degeneration. *Br. J. Ophthalmol.*, 85(12), 1426–1428.
- Tressider, A. (2017). Electrosensitivity—an environmental illness, an authentic diagnosis, not a delusional disorder: *To my Medical Colleagues, GPs, Psychiatrists, Neurologists and Others* [letter]. <http://www.es-uk.info/wp-content/uploads/2018/05/ES%20letter%20psych%20paper%20Jan2%20217.pdf>
- Trisciuglio, D., Krasnowska, E., Maggi, A., Pozzi, R., Parasassi, T., & Sapor, O. (2002). Phototoxic effect of fluoroquinolones on two human cell lines. *Toxicol. In Vitro*, 16(4), 449–456.
- Tseng, M.-C. M., Lin, Y.-P., & Cheng, T.-J. (2011). Prevalence and psychiatric comorbidity of self-reported electromagnetic field sensitivity in Taiwan: A population-based study. *J. Formosan Medical Association*, 110, 634–641.
- Tucker, P. (2018). Here's what invisible brain weapons did to us diplomatic workers in Cuba. *DefenseOne*, February 15.
- Tunali, T., Sener, G., Yarat, A., & Emekli, N. (2005). Melatonin reduces oxidative damage to skin and normalizes blood coagulation in a rat model of thermal injury. *Life Sci.*, 76(11), 1259–1265.
- Tunez, I., Montilla, P., Del Carmen Munoz, M., Feijoo, M., & Salcedo, M. (2004). Protective effect of melatonin on 3-nitropropionic acid-induced oxidative stress in synaptosomes in an animal model of Huntington's disease. *J. Pineal Res.*, 37(4), 252–256.
- Tunez, I., Munoz Mdel, C., Feijoo, M., Munoz-Castaneda, J. R., Bujalance, I., Valdelvira, M. E., Montolla Lopez, P., et al. (2003). Protective melatonin effect on oxidative stress induced by okadaic acid into rat brain. *J. Pineal Res.*, 34(4), 265–268.
- Turedi, S., Hanci, H., Topal, Z., Unal, D., Mercantepe, T., Bozkurt, I., Kaya, H., et al. (2015). The effects of prenatal exposure to a 900-MHz electromagnetic field on the 21-day-old male rat heart. *Electromagn. Biol. Med.*, 34(4), 390–397.
- Uygur, R., Aktas, C., Caglar, V., Uygur, E., Erdogan, H., & Ozen, O. A. (2013). Protective effects of melatonin against arsenic-induced apoptosis and oxidative stress in rat testes. *Toxicol. Ind. Health*, 32, 848–859.
- Vallis, K. A. (1991). Glutathione deficiency and radiosensitivity in AIDS patients. *Lancet*, 337(8746), 918–919.
- Van Campen, L. E., Murphy, W. J., Franks, J. R., Mathias, P. I., & Toraason, M. A. (2002). Oxidative DNA damage is associated with intense noise exposure in the rat. *Hear. Res.*, 164(1–2), 29–38.
- Vasin, M. V., Ushakov, I. B., Kovtun, V., Komarova, S. N., Semenova, L. A., & Galkin, A. A. (2004). [Comparative effectiveness of antioxidant melatonin and radioprotectors indralin and phenylephrine in local radiation injuries]. *Radiats. Biol. Radioecol.*, 44(1), 68–71.
- Vayssier-Taussat, M., Kreps, S. E., Adrie, C., Dall'Ava, J., Christiani, D., & Polla, B. S. (2002). Mitochondrial membrane potential: A novel biomarker of oxidative environmental stress. *Environ. Health Perspect.*, 110(3), 301–305.
- Vladutiu, G. D., Simmons, Z., Isackson, P. J., Tarnopolsky, M., Peltier, W. L., Barboi, A. C., Sripathi, N., et al. (2006). Genetic risk factors associated with lipid-lowering drug-induced myopathies. *Muscle Nerve*, 34(2), 153–162.

- Vos, O., van der Schans, G. P., & Roos-Verheij, W. S. (1986). Reduction of intracellular glutathione content and radiosensitivity. *Int. J. Radiat. Biol. Relat. Stud. Phys. Chem. Med.*, 50(1), 155–165.
