September 29, 2017

Rosemary Chiavetta, Secretary
Pennsylvania Public Utility Commission
Commonwealth Keystone Building
400 North Street, 2nd Floor
P.O. Box 3265
Harrisburg, PA 17105-3265

Re: **Richard N. Myers v. PPL Electric Utilities Corporation**
Docket No. C-2017-2620710

Dear Secretary Chiavetta:

Enclosed is my answer to Post & Schell letter dated September 11, 2017 regarding my Complaint of August 10, 2017.

I disagree with PPL's response. In keeping with the mission of the Pennsylvania Utility Commission to ensure safe utility service is provided to the people of Pennsylvania I again ask for your assistance to obtain the following:

1. PPL identify and provide me with the scientific research by which PPL claims smart meter non-thermal RF-EMF radiation is safe for occupants in their home.

2. Exempt me and my rental property tenants from having smart meters installed in our homes.

Respectfully submitted,

Richard Myers

RESPONSE OF RICHARD N MYERS
to
ANSWER OF PPL ELECTRIC UTILITIES CORPORATION
TO THE COMPLAINT OF RICHARD N. MYERS

My responses below are keyed to the paragraph numbers in the PPL Electric Utilities Corporation letter to the Pennsylvania Public Utilities Commission dated September 11, 2017. For brevity, I've used the abbreviation "PPL" when referring to PPL Electric Utilities Corporation.

1. **PPL statement:** "It is denied that the smart meters pose any health or safety concerns."

   Myers response:

   - This is an astonishing statement. PUC is not able to cite any studies that were conducted to show smart meter RF-EMF radiation is safe, yet there is a wealth of information in the scientific literature to the contrary. Hundreds of reports and studies by scientists, Medical Doctors, medical writers, independent researchers, plus books by authors and articles in scholarly or peer review journals raise serious safety questions and/or report adverse health effects from non-thermal RF-EMF radiation. Many researchers assert that human absorption of non-thermal RF-EMF radiation contributes to brain cancer, neurological disease, immune dysfunction, reproductive disorders, electromagnetic hypersensitivity (EHS) and other illnesses. Victims suffering from EHS complain of insomnia, fatigue, strong headaches, heart palpitations, dizziness, memory loss, anxiety and other ailments. Clearly something is going on here.

   - As a layman I have ready access to scientific literature in the public domain. I do not understand **why does PPL and PUC (whose mission is to ensure the safety of public utility services) ignore this growing body of research or deny its validity**, especially since microwave industry-funded research has confirmed 32% of their studies FOUND and documented non-thermal radiation waves effects. However, ICNIRP, the **microwave industry's** premiere professional association states it will never accept those results. If this is scientific collusion to defraud the public and PUCs of the correct state of microwave EMF-RF science, that is prosecutable under U.S. RICO law.

     I am enclosing copies of 4 reports from U.S. and European scientific literature, just a few of many possible examples addressing the risks from non-thermal RF-EMF radiation:

     - U.S National Toxicology Program (NTP): Study Show Real Damage from Cell Phone Radiation (Enclosure 1)

       http://www.gummulator.com/cell-phone-radiation-damage/

- Clinical Study: Metabolic and Genetic Screening of Electromagnetic Hypersensitive Subjects as a Feasible Tool for Diagnostics and Intervention (Enclosure 2)

  http://dx.doi.org/10.1155/2014/924184

- Comments on the Draft Report by the California Council on Science and Technology "Health Impacts of Radio Frequency from Smart Meters" by Daniel Hirsch, 31 January 2011 (Enclosure 3)

  http://www.committeetobridgethegap.org/pdf/110212_RFrad_comments.pdf

- Electromagnetic and Radiofrequency Fields Effect on Human Health , a report by the American Academy of Environmental Medicine (Enclosure 4)

  https://www.aaemonline.org/emf_rf_position.php

- Approximately one-third (32%) of industry studies and two-thirds of non-industry studies on the effects of non-thermal RF-EMF showed adverse health effects. The author of this study is Dr. Henry Lai, Professor Emeritus - Department of Bioengineering, University of Washington and a renowned scientist in non-ionizing microwave research.

- The World Health Organization determined that non-thermal RF-EMF radiation emitted from wireless devices are a class 2B possible human carcinogen, in the same class as lead, DDT, and chloroform.

PPL statement: "The Complainant has failed to allege he or his tenants suffer from any specific or safety effects resulting from the installation of smart meters."

Myers response:

- Correct. But I do not have a smart meter installed on my home. So of course, I would never claim that I'm suffering from adverse health effects from a smart meter. I am a 72-year old male who is blessed with excellent health. I am responsible for my health, not PPL/PUC. I do not want to risk eroding my health by the cumulative effects of non-thermal RF-EMF radiation emitted by smart meters and other wireless devices.

- I could not report any adverse health effects for tenants at my 11 rental properties because their homes did not have smart meters installed at the time. Furthermore, I am not privy to their private health information, something I must remind all involved that health records are protected by HIPPA law.

PPL statement: "PPL Electric's smart meters meet all applicable safety requirements under state and federal law."

Myers response.

- A State Representative informs me that smart meters installed by utilities in Pennsylvania comply with FCC 's 1996 guidelines for safe human exposure. However, these FCC guidelines are 21 years old and are based on even older World War 2 radar and heat research. Furthermore, I understand those tests were conducted to determine safe exposure levels for thermal (heat) radiation. In 1996 the **FCC never tested for nor set safe non-thermal RF-EMF radiation limits for smart meters.**

- If testing specific to smart meters was not done how could spikes of intense radiation bursts (sinusoidal waves aka "dirty electricity") from smart meters into peoples' homes thousands of times each day 24/7/365 be deemed safe? Does anyone know the cumulative effect of such long-term exposure to smart meter radiation compounded with radiation from cell phones, wi-fi, and other wireless devices? As in my August 10 Complaint to the Pennsylvania Utilities Commission, I again request that you please identify and supply the studies that support PPL's claim that their smart meters do not pose short or long-term health or safety concerns for consumers. Those documents providing scientific proof should be in the PA PUC's records even before approving AMI Smart Meter rollouts or deployments. It is my understanding no such studies were done nor provided during the PA PUC's consideration to implement AMI Smart Meters, a violation of health and environmental impact studies that must be provided and were not, according to my knowledge.

- We are all witnesses to products or technologies that manufacturers and government regulators originally believed were safe but were later found to be unsafe. Asbestos, agent orange, lead based paint, benzene, smoking, air bags, mammograms, bad pharmaceutical drugs, etc. are just a few examples. If smart meters were not tested for safety and non-thermal RF-EMF exposure levels, the PA PUC, PPL and utilities could be creating adverse health effects on countless Pennsylvanians and inviting massive class action law suits.

PPL statement: "Nothing in the Public Utility Code, the Commission's orders and regulations, or PPL Electric's Smart Meter Plan states that a customer can opt-out of a smart meter installation".

Myers response: If that is PUC and PPL regulatory interpretation it violates federal and state laws as written. Specifically:

- The Federal Energy Policy Act of 2005 does not make the smart meter program mandatory. Customer participation is not mandatory (see Enclosure 5).

- Wording in PA HB 2200/Act 129 does not state that smart meters are mandatory. Quoting from Section 3.9 (7):

(2) Electric distribution companies shall furnish smart meter technology as follows:

(I) ***Upon request from a customer*** that agrees to pay the cost of the smart meter at the time of the request.

(II) In new building construction

(III) In accordance with a depreciation schedule not to exceed 15 years.

- PUC resorting to the vague language "depreciation schedule" in (III) above as grounds to overturn customer choice in (I) is agency over reach. PPL and PUC are violating the intent of federal and state lawmakers. Because of deceptive wording many lawmakers thought HB2200 was a bill that did not make smart meters mandatory when they voted for it.

5. **PPL statement:** "The averments in paragraph 5 of the Complaint are requests for relief to which no responsive pleading is required. To the extent a response is deemed necessary, PPL Electric denies that the Complainant is entitled to the relief requested."

Myers Note: Below in abbreviated form is what I requested in paragraph 5 and wish to address:

1. *That PPL identify for me the scientific research that shows high frequency electromagnetic radiation from smart meters is safe for occupants in their home*

2. *Exempt my home and my 11 rental properties/ tenants from smart meter installations or upgrades.*

Myers response. I am puzzled by PPL's inability to identify research showing their smart meters emit safe levels of RF-EMF radiation. PUC, by endorsing PPL's denials, is defaulting on the PUC's mission to ensure safe utility services for customers. Accordingly, I again request that:

- PPL identify and provide me with the scientific research by which they claim smart meter non-thermal RF-EMF radiation is safe for occupants in their home.

- Exempt me and my tenants in my rental properties from having smart meters installed on our homes.

Respectfully submitted:

Richard N. Myers

Dated: 29 September 2017

818-478-9283 info@qummulator.com

Study Shows Real Damage from Cell Phone Radiation

on SEPTEMBER 8, 2016 with NO COMMENTS

NTP: Cell Phone RF Breaks DNA

Consistent with Higher Tumor Counts 20 Years After Landmark Lai-Singh Study

Source:
http://microwavenews.com/news-center/ntp-comet-assay

ENCLOSURE (1)

In May, the U.S. National Toxicology Program (NTP) announced that male rats exposed to cell phone radiation developed higher rates of cancer. Soon, the NTP will explain how that might have happened.

The same RF/microwave radiation that led male rats to develop brain tumors also caused DNA breaks in their brains. Female rats —which did not have significant elevated tumor counts— had fewer DNA breaks.

All these findings are part of the same $25 million NTP project.

The NTP results provide "strong evidence for the genotoxicity of cell phone radiation," Ron Melnick told Microwave News. Melnick led the team that designed the NTP study; he is now retired. This "should put to rest the old argument that RF radiation cannot cause DNA damage," he said.

DNA breaks were also seen in the brains of the RF-exposed mice, though the increases were less pronounced than among the rats. The NTP has not yet released the tumor results for its study in mice.

The NTP project design called for a sample of rats to be sacrificed after 19 weeks of post-natal radiation exposure —five from each of the GSM and CDMA exposure groups, as well as five of the controls.1 Tissue samples for DNA assays were collected from those animals.2 The remainder of the rats continued to be exposed for the rest of the two-year cancer study.

A paper on the DNA findings has been submitted for publication and is currently under peer review, according to the NTP press office.3 Michael Wyde, who runs the NTP RF project day-to-day, presented some preliminary results at the BioEM2016 meeting in Ghent, Belgium, in June and later that month at the NTP Board of Counselors meeting. (His slides4 are here; and a video of his talk at the board meeting is here.5)

Are DNA Breaks Harbingers of Tumors?

The new results prompt this $64 question: Did the DNA breaks found at the interim kill cause or lead to the tumors that were seen at the end of the experiment?

"You can't say that the DNA assay supports the finding of increased glioma," said one NTP insider who asked not to be named. But then this person went on to add, "You can say they are consistent."

Melnick, who spent close to 30 years at NTP before retiring in 2009, offered a more direct answer: "Finding DNA damage in the brain of rats supports NTP's tumor data," he said.

We posed the same question to John Bucher, the associate director of the NTP who is in charge of the cell phone study. He declined to respond. Nor would he say whether the apparent consistency of the DNA and tumor results played a role in his decision to expedite the release of the tumor findings in May before they were published in a journal.

20 Years of War Games

The NTP's finding of DNA breaks is the latest, and perhaps most decisive, chapter in a controversy that goes back more than 20 years. In 1994, Henry Lai and N.P. Singh of the University of Washington in Seattle reported that RF radiation could damage DNA in the brain cells of rats. (They used pulsed 2450 MHz, not cell phone-like signals.) The Lai-Singh study was immediately challenged by the wireless companies as it threatened their central argument that cell phones cannot cause cancer.

Motorola led the charge. Q. Balzano, a senior Motorola executive, told us at the time that, even if the Lai-Singh experiment were to be validated, "the effects it purports to show may be inconsequential" (see MWN, N/D94, p.1). Balzano, an engineer by training, chose to sidestep the well-established principle that DNA damage can lead to cancer development and growth.

At the same time, PR operatives working for Motorola were developing a campaign to discredit the Lai-Singh work. The now-infamous "war gaming memo" was part of that effort (see MWN, J/F97, p.13).

Motorola went on to sponsor studies in Joseph Roti Roti's lab at Washington University in St. Louis. Roti Roti did not find DNA breaks (see "Two Labs at Odds over Microwaves and DNA Breaks.") As far as Motorola was concerned, Lai-Singh had been proved wrong and the matter was settled.

Nevertheless, the research continued.

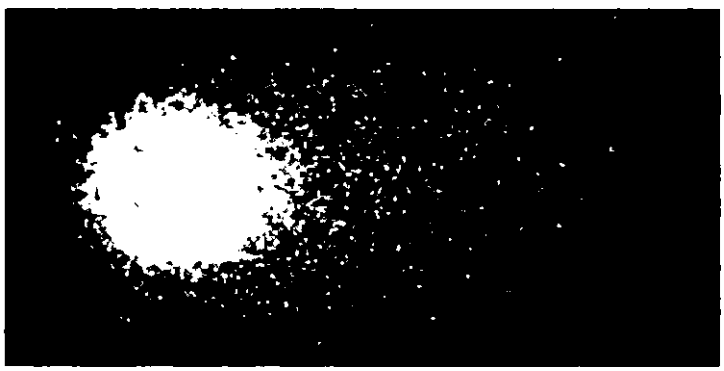
A decade later, a similar dispute arose when a team at the University of Vienna, working under the EC-sponsored REFLEX project, reported seeing RF-induced DNA breaks. Those experiments were carried out in vitro, that is, in cell cultures (see MWN, M/A03, p.7). This clash was just as nasty —perhaps more so— and led to formal accusations of fraud

and scientific misconduct. None of the charges stuck, but they left a taint on the whole enterprise. (Read about Science magazine's coverage.)

Today, no one talks much about DNA breaks anymore. Lai, who has retired from UW but still serves as the co-editor-in-chief of Electromagnetic Biology and Medicine (EBM) continues to keep close tabs on what others have been publishing. "There have been 73 studies on DNA breaks since our initial report," he told us in a recent interview, "A clear majority has found an effect similar to ours."6

The Comet Assay

All 73 studies on Lai's list measured DNA damage using what's known as the comet assay.7 The assay was developed by Singh, Lai's collaborator, close to 30 years ago.8 It can detect single- and double-strand DNA breaks, as well as other potentially genotoxic changes. The assay gets its name from the comet-like tail formed by fragments of the broken DNA. The more DNA damage, the longer and more diffuse the tail (see an example below).



The comet assay is one of the standard techniques for evaluating genetic hazards —sometimes with DNA taken from animals (in vivo) and sometimes from cell cultures (in vitro). The assay is used routinely by the NTP for testing chemicals. The OECD, for instance, has called the comet assay carried out in vivo "especially relevant" for evaluating potential cancer agents. Both the original Lai-Singh and the new NTP studies used RF –exposed rats.

"An in vivo comet assay is usually more informative than an in vitro comet assay," said Raymond Tice. Back in the 1980's, Tice helped Singh develop the comet assay. He joined the NTP in 2005, becoming the chief of its Biomolecular Screening Branch before retiring last year. He currently serves as an advisor to the NTP.

Of the 73 RF–comet assay papers that Lai has catalogued, there are 28 studies that used in vivo exposures. "Those showing DNA breaks outnumber those that don't by more than three to one," he told us (22 vs. 6).9

"I have no doubt that low-intensity RF radiation is toxic to DNA," Lai said.

NTP's Genotoxicity Results

Before the NTP study got underway, Melnick's team targeted a number of the rats' body parts, including three regions of the brain, to be tested for DNA breaks. One of these was the frontal cortex of the brain, because, as Christine Flowers, NTP's Director of Communications, told us, it is "an area in which tumors were reported in humans."

Indeed, according to Melnick, the finding of brain cancer among cell phone users, as well as the original Lai-Singh DNA experiment, prompted him to include the DNA analysis as part of the NTP protocol. (Cell phone epidemiological studies led IARC to classify RF radiation as a possible human carcinogen in 2011.)

As it turned out, the frontal cortex is where the NTP saw the most significant increases in DNA breaks. (See the color-coded slide below, taken from Wyde's presentation at the BioEM2016 meeting in June.)

The NTP later found brain tumors —gliomas— among those rats exposed for the full two years. The NTP has not specified the specific locations in the brain where the gliomas were seen. The DNA assayed in the brain was from a mix of various types, including glial cells, the kind that later turned cancerous.

Wyde has pointed that there were responders and non-responders among the male rats that were exposed to radiation.10 Only some of the animals showed DNA effects but these were large enough to move the averages up to indicate significant differences.

No DNA analysis was done for rat tissues with Schwann cells in the heart, the other site where tumors were seen after two years of exposure.11 The Schwannomas were unexpected, and only uncovered long after the samples had been collected following the interim kill.

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1. The exposures lasted 18 hours a day (power on for 10 minutes and off for 10 minutes), seven days a week. The total RF exposure time was 9 hours a day. Three different exposure levels were used: SARs of 1.5, 3 and 6 W/Kg. Details of the experiment are here.

2. The exposure of the rats began while they were still in the womb. The protocol for the mice was similar except that those exposures began at the age of six weeks. After 13 weeks or exposure, 15 mice were sacrificed and their DNA analyzed.

3. Stephanie Smith-Roe et al., "Evaluation of the Genotoxicity of Cell Phone Radiofrequency Radiation Male and Female Rats and Mice Following Subchronic Exposure," in press.

4. See also Wyde's PowerPoint presentation at the GLORE meeting in November 2013. GLORE stands for Global Coordination of Research and Health Policy on RF Electromagnetic Fields.

5. Wyde's discussion of the genotoxicity results begins at the 31:51-minute mark of the video.

6. Of the 73 studies on Lai's list, 46 (63%) found an effect and 27 (37%) did not. Lai and Singh have also shown that power-frequency (ELF) EMFs can cause DNA breaks (see their 1997 paper). Here again, Lai has catalogued the papers that followed their initial report. As of now, there have been 44 studies, of which 32 (73%) found an effect and 12 (27%) did not.

7. This summer, a team from Germany published a detailed review of the comet assay. Note that the assay is just one of a number of techniques used to measure genetic effects. Lai has also catalogued this larger RF-genotoxicity literature. At last count, in 2014, there was a total of 125 papers, of which 84 (66%) showed effects and 41 (34%) did not.

8. Singh recently wrote up his reflections on the comet assay's development, evolution and applications.

9. Not taken into account in Lai's analysis is the funding source of the studies. For a discussion of industry, military and other influences on RF–DNA research, see our "Radiation Research and the Cult of Negative Studies," written ten years ago.

10. Wyde says this in a talk to the NTP Board of Scientific Counselors in June 2016. The discussion of the genotoxicity results begins at the 31:51-minute mark of the video (also here).

11. At the same NTP Board of Scientific Counselors meeting, Linda Birnbaum, the director of NTP (& NIEHS), talked about the link between RF and the Schwannommas of the heart. In the video of the meeting, she called the association "unequivocally clear" (@43:20-minute mark) and a few minutes later described it as having a "beautiful dose-relationship." A full set of videos from the meeting are here.

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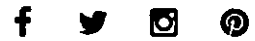

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Clinical Study

Metabolic and Genetic Screening of Electromagnetic Hypersensitive Subjects as a Feasible Tool for Diagnostics and Intervention

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Academic Editor: Beatriz De las Heras

Abstract

Growing numbers of "electromagnetic hypersensitive" (EHS) people worldwide self-report severely disabling, multiorgan, non-specific symptoms when exposed to low-dose electromagnetic radiations, often associated with symptoms of multiple chemical sensitivity (MCS) and/or other environmental "sensitivity-related illnesses" (SRI). This cluster of chronic inflammatory disorders still lacks validated pathogenetic mechanism, diagnostic biomarkers, and management guidelines. We hypothesized that SRI, not being merely psychogenic, may share organic determinants of impaired detoxification of common physic-chemical stressors. Based on our previous MCS studies, we tested a panel of 12 metabolic blood redox-related parameters and of selected drug-metabolizing-enzyme gene polymorphisms, on 153 EHS, 147 MCS, and 132 control Italians, confirming MCS altered ($P < 0.05$–0.0001) glutathione-(GSH), GSH-peroxidase/S-transferase, and catalase erythrocyte activities. We first described comparable—though milder—metabolic pro-oxidant/proinflammatory alterations in EHS with distinctively increased plasma coenzyme-Q_{10} oxidation ratio. Severe depletion of erythrocyte membrane polyunsaturated fatty acids with increased $\omega 6/\omega 3$ ratio was confirmed in MCS, but not in EHS. We also identified significantly ($P = 0.003$) altered distribution-versus-control of the CYP2C19*1/*2 SNP variants in EHS, and a 9.7-fold increased risk (OR: 95% C.I. = 1.3–74.5) of developing EHS for the haplotype (null)GSTT1 + (null)GSTM1 variants. Altogether, results on MCS and EHS strengthen our proposal to adopt this blood metabolic/genetic biomarkers' panel as suitable diagnostic tool for SRI.