- Vurucu, S., Karaoglu, A., Paksu, M. S., Yesilyurt, O., Oz, O., Unay, B., & Akin, R., et al. (2013). Relationship between oxidative stress and chronic daily headache in children. *Human and Experimental Toxicology*, 32(2), 113–119.
- Wagai, N., & Tawara, K. (1991). Important role of oxygen metabolites in quinolone antibacterial agent-induced cutaneous phototoxicity in mice. *Arch. Toxicol.*, 65(6), 495–499.
- Wagai, N., Yamaguchi, F., Sekiguchi, M., & Tawara, K. (1990). Phototoxic potential of quinolone antibacterial agents in Balb/c mice. *Toxicol. Lett.*, 54(2–3), 299–308.
- Waldman-Selsam, C. (2004). *Bamberg Appeal, on behalf of 114 physicians*. Open Letter to Edmund Stoiber, Prime Minister, Germany, August 3. <http://www.vws.org/documents/cell-project-documents/BambergAppeal.pdf>
- Wallace, D. C. (2001). Mitochondrial defects in neurodegenerative disease. *Ment. Retard. Dev. Disabil. Res. Rev.*, 7(3), 158–166.
- Wallace, F. (2017). Online comment in response to Blog post. *Between a Rock and a Hard Place. Science Blog on Mobile Phone Radiation and Health by Dariusz Leszczynski*, November 9. <https://betweenrockandhardplace.wordpress.com/2017/2011/2008/ehs-researchis-scientificallly-worthless-for-two-reasons/>
- Wang, C., Cong, J., Xian, H., Cao, X., Sun, C., & Wu, K. (2002). [The effects of electromagnetic pulse on fluidity and lipid peroxidation of mitochondrial membrane]. *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi*, 20(4), 266–268.
- Wang, G., Cai, P., Ansari, G. A., & Khan, M. F. (2007). Oxidative and nitrosative stress in trichloroethene-mediated autoimmune response. *Toxicology*, 229(3), 186–193.
- Wang, H., Wei, W., Wang, N. P., Gui, S. Y., Wu, L., Sun, W. Y., & Xu, S. Y., et al. (2005). Melatonin ameliorates carbon tetrachloride-induced hepatic fibrogenesis in rats via inhibition of oxidative stress. *Life Sci.*, 77(15), 1902–1915.
- Wang, H., Wei, W., Zhang, S. Y., Shen, Y. X., Yue, L., Wang, N. P., & Xu, S. Y., et al. (2005). Melatonin-selenium nanoparticles inhibit oxidative stress and protect against hepatic injury induced by *Bacillus Calmette-Guerin*/lipopolysaccharide in mice. *J. Pineal Res.*, 39(2), 156–163.
- Watanabe, K., Wakatsuki, A., Shinohara, K., Ikenoue, N., Yokota, K., & Fukaya, T. (2004). Maternally administered melatonin protects against ischemia and reperfusion-induced oxidative mitochondrial damage in premature fetal rat brain. *J. Pineal Res.*, 37(4), 276–280.
- Wei, Y. H. (1998). Oxidative stress and mitochondrial DNA mutations in human aging. *Proc. Soc. Exp. Biol. Med.*, 217(1), 53–63.
- Wei, Y. H., & Lee, H. C. (2002). Oxidative stress, mitochondrial DNA mutation, and impairment of antioxidant enzymes in aging. *Exp. Biol. Med. (Maywood)*, 227(9), 671–682.
- Weissenstein, M. (2018). US senator says no evidence of “sonic attacks” in Cuba. Associated Press International, January 6.
- Weissenstein, M., & Rodriguez, A. (2017). Cuba presents detailed defense against sonic attack charges. Associated Press, October 27.

- Weller, S. (2015). *Electromagnetic hypersensitivity*. Presented to the Electromagnetic Energy Reference Group Committee Meeting, May 20, electromagnetichealth.org/wp-content/uploads/2015/2006/EHS-Presentation-Steven-Weller.pdf
- Wernicke, A. G., Swistel, A. J., Parashar, B., & Myskowski, P. L. (2010). Levofloxacin-induced radiation recall dermatitis: A case report and a review of the literature. *Clin Breast Cancer*, 10(5), 404–406.