1. Introduction

The term *electromagnetic hypersensitivity* or *electrosensitivity* (EHS) referred to a clinical condition characterized by a complex array of symptoms typically occurring following exposure to electromagnetic fields (EMFs) even below recommended reference levels and is followed by remission through the complete isolation [1, 2]. The most frequently claimed trigger factors include video display units, radio, televisions, electrical installations, extremely low-frequency ranges of electromagnetic fields or radio-frequencies—including the so-called dirty electricity due to poor isolation of electric wires and telephonic lines, wireless devices, and wi-fi—fluorescent lamps and low-energy lights, appliances with motors, photocopiers, microwave transmitters, and high tension power lines (reviewed in [3, 4]). EHS is characterized by a broad range of nonspecific multiple-organ symptoms implying both acute and chronic inflammatory processes, involving mainly skin and nervous, respiratory, cardiovascular, musculoskeletal, and gastrointestinal systems, in most cases self-reported in absence of organic pathological signs except skin manifestations (reviewed in [2, 5]).

Many efforts have been made to determine if a causal relationship between exposure to EMFs and claimed health symptoms does exist and to identify biologically plausible mechanisms underlying this syndrome (for review, see [2, 6, 7]). Despite the growing wealth of evidences gathered

ENCLOSURE (2)

both *in vitro* and *in vivo* on animal models, data from human case-control and double-blind trials attempting to correlate EMFs exposure and claimed symptoms, resulted so far controversial [8–10]. Nowadays, wide gaps still exist in understanding EHS, which most often remains neglected by the medical community or confined within the frame of mere psychogenic etiology [11, 12]. In the persistent lack of a proven pathogenetic mechanism for electromagnetic hypersensitivity and of clinical consensus on the few proposed diagnostic and therapeutic approaches hypothesized, no guideline for safe and efficient validated treatments has been made available until now to the patients worldwide [13, 14].

Nevertheless, the number of subjects self-reporting EHS is progressively increasing, especially in European countries [15–17], with symptoms that are often strongly disabling both professionally and socially, motivating patients to leave home and job to find rescue in "electromagnetic pollution-free" environmental settings. Because of the huge socioeconomic impact anticipated for EHS syndrome worldwide, the World Health Organization has devoted considerable attention to EHS, acknowledging this condition and recommending that people self-reporting sensitivities receive a comprehensive health evaluation [18].

Clinical similarities and frequent comorbidity between EHS and the other medically unexplained multisystem conditions of environmental origin, like *multiple chemical sensitivity* (MCS), *fibromyalgia* (FM), *chronic fatigue syndrome* (CFS), *sick building syndrome, Persian Gulf War veteran syndrome*, and *amalgam disease*, to which EHS is often associated [19, 20], have induced many authors to hypothesize that these so-called *idiopathic environmental intolerances* (IEI), more extensively also defined as *sensitivity-related illnesses* (SRI) [21], may share common genetic and/or metabolic molecular determinants connected with an impaired capability to detoxify xenobiotics (for review, see [19, 22]). Our group has evidenced for the first time a set of altered metabolic blood parameters—comprising selected redox-active and detoxifying enzymes, low-molecular weight antioxidants and oxidation markers, membrane polyunsaturated fatty acid, and proinflammatory cytokine patterns—specifically and selectively compatible with the MCS condition [23]. Recently, we contributed to the still open issue of possible genetic polymorphic patterns associated with MCS proneness, proposing a pattern of genotypic alterations of the cytochrome P450 isoenzymes CYP2C9, CYP2C19, and CYP2D6, as candidate risk factors for this specific condition, also being potentially able to discriminate different environmental-borne hypersensitivities (MCS, FM, and CFS), depending on specific combinations of their mutated alleles [24].

In this study, the working hypothesis was that EHS, as previously proposed for MCS and other environmental SRI [19, 22], may as well be based on aberrant responses to physic or chemical xenobiotic stressors through airborne or other routes of exposure, due to inherited or/and acquired dysfunction of the chemical defensive system, that is the interrelated network of phase I and II xenobiotic-metabolizing and antioxidant enzymes [19]. Based on the results of our past clinical studies on MCS, FM, and CFS, we sought to assess if similar profiles of metabolic or genetic dysfunctions could be found in those subjects self-reporting EHS phenotype. To this purpose, we measured possible alterations of a previously identified panel of twelve blood redox and lipid parameters and frequencies of selected genetic mutated variants of a set of drug-metabolizing enzymes and transcription factors with first-line roles in the detoxification of physical and chemical xenobiotics, in a group of 153 patients self-reporting EHS symptoms, co-morbid in most cases with different degrees of MCS symptoms. Results were compared to those obtained on 147 MCS patients without EHS symptoms and on a healthy control group of 132 age- and sex-matched subjects, all groups enrolled within the Italian population.

2. Materials and Methods

2.1. Patients

A group of 153 Italian Caucasian consecutive subjects self-reporting hypersensitivity to electro-magnetic fields (EHS group) as described in Figure 1 were enrolled in the study at a specialized Diagnostic Unit for Redox Balance of Istituto Dermopatico dell'Immacolata, IDI IRCCS, Rome, Italy. Age ranged from 16 to 75 years of age (mean ± SD: **46.8 ± 11.7**) and female sex represented 85.6% (131 subjects). This group was compared with a size-matched group of 147 patients (age range 19–72 y, mean ± SD: **49.6 ± 12.8**, 129F (87.8%)/18M), diagnosed with MCS, but not reporting any symptom of EHS (MCS group). MCS diagnosis was set in both groups according to Cullen's criteria [25] and modified Quick environmental Exposure and Sensitivity Inventory (QEESI) questionnaire scoring [26, 27]. Cullen's criteria refer to a disorder characterized by symptoms that involve more than one organ system and are regularly elicited by chemically unrelated compounds at doses far below those known to cause adverse effects in the general population. Symptoms typically improve considerable or heal completely after trigger withdrawal [25]. QEESI is a validated self-administered questionnaire developed as a screening tool for patients with multiple chemical sensitivity. It is based on five different scales of assessment: symptoms severity, chemical triggers, other triggers, life impact, and finally a masking index to ongoing exposures [26, 27]. A modified QEESI score of 10 common environmental exposures and 10 major symptoms enabled the diagnosis of MCS: full diagnosis ($20 \leq$ Score ≤ 30) or strongly suspected diagnosis (sMCS, suspected MCS), that is subjects fulfilling diagnostic criteria only partially ($10 \leq$ Score ≤ 20), or subjects excluded from enrollment ($0 \leq$ Score ≤ 10) [23]. As commonly seen by our group occurring in the Italian patient population, the large majority (94.7%) of the EHS group was also affected with multiple chemical sensitivity (fully diagnosed or suspected MCS).

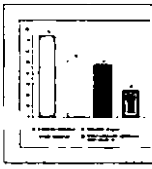

Figure 1: Electromagnetic field sources reported as symptom triggers in the group of patients self-reporting electromagnetic hypersensitivity (EHS, $n = 153$). Data are expressed as percent of patients affected on the total number of patients.

A cohort of 132 healthy age- and sex-matched subjects was enrolled as the control group (CTR group), (age range 18–74 y, mean ± SD: **45.3 ± 12.4**, 109F (82.6%)/23M), according to the established criteria of (i) absence of any clinically diagnosed disease, in particular allergic or immunologic disturbances, (ii) no drug or nutraceutical supplement since at least six weeks, at the time of blood sampling, and (iii) whole blood total production of reactive oxygen and nitrogen species (ROS/RNS) below 650 cps/μL, as determined by luminol-dependent chemiluminescent response to phorbol 12-myristate 13-acetate (PMA) [28] (Study protocol approval by Istituto Dermopatico dell'Immacolata—IDI IRCCS, Rome, Italy—Ethical Committee, n.52/CE/2010).

All patients and controls entering the study had taken no drugs or nutraceutical supplements known to interfere with metabolizing/antioxidant enzymes activity since at least six weeks, at the time of blood sampling. Nonsmokers in the patient groups were, respectively, 89.3% in EHS and 81.8% in MCS, and 85.2% in the CTR group; undetermined smoking habits were registered in 2% of EHS and 7% of MCS patients, and in 5% of

controls. Patients and controls were selected from different Italian regions in the attempt to minimize the historical genetic variability in this country [29]. Demographic information (age, race, weight, and height) and a detailed medical history were recorded in a standardized questionnaire-assisted interview, by trained medical personnel. In particular, subjects were asked to report age at onset of symptoms, agents or events likely to initiate EHS and MCS condition, if recognized, and those capable of triggering symptoms once the condition was established. No alcohol or drug abusers were present in any of the three cohorts studied.

The study protocol was reviewed and approved by the Hospital Ethical Committee Board (IDI IRCCS n.121/CE/2008). All subjects gave informed consent to personal and anamnestic data collection, blood sampling for the specific sets of analyses, and blood fraction's banking.

2.2. Reagents and Assay Kits

Majority of chemical reagents, HPLC standards, mediums, fluorogenic probes, and reverse transcription polymerase chain reaction (RT PCR) primers for gene polymorphism analyses were from Sigma Chemical Co. (St. Louis, MO, USA); kits were from Cayman Chem. Co. (Ann Arbor, MI, USA)—enzyme activities are from Qiagen (Hilden, Germany)—DNA extraction is from Applied Biosystems Inc. (Foster City, CA, USA) —polymerase chain reaction is from PCR Kit for CYPs.