- West, J. G., Kapoor, N. S., Liao, S. Y., Chen, J. W., Bailey, L., & Nagourney, R. A. (2013). Multifocal breast cancer in young women with prolonged contact between their breasts and their cellular phones. *Case Rep. Med.*, 2013, 354682.
- Weydahl, A., Sothorn, R. B., Cornélissen, G., & Wetterberg, L. (2000). Geomagnetic activity influences the melatonin secretion at latitude 70° N. *Biomedicine and Pharmacotherapy*, 55 (Suppl. 1), s57–s62.
- Wilkinson, T. (2017). Cuban diplomats expelled from Washington over incident that harmed U.S. personnel in Havana, State Department says. *Los Angeles Times*, August 8.
- Williams, R. J., & Finch, E. D. (1974). Examination of the cornea following exposure to microwave radiation. *Aerospace Medicine*, (April), 393–396.
- Williams, S., Tamburic, S., & Lally, C. (2009). Eating chocolate can significantly protect the skin from UV light. *J. Cosmet. Dermatol.*, 8(3), 169–173.
- Witt, K. A., Mark, K. S., Sandoval, K. E., & Davis, T. P. (2008). Reoxygenation stress on blood-brain barrier paracellular permeability and edema in the rat. *Microvasc. Res.*, 75(1), 91–96.
- Wood, A. W., Armstrong, S. M., Sait, M. L., Devine, L., & Martin, M. J. (1998). Changes in human plasma melatonin profiles in response to 50 Hz magnetic field exposure. *J. Pineal Res.*, 25, 116–127.
- Wood, A. W., Loughran, S. P., & Stough, C. (2006). Does evening exposure to mobile phone radiation affect subsequent melatonin production? *Int. J. Radiat. Biol.*, 82(2), 69–76.
- Woolston, C. (2010). Victims of electrosensitivity syndrome say EMFs caused symptoms. *Los Angeles Times*, February 15. <http://articles.latimes.com/2010/feb/15/health/la-he-electromagnetic-syndrome2011-2010feb2015>
- Wright, K. (2013). Online comment in response to website article, May 31. <https://stopsmartmeters.org/direct-action/>
- www.es-uk. (2012). Gro Harlem Brundtland and EHS. *Electrosensitivity UK newsletter* www.es-uk.info. ElectroSensitivity UK.
- www.esnztrust. Electrosensitivity New Zealand.
- www.felo.no. Foreningen for el-overfølsomme (Norwegian Electrosensitive Society).
- www.iervn.com. (Electrosensitivity organization in Ireland).
- Xu, S. C., He, M. D., Zhong, M., Zhang, Y. W., Wang, Y., Yang, L., Yang, J., et al. (2010). Melatonin protects against nickel-induced neurotoxicity in vitro by reducing oxidative stress and maintaining mitochondrial function. *J. Pineal Res.*, 49(1), 86–94.
- Xu, S., Zhou, Z., Zhang, L., Yu, Z., Zhang, W., Wang, Y., Wang, X., et al. (2010). Exposure to 1800 MHz radiofrequency radiation induces oxidative damage to mitochondrial DNA in primary cultured neurons. *Brain Res*, 1311, 189–196.

- Yakymenko, I., Tsybulin, O., Sidorik, E., Henshel, D., Kyrylenko, O., & Kyrylenko, S. (2015). Oxidative mechanisms of biological activity of low-intensity radiofrequency radiation. *Electromagn. Biol. Med.*, 35(2), 186–202.
- Yalcinkaya, S., Unlucerci, Y., Giris, M., Olgac, V., Dogru-Abbasoglu, S., & Uysal, M. (2009). Oxidative and nitrosative stress and apoptosis in the liver of rats fed on high methionine diet: Protective effect of taurine. *Nutrition*, 25(4), 436–444.
- Yamasoba, T., Someya, S., Yamada, C., Weindruch, R., Prolla, T. A., & Tanokura, M. (2007). Role of mitochondrial dysfunction and mitochondrial DNA mutations in age-related hearing loss. *Hear. Res.*, 226(1–2), 185–193.
- Yi, X., Ding, L., Jin, Y., Ni, C., & Wang, W. (1994). The toxic effects, GSH depletion and radiosensitivity by BSO on retinoblastoma. *Int. J. Radiat. Oncol. Biol. Phys.*, 29(2), 393–396.
- Yilmaz, S., & Yilmaz, E. (2006). Effects of melatonin and vitamin E on oxidative-antioxidative status in rats exposed to irradiation. *Toxicology*, 222(1–2), 1–7.
- Young, A. J., & Lowe, G. M. (2001). Antioxidant and prooxidant properties of carotenoids. *Arch. Biochem. Biophys.*, 385(1), 20–27.
- Yu, J., Wu, L., & Lin, X. (1997). [Preliminary study of mitochondrial DNA deletions in age-related macular degeneration]. *Yan Ke Xue Bao*, 13(2), 52–55.
- Yuksel, M., Naziroglu, M., & Ozkaya, M. O. (2016). Long-term exposure to electromagnetic radiation from mobile phones and Wi-Fi devices decreases plasma prolactin, progesterone, and estrogen levels but increases uterine oxidative stress in pregnant rats and their offspring. *Endocrine*, 52(2), 352–362.
- Yurekli, A. I., Ozkan, M., Kalkan, T., Saybasili, H., Tuncel, H., Atukeren, P., Gumustas, K., et al. (2006). GSM base station electromagnetic radiation and oxidative stress in rats. *Electromagn. Biol. Med.*, 25(3), 177–188.
- Zaret, M. M. (1973). Microwave cataracts. *Medical Trial Technique Quarterly*, 19(3), 146–252.
- Zavodnik, I. B., Lapshina, E. A., Zavodnik, L. B., Labieniec, M., Bryszewska, M., & Reiter, R. J. (2004). Hypochlorous acid-induced oxidative stress in Chinese hamster B14 cells: Viability, DNA and protein damage and the protective action of melatonin. *Mutat. Res.*, 559(1–2), 39–48.
- Zehndner, C. M., Librizzi, L., Hedrich, J., Bauer, N. M., Angamo, E. A., de Curtis, M., & Luhmann, W. J., et al. (2013). Moderate hypoxia followed by reoxygenation results in blood-brain barrier breakdown via oxidative stress-dependent tight-junction protein disruption. *PLoS One*, 8(12), e82823.
- Zhang, J., Peng, R. Y., Ren, J. H., Li, J., Wang, S. M., Gao, Y. B., et al. (2011). [The protective effects of AduoLa Fuzhenglin on the heart injury induced by microwave exposure in rats]. *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi*, 29(5), 367–370.
- Zhang, L., Zhang, H. Q., Liang, X. Y., Zhang, H. F., Zhang, T., & Liu, F. E. (2013). Melatonin ameliorates cognitive impairment induced by sleep deprivation in rats: Role of oxidative stress, BDNF and CaMKII. *Behav. Brain Res.*, 256, 72–81.
- Zhang, X., Gao, Y., Dong, J., Wang, S., Yao, B., Zhang, J., Hu, Shaohua, et al. (2014). The compound Chinese medicine “Kang Fu Ling” protects against high power microwave-induced myocardial injury. *PLoS One*, 9(7), e101532.
- Zhang, Y., Zhang, X., Rabbani, Z. N., Jackson, I. L., & Vujaskovic, Z. (2012). Oxidative stress mediates radiation lung injury by inducing apoptosis. *Int. J. Radiat. Oncol. Biol. Phys.*, 83(2), 740–748.

- Zhu, W., Zhang, W., Wang, H., Xu, J., Li, Y., & Lv, S. (2014). Apoptosis induced by microwave radiation in pancreatic cancer JF305 cells. *Can. J. Physiol. Pharmacol.*, 92(4), 324–329.
- Zimmer, C. (2017a). The “sonic attack” that likely wasn’t. *New York Times*, October 6.
- Zimmer, C. (2017b). What’s a science reporter to do when sound evidence isn’t sound? *New York Times*, October 6.
- Zoric, L., Kosanovic-Jakovic, N., Colak, E., Radosavljevic, A., Jaksic, V., & Stevic, S. (2008). [Oxidative stress in association with risk factors for the occurrence and development of age-related macular degeneration]. *Vojnosanit. Pregl.*, 65(4), 313–318.

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