2.3. Redox Studies

Complete differential blood cell counts and metabolic/genetic analyses were performed on fresh EDTA-anticoagulated venous blood of 12-hour fasting subjects. Biochemical assays were performed on plasma or erythrocytes (RBC) either immediately (coenzyme Q_{10}—CoQ_{10}) or within 72 hr. on sample aliquots stored at −80°C under argon. Whole blood luminol-dependent chemiluminescence (CL) response to phorbol 12-myristate 13-acetate (PMA) was quantified by chemiluminescence according to [28], levels of (nitrites/nitrates) by Griess reagent [30]. Plasmatic total antioxidant capacity (TAC) was determined as described previously [31]. Reduced and oxidised glutathione (GSH and GSSG) levels in erythrocytes [32], reduced and oxidized CoQ_{10}, and alpha-tocopherol levels in plasma [33] were quantified by HPLC equipped with array photodiode and electrochemical detection. Activities of CuZn superoxide dismutase (CuZn-SOD) [34], catalase [35], glutathione S-transferase (GST) [36], and glutathione peroxidase (GPX) [37] in erythrocytes were measured spectrophotometrically.

2.4. Erythrocyte Membrane Fatty Acid Profiling

The fatty acid (FA) pattern of erythrocyte membrane phospholipids was analyzed by gas-chromatography coupled with mass spectrometry with the selected ion monitoring technique, set to identify C16:0, C16:1, C18:0, C18:1*cis*, C18:1*trans*, C18:2ω6, C18:3ω6, C20:4ω6, C20:5ω3, C22:4ω3, C22:5ω3, and C22:6ω3 peaks [38]. Results were expressed as percent of the total fatty acid content of membrane phospholipids for saturated + monounsaturated FA (SFA), polyunsaturated FA (PUFA), and single representative FA of the ω3 and ω6 series.

2.5. Genotyping of Drug Metabolism-Related Enzymes

Targeted genotype analysis was performed on subgroups of EHS (n = 127) and MCS patients (n = 85) and of controls (n = 68), with reduced due to financial limitations—but yet representative—group sizes for single genotype. Genomic DNA was purified from 400 μL of human whole blood using the QIAamp DNA Blood Mini Kit (Qiagen, Hilden, Germany) according to the manufacturer's instructions. DNA was quantified spectrophotometrically at 260 nm, aliquoted, and stored at −20°C until being assayed. Genotyping and controls for eight single nucleotide polymorphisms in drug metabolism- and inflammation-related genes were carried out by real-time PCR allelic discrimination using predesigned TaqMan single nucleotide polymorphism (SNP) genotyping assays available from Applied Biosystems (Applera Italia, Monza, Italy). The polymorphisms analyzed were those of genes coding for the following: cytochrome P450 (CYP), family 2, subfamily C, polypeptides 9 and 19, namely, CYP2C9*2 (C>T, rs1799853; assay ID: C_25625805_10), CYP2C9*3 (A>C, rs1057910; assay ID: C_27104892_10), and CYP2C19*2 (G>A, rs4244285; assay ID: C_25986767_70); CYP2 subfamily D, polypeptide 6, namely, CYP2D6*4 (1846G>A, rs3892097; assay ID: C_27102431_D0) and CYP2D6*41 (C>T, rs28371725; assay ID: C_34816116_20); aryl hydrocarbon receptor (AHR) Arg554Lys variant (G>A, rs2066853; assay ID: C_11170747_20). Genotyping reactions were set up in a 96-well plate on a 7900HT fast real-time PCR System (Applied Biosystems, Foster City, CA) and were carried out in a final volume of 20 μL containing 1× TaqMan Genotyping Master Mix, 1× TaqMan-specific assay, and 10 ng genomic DNA, using thermal cycling conditions suggested by manufacturer's protocols.

The GSTP1 polymorphisms resulting in an Ile (wild type) to Val (mutant) substitution at residue 104 in exon 5 and Ala (Wild Type) to Val (mutant) substitution at residue 113 in exon 6 were determined by real time PCR using two different fluorogenic probes for the wild type and the mutant. By combining the results of the analysis of exon 5 and exon 6, the allelic setup was determined (GSTP1*A = Ile104/Ala113; GSTP1*B = Val104/Ala113; GSTP1*C = Val 104/Val113). The deletion polymorphisms for the GSTM1 and the GSTT1 genes were determined simultaneously in a single assay using a multiplex PCR approach with the amplification of the GSTM1 and the GSTT1 genes from genomic DNA and using β-globin as internal control [39].

2.6. Statistical Analysis

Statistic significance of redox and fatty acid parameters was evaluated using STATISTICA 6.0 program (StatSoft Inc., Tulsa, OK, USA). Normality of data was checked using the Shapiro-Wilk test. Since the distribution of the data in the three groups was significantly different from normal, nonparametric statistics was used. Values were presented as mean, standard error of the mean, and 1.96× standard error. Mann-Whitney U-test for independent samples was employed for comparison between case groups and controls. All reported P values are from two-tailed tests, and P values of less than 0.05 were considered to indicate statistical significance. If necessary, P values were adjusted for multiple comparisons using the Bonferroni adjustment.

The comparison of allele and genotype frequencies between patients and controls, or in-between patient cohorts, was performed using the GraphPad Prism 4 software (San Diego, CA, USA). Genotypes frequencies of patients' and control groups were compared with Fisher's exact test. A P value ≤0.05 or lower was regarded as statistically significant. Odds ratio (OR) and 95% confidence interval (CI) were used to analyze the frequency of genotypes since they provide a measure of the strength of association, compared to the control population.

3. Results

3.1. Anamnestic and Lifestyle Data

Among EMFs emissions recognized as trigger factors in the group of 153 patients self-reporting electromagnetic hypersensitivity-EHS, video display units and television were the most frequently reported sources (75% of patients), followed by mobile and landline phones (53%) and by domestic appliances (48%), while 25% of the electrosensitive population studied could not indicate a specific triggering factor (Figure 1). Potential exposure patterns to indoor EMFs can be inferred from the analysis of the percent distribution of occupational features in the EHS group, described in Figure 2.

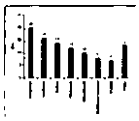

Figure 2: Occupational features in the group of patients self-reporting electromagnetic hypersensitivity (EHS, $n = 153$). Data are expressed as percentage of the total number of patients.

The percent distribution of concomitant organ diseases (comorbidities) in the EHS patient cohort, as obtained by clinical anamnestic evaluation, is presented in Figure 3(a). Body mass index (BMI) in the EHS subjects ranged between 15 and 37 (mean ± SD: **23.3 ± 5.06**), while in the group of MCS without electro-hypersensitivity there were 20% overweight patients (BMI: 25.00–29.99), 11% obese (BMI: 30.00–34.99), 2% severely obese (BMI: 35.00–39.99), 11% underweight (BMI: 18.49–16.00), and only 56% normal-weight patients (BMI: 18.50–24.99). Figure 3(b) shows the percent distribution of the other sensitivity-related illness-SRI coexisting with electromagnetic hypersensitivity in the EHS study cohort, where the 52.7% of MCS cases and the 42% of suspected MCS cases sum up clearly predominant 94.7% of multiple chemical sensitivity symptomatic subjects, within the patients self-reporting EHS symptoms.

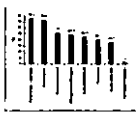

Figure 3: Distribution of specific organ comorbidities (a) and sensitivity-related illness-SRI comorbidities (b) registered in the case history of the group of patients self-reporting electromagnetic hypersensitivity (EHS, $n = 153$). Data are expressed as percentage of the total patient group, for patients affected by each single category of organ pathologies (a), and by each SRI (b), specifically multiple chemical sensitivity (MCS) or suspected MCS (sMCS), chronic fatigue syndrome (CFS), fibromyalgia (FM), and posttraumatic stress disorders (PTSD).

In Figure 4, the main classes of cutaneous symptoms or specific diseases recorded by the clinical operators through questionnaire-assisted anamnestic interview are represented, evidencing remarkable prevalence of acute dermatitis or chronic eczema conditions (both symptoms referable to different etiologies) among EHS subjects, whilst in the MCS group without electro-hypersensitivity urticaria and itching referable to (different etiologies) represented the most common findings.

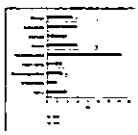

Figure 4: Skin manifestations (common symptoms and specific diseases) registered in the case histories of the groups of patients self-reporting electromagnetic hypersensitivity (EHS, $n = 153$) and of patients affected by multiple chemical sensitivity without EHS symptoms (MCS, $n = 147$). Data are expressed as percentage of patients affected by each specific class of cutaneous manifestations.

3.2. Blood Metabolic Parameters

Candidate metabolic biomarkers of electrhypersensitivity, as compared to multiple chemical sensitivity without EHS manifestations and to the corresponding values of the same blood parameters in the group of healthy controls, are shown in Figures 5–8.

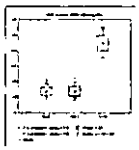

Figure 5: Metabolic redox parameters: the antioxidant/detoxification enzymatic activities of erythrocyte GST (a), GPX (b), CuZnSOD, (c) and catalase (d), in the groups of patients self-reporting electromagnetic hypersensitivity (EHS, $n = 153$), of patients affected by multiple chemical sensitivity without EHS symptoms (MCS, $n = 147$), and of control healthy subjects (CTR, $n = 132$). Values are represented as mean (□), standard error of the mean (upper and lower limits of the box), 1.96× standard error (upper and lower whiskers). Intergroup significant differences (P) are reported under each panel. RBC: red blood cells; SOD (CuZn superoxide dismutase); GST: glutathione S-transferase; GPX: glutathione peroxidase; prot.: proteins; Hb: haemoglobin.

Figure 6: Metabolic redox parameters: levels of the low-molecular weight antioxidants/cofactors, erythrocyte glutathione ((a) and (b)), and plasma coenzyme Q_{10} ((c) and (d)), in the groups of patients self-reporting electromagnetic hypersensitivity (EHS, $n = 153$), of patients affected by multiple chemical sensitivity without EHS symptoms (MCS, $n = 147$), and of control healthy subjects (CTR, $n = 132$). Values are represented as mean (□), standard error of the mean (upper and lower limits of the box), and 1.96× standard error (upper and lower whiskers). Intergroup significant differences (P) are reported under each panel. RBC: red blood cells: GSH: glutathione reduced form; GSSG: glutathione oxidized form; GS TOT: total glutathione; CoQ_{10}: coenzyme Q_{10}.

Figure 7: Selected representative parameters describing fatty acid (FA) patterns of erythrocyte membrane phospholipids, in the groups of patients self-reporting electromagnetic hypersensitivity (EHS, $n = 58$), of patients affected by multiple chemical sensitivity without EHS symptoms (MCS, $n = 54$), and of control healthy subjects (CTR, $n = 70$). (a) % saturated and monounsaturated acid (SFA) on total FA content of phospholipids, (b) % polyunsaturated fatty acids (PUFA) on total FA content of phospholipids, and (c) ratio omega-6/omega3 PUFA. Values are represented as mean (□), standard error of the mean (upper and lower limits of the box), and 1.96× standard error (upper and lower whiskers). Intergroup significant differences (P) are reported under each panel. RBC: red blood cells.

Figure 8: Selected representative omega-6 and omega-3 polyunsaturated fatty acids (PUFA) of erythrocytes membrane phospholipid fatty acids (FA), in the groups of patients self-reporting electromagnetic hypersensitivity (EHS, $n = 58$), of patients affected by multiple chemical sensitivity without EHS symptoms (MCS, $n = 54$), and of control healthy subjects (CTR, $n = 70$). (a) % C18:2ω6; (b) C20:4ω6; (c) C18:3ω6; (d) C22:6ω3 FAs, on total FA content of phospholipids. Values are represented as mean (□), standard error of the mean (upper and lower limits of the box), and 1.96× standard error (upper and lower whiskers). Intergroup significant differences (P) are reported under each panel. RBC: red blood cells. 18:2ω6 (linoleic acid), 18:3ω6 (alpha linolenic acid), 20:4ω6 (arachidonic acid), and 22:6ω3 (docosahexaenoic acid).

A set of 12 metabolic enzymatic and nonenzymatic redox parameters were measured in the blood of the 153 EHS patients, 147 patients with MCS reporting no EHS, and in the 132 healthy age- and sex-matched CTR subjects. Figure 5 shows the respective alterations of all four enzymatic activities studied in the EHS group, compared to MCS and to control values. More specifically, GST activity in erythrocytes was severely decreased in both EHS and MCS groups, compared to the CTR group ($P < 0.0001$), with no significant difference between the patients' subgroups (Figure 5(a)). A clearly uprisen erythrocyte GPX activity was registered in the EHS and more markedly in the MCS groups versus controls ($P < 0.05$ and $P < 0.001$ resp.) (Figure 5(b)), and the same was true for RBC CuZnSOD activity of MCS group versus CTR ($P < 0.0001$), while EHS patients showed only a trend towards increased activity ($P < 0.05$ versus MCS) (Figure 5(c)). Finally, Figure 5(d) shows how catalase activity rate in RBC was found decreased in both EHS and MCS patients as compared to healthy CTR, though reaching a clear-cut and elevated statistical significance only in the MCS group ($P < 0.0001$), as previously already reported [23].

Figure 6 describes the alteration of the blood levels of four redox-active low-molecular weight parameters investigated as suitable biomarkers of EHS condition, in comparison to the uncomplicated MCS and the healthy control study cohorts. The levels of both reduced (GSH) and oxidized (GSSG) glutathione forms (data shown in the figure only for GSH (Figure 6(a))) were strongly decreased in the RBC of EHS and MCS environmentally sensitive groups as compared to CTR subjects (GSH: $P < 0.0001$ for both groups; GSSG: $P < 0.001$ and $P < 0.0001$, resp., for EHS and MCS), although decrease scores for both glutathione forms were inferior in the EHS than in the MCS subgroup (GSH: $P < 0.05$; GSSG: $P < 0.001$ in EHS versus MCS). Also the ratio of GSSG/GSH (Figure 6(b)), indicating the relative oxidation grade of the erythrocyte glutathione marker, displayed a trend to elevation in the two patient subgroups versus control, although data were too scattered to reach any statistical value.

The plasmatic levels of coenzyme Q_{10} and alpha-tocopherol displayed a similar trend-to-depletion in both patient subgroups versus controls. Figure 6(c) reports results of ubiquinol ($CoQ_{10}H_2$, the reduced form of coenzyme Q_{10}) analysis which, together with levels of total CoQ_{10} (reduced + oxidized forms) and of alpha-tocopherol (both groups of data not shown)—showed similar trend of reduction for EHS as well as MCS subgroups, as compared to CTR group, though lacking statistical significance. Indeed, we found a higher percent coenzyme Q_{10} oxidation (ratio oxidized-CoQ_{10}/total-CoQ_{10}), significant versus CTR at $P < 0.001$ in EHS patients, not confirmed for MCS patients, as reported in Figure 6(d).

Although a trend-to-increase in the values of whole blood chemiluminescence (CL) and to decreased levels of plasmatic total antioxidant capacity (TAC) were recorded for both patient subgroups compared to controls, differences were unable to reach any statistical significance (data not shown). The increase of NO_2^-/NO_3^- plasma levels of MCS patients obtained in our previous study [23] was not confirmed in this new MCS subgroup, as well as in the EHS group of the present study, respectively, averaging or being inferior to control values (data not shown).

Since the majority of the above metabolic data were similar for EHS and MCS subgroups, the costly and time-consuming analyses of fatty acid profiles were carried out on a more limited subgroup of patients who fully corresponded to all diagnostic criteria. Representative fatty acid profiles in the phosholipid fraction of the erythrocyte membranes of EHS ($n = 58$), MCS ($n = 54$) and CTR ($n = 70$) patients are shown in Figures 7 and 8. The comparative analysis of the fatty acid (FA) profiles in the erythrocyte membranes of the 3 studied groups showed elevated levels of the saturated and monounsaturated fatty acid fraction (SFA) for both environmental-sensitive patients (Figure 7(a)) and correspondingly depleted levels of the polyunsaturated fatty acid fraction (PUFA) (Figure 7(b)), with both parameters statistically significant at $P < 0.05$ for MCS patients versus controls, whilst the EHS group differed sensibly from MCS in displaying only a mild trend-to-alteration of fatty acid patterns versus control group. In detail, the percent levels of the omega-6 FA linoleic (18:2ω6), alpha linolenic (18:3ω6), arachidonic (C20:4ω6), and the omega-3 FA docosahexaenoic (C22:6ω3) (Figures 8(a)–8(d)) were lower than control values in both EHS and MCS cohorts, although the clear-cut statistical significance registered for the MCS group ($P < 0.05$–0.001 for all 4 parameters) was confirmed in EHS patients only for linoleic acid fraction ($P < 0.001$) (Figure 8(a)). Finally, the range of the ω6/ω3 PUFA ratio in electrosensitive subjects practically equalled that of controls, whilst MCS patients showed significantly increased values versus both CTR ($P < 0.001$) and EHS group ($P < 0.05$), as reported in Figure 7(c).

3.3. Genetic Parameters

The main results of genotype analysis for a selected panel of detoxifying enzymes, obtained on limited subgroups of EHS, MCS, and controls, are illustrated in Table 1. Having previously demonstrated in the MCS population a significantly higher-versus-CTR frequency of the homozygous mutated *1 allele and a CYP2C19*2 heterozygous genotype *1/*2, with a lower frequency of the *2 allele in the homozygous and heterozygous forms [24], we here confronted the panel of previously investigated CYP isozymes in the EHS versus the already studied MCS cohort previously studied. Genotype frequencies for cytochrome P450 CYP2C19 SNP variants in EHS and MCS patients' groups showed that the CYP2C19*1/*1 and the CYP2C19*1/*2, *2/*2 genotypes differed with statistical significance at $P = 0.003$ between EHS ($n = 29$) and MCS ($n = 85$) groups. The other gene polymorphisms of CYPs studied (CYP2C9 and CYP2D6), as well as the aryl hydrocarbon receptor (AHR) variant Arg554Lys, displayed similar frequency distributions for EHS and MCS patients (data not shown).

Table 1: Statistical analysis of genotype distribution of cytochrome P450 (CYP) isoenzymes in EHS-patients self-reporting electromagnetic hypersensitivity ($n = 29$) versus MCS-multiple chemical sensitivity patients without EHS ($n = 85$) and of glutathione S-transferase P1 (GSTP1), glutathione S-transferase M1 (GSTM1), and glutathione S-transferase T1 (GSTT1) isoenzymes in CTR-healthy control subjects ($n = 68$) versus EHS-patients ($n = 127$).

Genotype frequencies of the glutathione S-transferase (GST) isoenzymes GSTP1, GSTM1, and GSTT1, previously found not significantly differing in MCS versus healthy control populations [23], were compared in 127 EHS patients versus 68 CTR subjects. No statistically significant differences were observed for GSTP1 in the frequency of the GSTP1*A, GSTP1*B, or GSTP1*C homozygous and heterozygous variants between the EHS patient and control groups (Table 1).

The statistical analysis of the distribution of GSTM1 and GSTT1 isoenzymes showed no statistical difference in homozygous + heterozygous and null genotype variants neither in GSTM1 nor in GSTT1, when analyzed independently. Conversely, the combined GSTM1 (*0/*0) + GSTT1 (*0/*0) null genotypes differed significantly (13% versus 1.5%, resp.), with $P = 0.007$, in EHS patients versus CTR subjects, conferring to this association of gene variants 9.7 times higher risk (OR: 95% C.I. = 1.3–74.5) of developing EHS compared to other GSTM1 and GSTT1 combinations of genotypes examined (Table 1).

4. Discussion

Till now, no causal relationship between electromagnetic fields exposure and onset of clinical symptoms has been clearly proven. Nevertheless, the term electric hypersensitivity is currently used both by patients who claim health effects of environmental electromagnetic pollution and doctors to define patient clusters of symptoms [40]. Most of the evidences about altered organic parameters due to EMF exposure have been so far obtained on cell or animal models. Very few human studies investigated possible organic parameters distinctive of the hypersensitivity to electromagnetic stressors ([41, 42]; for review, see [2]).

Main difficulties for clinical studies' implementation arise from the necessity to deal with patients in a protected environment, sheltered from EMF sources and also free of chemical barriers, since the majority of electrosensitive patients are also intolerant to a multiple array of chemical triggers [43]. Indeed, in the group of 153 EHS subjects enrolled for this study, 145 were also affected at different degrees by MCS symptoms (Figure 3(b)). The experimental group of EHS patients was exposed by lifestyle to the most common electromagnetic sources deriving mainly from indoor or outdoor urban electromagnetic pollution and no heavy professional exposure in industrial settings was recorded in the group (Figure 2). In addition, EHS patients shared with MCS patients the sensitivity to the most frequent organic chemical triggers initiating and sustaining MCS.

Another relevant issue complicating human studies is connected with the difficulties encountered in provocation studies, aimed at connecting the electromagnetic trigger with electrohypersensitivity symptoms' onset. These difficulties arise generally from the necessity to standardize types and dosages of EMF sources, from the broad qualitative and quantitative range of individual multiorgan responses to trigger, difficult to measure objectively, and also from heavy psychoemotional bias factors affecting experimental protocols and their repeatability [44, 45]. Notably, provocation studies commonly proposed as the main milestone for EHS assessment and validation are based on the questionable assumption that the individual capability to directly perceive EMFs at low or very low intensities below established toxicological thresholds, claimed by EHS subjects in analogy with MCS odor perception, may be *conditio sine qua non* for EHS symptom manifestation [40, 46]. Waiting for a consensus on a standardized methodology for an objective clinical assessment of electro-sensitivity, our present work referred to self-reported EHS as registered in the course of the anamnestic evaluation performed by trained medical personnel.

Data concerning the involvement of organic causes connected with chronic oxidative damage as a key factor in the induction and perpetuating of symptoms in functional SRI syndromes has been growing in the last decade (reviewed in: [22]). Our previous studies provided evidence of a specific and peculiar metabolic disease-marker profile in multiple chemical sensitivity, the prototype of all medically unexplained environmental illnesses so far described. In fact, moving from published data accounting for the altered redox balance in favor of a prooxidative and proinflammatory state in patients with fibromyalgia or chronic fatigue symptoms [7, 22], we identified a profile of 12 specifically altered blood parameters connected with systemic oxidative stress and impaired detoxification, in a representative sample of the Italian population fully or partially complying with MCS diagnosis [23]. In the same line, the present study was conceived to verify if analogous alterations of this pattern of MCS reliable organic biomarkers may also apply to EHS condition, in order to seek evidences of the organic etiology of this group of environmental sensitivity disorders and provide the clinicians with suitable tools for laboratory diagnosis and treatment follow-up.

The profiles of metabolic parameters' alteration observed in EHS subjects were comparable to those of the "pure MCS" group, though generally less pronounced (Figures 5–8). Similarly to those MCS patients self-reportedly nonelectrosensitive, the EHS cohort showed a highly significant-versus-control decrease in the erythrocyte GST activity and an increase in GPX activity levels (Figure 5), coupled with a marked decrease of GSH levels (Figure 6). Again in line with MCS, EHS group showed a trend to the increase in erythrocyte CuZnSOD activity and to the depletion of the main lipophilic antioxidants in plasma-reduced coenzyme Q_{10} and alpha-tocopherol (vitamin E) (Figures 5 and 6). The most striking difference between the two patient subgroups was recorded, instead, for erythrocyte catalase. Enzymatic activity was in fact only slightly and not significantly, reduced in EHS as compared to control values, while the highly significant ($P < 0.0001$) reduction recorded in the MCS group (Figure 5) confirmed our previous reports, validating the relevance and selectivity of this blood metabolic marker specifically for the MCS condition [23], being previously confirmed also in those patients only partially complying with MCS criteria (suspected MCS group).

We also calculated the ratios between oxidized and reduced forms of glutathione and coenzyme Q_{10} as suitable indicators of a systemic oxidative and proinflammatory *status* [47]. Relative oxidation of the two redox molecules was increased, though not significantly, in both EHS and MCS groups versus CTR (Figure 6). Interestingly, only in electrosensitive subjects, the oxidized/total CoQ_{10} ratio reached statistical significance ($P < 0.001$) versus normal values. Due to its marked lipophilicity, coenzyme Q_{10} is essential, along with alpha-tocopherol and squalene, for skin protection against oxidizing environmental physicochemical stressors, and it is able to efficiently reach the skin from the blood compartment [48, 49]. The elevated oxidation of plasma coenzyme Q_{10} observed in EHS appears to be consistent with the higher frequency of cutaneous involvement in EHS (40.7%) symptoms self-reported by our experimental group (Figure 3(a)), as compared to the minor relative clinical relevance assessed in the classical MCS condition, previously described [23]. Accordingly, Figure 4 shows how the prevalent skin symptom, in the EHS but

not in the MCS cohort, resulted in being acute or chronic dermatitis (eczema), a group of inflammatory skin diseases where systemic and local lipophilic antioxidant depletion is strongly implicated [48].

A second parameter proved to be significantly different ($P < 0.05$) between EHS and MCS groups that is the ratio omega-6/omega-3 polyunsaturated fatty acids in the erythrocyte membrane phospholipid fraction (Figure 7(c)). The ratio showed a remarkable elevation versus CTR in favor of the more proinflammatory $\omega 6$ PUFA in the MCS group ($P < 0.001$), while EHS values were instead nearly overlapping CTR values, data that appears consistent with the overall less pronounced prooxidative and proinflammatory state evidenced in EHS versus MCS, from the whole pattern of redox parameters investigated in this study. Again, this molecular marker difference between the two environmental hypersensitivities can possibly be connected with the clinical setting, where, for example, a higher frequency of pathological obesity with metabolic syndrome is observed in MCS [50], whereas EHS condition features a milder chronic inflammatory *status* [51].

As a whole, MCS values of all metabolic parameters studied confirmed our previous results obtained in a larger cohort of 226 MCS + sMCS patients [23], highlighting the reliability of the selected redox-marker panel on this additional study cohort. With two exceptions, (a) erythrocyte CuZnSOD activity, now found significantly increased ($P < 0.0001$) in MCS versus CTR (Figure 5(c)) whilst nonsignificant in the first study, and (b) plasma nitrites/nitrates values, significantly elevated in the previous study MCS cohort [23], a finding not confirmed in the present study (data not shown). These differences may possibly be related to the extreme individual genetic and metabolic variability characterizing MCS populations, even within the same ethnic, geographic, lifestyle, and cultural setting, which represented one of the difficulties facing SRI human studies [52].

The question as to whether genetic background may determine a proneness to environmental hypersensitive syndromes remains still unanswered, from the time of the first pioneer studies on multiple chemical sensitivity [53, 54], followed by a wealth of extensive investigations on MCS, FM, and CFS western populations worldwide [19, 23, 55]. We attempted to contribute to this unresolved issue of utmost relevance for diagnostic purposes in these poorly defined clinical settings. In previous works, we had investigated gene and allele frequencies of selected polymorphisms of a wide array of phase I and II xeno- and endobiotic metabolizing enzymes, GST (M1, T1 and P1), UDP-glucuronosyl transferase (UGT), and cytochrome P450 (CYP) variants belonging to the CYP2C9, CYP2C19, CYP2D6, and CYP3A5*3 isoenzymes. After a first study not showing any significant prevalence of the studied CYP, UGT, and GST gene polymorphisms in a group of 110 MCS patients [23], we proceeded to a second investigation on a clinically better characterized MCS group of 156 patients and of 113 matched controls, where we identified significantly ($P < 0.05$–0.0001) higher frequencies versus CTR for the polymorphisms CYP2C9*2, CYP2C9*3, CYP2C19*2, CYP2D6*4, and CYP2D6*41, confirming other studies indicating these genetic variants as a risk factor for SRI [24]. Starting from these results, in the present study, genotyping for the CYP2C19 single nucleotide variants showed that the frequency of the homozygous mutated *1 allele was significantly higher in EHS, than in MCS cases, whilst the *2 allele in the homozygous and heterozygous forms was less frequent in EHS than in MCS ($P = 0.003$) (Table 1). Moreover, our previous work had shown that the CYP2C19*2 heterozygous genotype *1/*2 was significantly more frequent ($P = 0.05$) in MCS cases, not only versus controls but also versus FM + CFS cases [24]. The same study showed for the first time that the Arg554Lys mutated variant of the aryl hydrocarbon receptor-AHR gene did not reach significant differences in distribution between SRIs and controls when analyzed alone but showed in specific haplotype combinations with CYP variants promising implications for in-between group discrimination within SRI comorbidities, namely, MCS versus sMCS and FC + FM versus controls [24]. In the present work, we were able to confirm the absence of significant differences for AHR genotype between EHS and CTR groups (data not shown).

Having previously found no significant difference between MCS patients and controls, in the distribution of GST isoenzyme genotypes [23], in the GST study we now compared EHS and healthy controls. Differently from our previous results on MCS, we here identified a mutated (null) allele combination of GSTT1 and GSTM1 variants able to predict risk of developing EHS by a 9.7 fold versus CTR (Table 1).

Taken together, our genetic results obtained on a number of cases due to be enlarged in the studies to come, although being far to be conclusive on such a controversial matter, can at least contribute additional indications to the complex mosaic of genetic risk factors in environmental hypersensitivities, still waiting to be correlated with individual metabolic phenotypes.

The outcomes of this work confirmed, in the whole, our previous results on MCS and provided additional evidences for the validity of the selected panel of metabolic blood parameters also in the self-reported EHS condition. Further developments must necessarily include a more objective and standardized classification of individual electromagnetic sensitivity scores, to conclusively assess the proposed parameters as a distinctive and specific panel of disease biomarkers for EHS. Our findings will hopefully contribute, in combination with the so-far putative genetic-risk factors, a better molecular definition of environmental-borne sensitivity-related illnesses and a tool to discriminate single SRI comorbidities, based on sufficiently proven molecular evidences able to gain clinical consensus.

Conflict of Interests

The authors declare that they have no conflict of interests.

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Comments on the Draft Report
by the California Council on Science and Technology
"Health Impacts of Radio Frequency from Smart Meters"

by Daniel Hirsch[1]
31 January 2011

Abstract

The draft report by the California Council on Science and Technology (CCST) does not appear to answer the questions asked of it by the requesting elected officials. Furthermore, rather than being an independent, science-based study, the CCST largely cuts and pastes estimates from a brochure by the Electric Power Research Institute, an industry group, issued some weeks earlier. The EPRI estimates appear incorrect in a number of regards. When two of the most central errors are corrected – the failure to take into account duty cycles of cell phones and microwave ovens and the failure to utilize the same units (they should compare everything in terms of average whole body exposure) **the cumulative whole body exposure from a Smart Meter at 3 feet appears to be approximately two orders of magnitude higher than that of a cell phone, rather than two orders of magnitude lower.**

It is strongly recommended that CCST revise its Draft Report and conduct actual measurements of cell phone, microwave oven, and SmartMeter RF cumulative whole body power densities. If measurements aren't made, then rigorous calculations correcting for cell phone and microwave oven duty cycles and whole body exposures should be made.

A summary figure below shows how rough estimates of the effect of those corrections suggest SmartMeters may produce cumulative whole body exposures far higher than that of cell phones or microwave ovens.

[1] The assistance of two UCSC student research assistants, Bailey Hall and Catherine Wahlgren, in the preparation of this review is gratefully acknowledged.

ENCLOSURE (3)

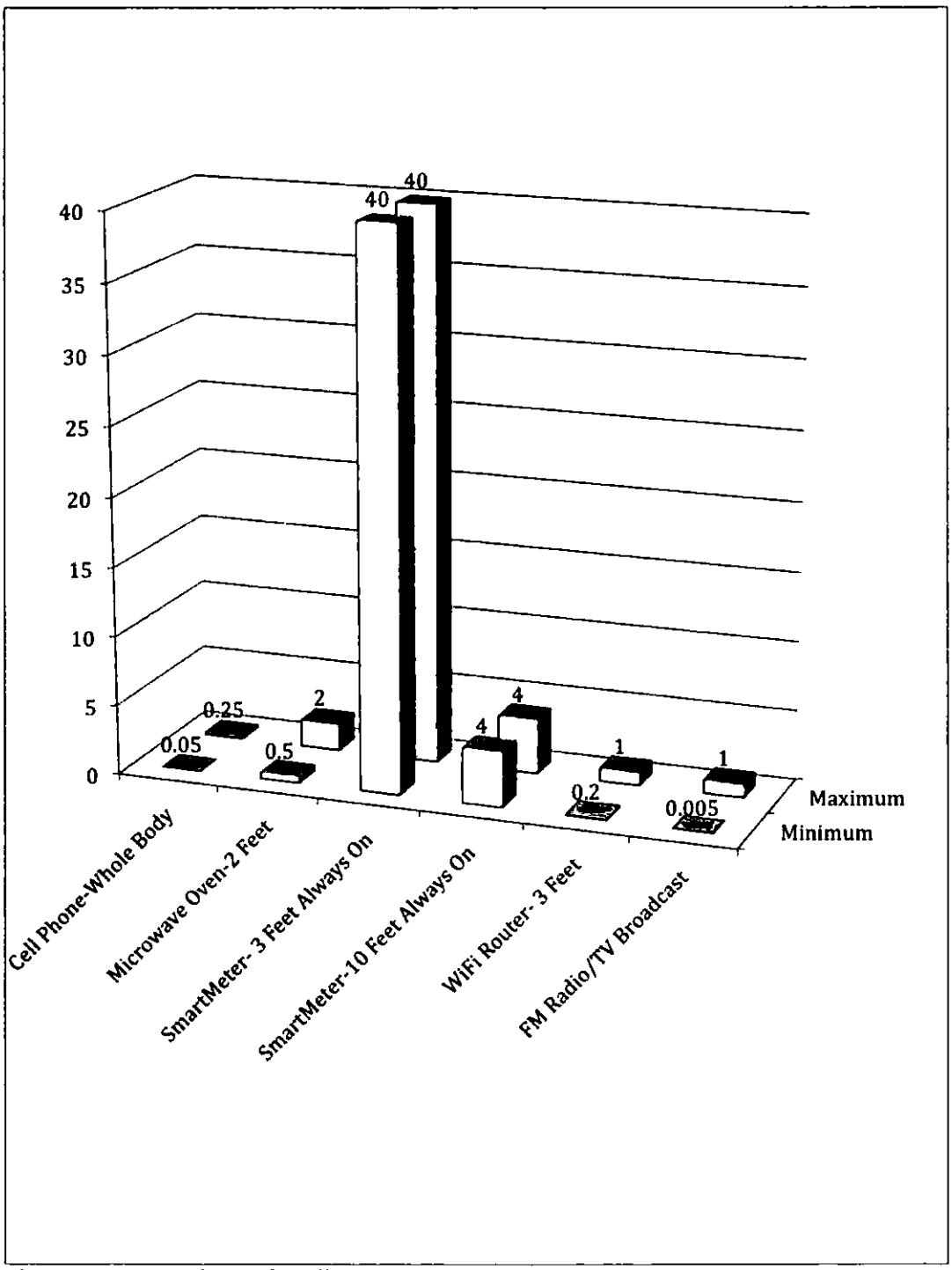

Figure A. Comparison of Radio-Frequency Levels to the Whole Body from Various Sources in $\mu W/cm^2$ over time [corrected for assumed duty cycle and whole body exposure extrapolated from assumed cell phone dose at ear].

On 30 July 2010 Assemblymember Jared Huffman requested that CCST undertake an "independent, science-based study" of two questions: "whether FCC standards for SmartMeters are sufficiently protective of public health taking into account current exposure levels to radiofrequency and electromagnetic fields, and further to assess whether additional technology specific standards are needed for SmartMeters and other devices that are commonly found in and around homes, to ensure adequate protection from adverse health effects."

Unfortunately, the Council draft report answers neither question.

In September, Assemblymember William Monning and Mill Valley Mayor Stephanie Moulton-Peters joined in the request, asking in particular that CCST review the central issue associated with the current FCC standards, which are decades old and based solely on protecting against prompt thermal effects (heating of tissue)—that they fail to take into consideration long-term and cumulative exposures to these devices and potential non-thermal health impacts (e.g., latent cancers).

Again, the Council's draft report provides little if any useful information or analysis of this matter. There is no mention or analysis of the specific studies that have suggested, for example, a cancer effect from RF exposure such as the large, international study funded by the cell phone industry, the Interphone study, that found a significant increase in brain cancers in people who used cell phones half an hour a day for ten years. Given the long latency period generally for solid cancers, such a finding gives pause as to what might be seen over the long term. Some other studies have suggested an increased risk of brain cancer on the side of the head where the cellphone is normally used. Other studies, however, have not found an effect. Given the nature of the request from the elected officials for a review of this critical scientific issue—whether there is the potential for non-thermal health effects from cumulative, long-term exposure to RF radiation—one would have hoped that there would have been a more detailed analysis of this question in the report.

The report is candid, however, that at present the issue is unresolved. But it goes on to then say there is no basis for changing the FCC standards which are based only on prompt, thermal effects. One could equally well say there is no basis for maintaining the FCC standards, given the uncertainties about latent, non-thermal effects.

What the CCST draft report does focus on, however, is the relative exposure from SmartMeters compared to other RF-emitting devices in common use. Here, again, the draft report disappoints. The elected officials cited claims made by the electric utility industry regarding safety of SmartMeters and purportedly relative low exposures compared to other common devices and requested "an independent, science-based study."

However, the CCST draft report does not appear to include much if any independent work on the subject but rather merely pastes in a table taken from an 8-page pamphlet released a few weeks earlier by the Electric Power Research Institute (EPRI), an advocacy group for the

electric power industry.[2] This EPRI table and the graph made from it constitute the core of the CCST report, and is reproduced here as Figure 1.

The EPRI pamphlet is not a peer-reviewed scientific study. It is a brief item for an advocacy group that is supported by industry. If the elected officials wanted the industry's views, it would have asked for them. Instead, it wished an independent, science-based study by an entity without the kinds of conflicts of interest EPRI has on this matter. But the CCST draft report is basically simply a cut-and-paste job from the EPRI brochure.

Note also that the estimate for exposure from a single SmartMeter contained in the EPRI item and repeated in the CCST draft is not a measured value but estimated—how is not made clear. EPRI's measurements were for a bank of ten SmartMeters; it didn't measure one alone but somehow estimated for it, despite the difference in how exposure falls off from one versus ten. The latter is inverse of the distance, the former inverse square of the distance. One presumes the electeds wanted actual measured values from an independent source, not a calculated value from the electric industry, without even an explanation of how it is was calculated and without independent verification.

CCST does correct one error made in the EPRI brochure whereby it reduced the presumed power density estimates for the SmartMeter by duty cycles of 1 and 5%. CCST rightly indicated that future duty cycles could be much higher as "new applications and functionality are added to the meter's communication module in the future." For this reason, it assumed a 100% duty cycle in its calculations.

HOWEVER, CCST did not correct numerous other apparent errors from the EPRI brochure when it adopted EPRI's values. For example, for cell phone exposures, CCST did not correct for the presumed duty cycle of the cell phone (which CCST indicates on average is 1%). Nor did it convert the EPRI cell phone power density estimate into comparable units. EPRI (and thus CCST) compared a *whole body average* exposure to SmartMeter radiation to *peak exposure to the ear* for the cell phone. One needs to compare apples and apples, or whole body exposures to whole body exposures. Comparing the peak dose to the ear from a cell phone, when the rest of the body gets vastly less radiation, with a whole body exposure where all organs get roughly the same dose from a SmartMeter, doesn't seem appropriate. If there is a cancer effect, it is likely associated with the total RF energy the body receives.

Similar apparent errors were made in the comparison to microwave ovens. Again, the duty cycle of the microwave oven is ignored. It is used perhaps fifteen minutes a day, and it is unlikely people are 2 feet away from the device for the full time it is on. Its "down time" must be included if one is looking, as requested by the elected officials, at potential cumulative, long-term exposures.

[2] The EPRI brochure was apparently released on November 17, providing little if any time for serious review of it by CCST prior to the release a few weeks later (with the holidays intervening) of the CCST report on which it was based.

[Additionally, the values given for microwave oven exposures by EPRI and adopted without changed in the CCST draft report seem questionable. Three references are given in the EPRI report, although for which claim each applies is not made clear. The first reference, the ICNIRP report, does not in fact give measured values for microwave ovens, but instead reports what the legal limit for leakage is, generally reported to be orders of magnitude above what typical exposures from microwave ovens really are. The second reference is to a 1978 paper by PG&E's consultant, RA Tell. That paper CCST has not made available for review, but it is over three decades old, and thus of little relevance to today's microwave ovens. The third reference is merely to a personal communication with Tell, without any information as to the content of that communication. When one checks the values reported by EPRI and uncritically adopted by CCST, it appears that the first value, 5 mW/cm^2 at 2 inches from the device, is in fact not a measured value of typical exposures but the vastly higher legal limit for leakage. The literature in fact indicates that 50% of microwave ovens produce less than 0.062 mW/cm^2 at 5 cm, or two orders of magnitude below the value reported by EPRI and reproduced by CCST without question. See, e.g., R, Mathes, "Radiation Emission from Microwave Ovens," *Journal of Radiation Protection,* Vol. 12, No. 3, September 1992. One presumes the leakage rate has been reduced even further since then.]

One recognizes that if one is comparing to FCC existing standards based solely on acute, thermal effects that duty cycle might be treated differently. But if there is a cancer effect, which is what the electeds asked CCST to study, a likely key aspect of the dose-response relationship is the cumulative whole body dose. For ionizing radiation, about which I have spent much of my career, the determining factor is largely how much radiation energy the body has absorbed. [There are of course other factors, such as the relative biological effectiveness (RBE) of different types of ionizing radiation and varying sensitivity of different organs.) So, if the question were how does SmartMeter and cell phone RF radiation compare to FCC limits, duty cycle may be treated in a different fashion. But since the question is what if FCC limits, based solely on thermal effects, may be inadequate to protect against cancer and other non-thermal effects, then the duty cycle—which determines the cumulative total exposure received—and whole body exposure must be factored in. My fundamental recommendation is that the draft report should be revised to correct for these two factors.

I have taken the liberty, with the help of two student assistants, to demonstrate the potential impact of some of these corrections.

Figure 1 is simply the CCST Figure 1, which in turn was largely taken from the estimates in the EPRI pamphlet. Units were simply converted by CCST from mW/cm^2 to $\mu W/cm^2$ and it corrected the duty cycle for the SmartMeter, otherwise the data are unchanged from EPRI's estimates. One will note that the estimated exposure from the cell phone is just to the ear, in direct contact with the cell phone, whereas the other comparisons, including the SmartMeter, are for whole body exposures, and that the duty cycle of the cell phone and microwave oven were not corrected. In other words, the chart compares a SmartMeter that is always on with a cell phone or microwave oven when they are being used, even though 99% of the time they are not in use. This overestimates the cumulative exposure by a factor of 100 for the cell phone and microwave oven, and dramatically skews the comparison.

Figure 2 fixes the error regarding duty cycle for the cell phone and microwave oven, markedly altering the comparison. The minimum cumulative exposure over time from the SmartMeter at 3 feet is 80 times the minimum cumulative exposure from the microwave oven and four times the minimum cumulative exposure from the cell phone, for example. This does not involve any correction of the while-on exposure values for either the cell phone or microwave oven, only the duty cycle factor.

Figure 3 provides a very rough approximation of the correction of the cell phone at the ear estimate to a whole body estimate so it is comparable to the whole body estimate for the SmartMeter. *It should be stressed that neither this estimate nor that in Figure 4 using a different approach is intended to be a definitive figure, but is intended to be exemplary of the kind of change to the comparison a detailed analysis may produce. It is my recommendation that CCST carefully measure, or at minimum thoroughly calculate, the average power density over the whole body from a cell phone held at the ear. We here have made two very rough estimates just to make the point what a far more detailed analysis may show.*

The value used for the peak cell phone power density for a cell phone held to the ear in the CCST draft report is taken directly from the EPRI pamphlet, without apparent independent review or correction. According to p. 6 of the EPRI pamphlet, the value it gives apparently is not a measured value but an estimate. How the estimate was arrived at is not detailed in the brochure. All that is said is in footnote 1, "Based on a 3-inch 250mW antenna emitting in a cylindrical wavefront." A quick calculation to try to reproduce what EPRI must have done indicates that if it merely assumed that all of the energy from a 250mW cell phone was transmitted by holding directly against the ear into a circular area with a 3 inch diameter, the power density in that small circular area around the ear would be 5 mW/cm^2. That is precisely the upper value given by EPRI in its table. We don't know if that is what EPRI did, since it doesn't tell us what it did and CCST does not appear to have tried to confirm the asserted value. But in any case, 5 mW/cm^2 from a 250mW cell phone would indeed appear to require that that power be deposited solely in that very small circular area.

Averaging over the full potentially exposed surface area of the body (presuming only half the body surface could be exposed to the cell phone from any one angle), the whole body exposure would be approximately on average 0.25 mW/cm^2 given the maximum value to the ear of 5 mW/cm^2 put forward by EPRI and the CCST draft report and correcting as well for the duty cycle. **The SmartMeter thus would produce 160 times more cumulative whole body exposure than the cell phone assuming this estimate for whole body exposure.** This is shown in Figure 3.[3]

[3] In these graphs we have used the values for a microwave oven at 2 feet put forward by EPRI and repeated by CCST even though, as discussed above, they appear questionably high. Note that measured values indicate typical measured microwave oven RF fields 5 cm from the oven are in the range of 0.062 mW/cm^2, whereas the EPRI estimates used by CCST are for comparable values 2 feet away, which, if the exposure were drop by inverse square of the distance, should be very much lower. It is unclear whether EPRI is actually referring to measured values or to the legal limits, the latter being irrelevant in this context.

Since the EPRI estimate for cell phone peak power density at the ear is unexplained as to its derivation, we have also made a very rough estimate of whole body exposure from a cell phone from an independent line of calculation. Taking the values EPRI (and thereby CCST) put forward for exposure at three feet from a 250 mW SmartMeter, and noting that EPRI assumed the cell phone would also be 250mW, one can make a rough estimate of power density for the whole body from a cell phone held at the head. The exposure at one's waist would be approximately three feet from the source, just as in the assumed case of the SmartMeter. Presuming that the dose falls off as the inverse square of the distance, a very rough estimate of power density averaged over half the surface of the whole body, and taking into account duty cycle, yields a cumulative cell phone whole body power density of roughly 0.75 $\mu W/cm^2$. **Using this way of estimating suggests the SmartMeter would produce 50 times the cumulative whole body exposure as a cell phone.** The results of this comparison are found in Figure 4.

We are here using the duty cycles proposed by CCST itself in its draft report. We recognize other duty cycles can be considered. Perhaps one should presume maximum duty cycle in the future for SmartMeters, when all additional features are incorporated, might be only 50%, for example. But other factors also need to be considered, including exposures from banks of SmartMeters attached to an apartment building, and the exposure from all the devices within a home that are planned to be constantly communicating by RF with the SmartMeter.[4]

It is strongly recommended that CCST revise the report and perform actual measurements. At minimum, revised calculations that correct for duty cycle and cumulative whole body exposure should be conducted.

[4] It is noted that EPRI claims a diminished dose in back of a bank of SmartMeters, but it is unclear that that claim can be relied upon. The particulars of the specific test done by EPRI, in connection with the manufacturer of the devices (who has an obvious interest in findings suggesting safety), are not spelled out. Furthermore, it is unclear how the SmartMeter can communicate with devices inside the home—the key purpose—if the back of the device blocks most of the signal from getting through.

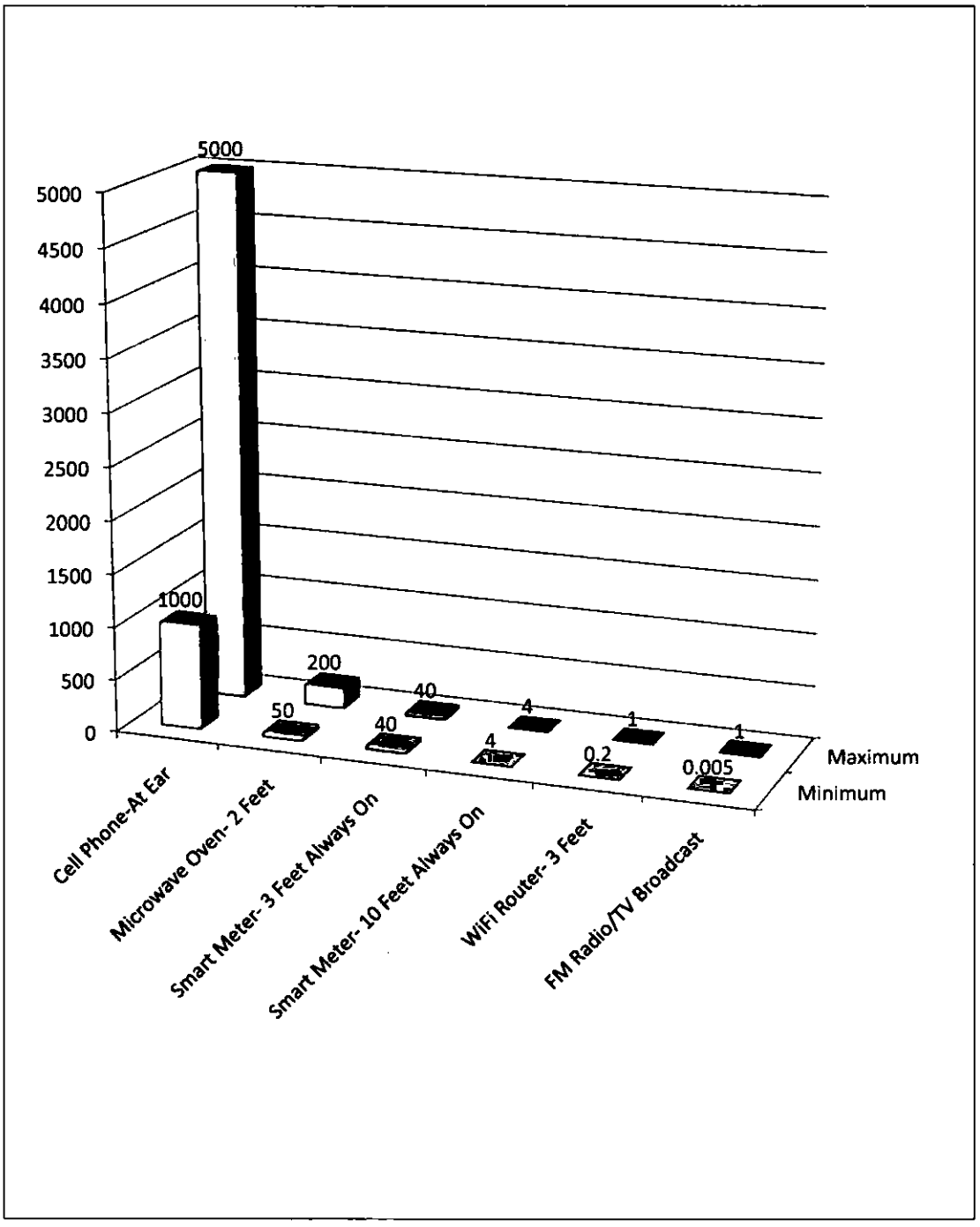

Figure 1: Graph from CCST Report in $\mu W/cm^2$—uncorrected for whole body exposure or duty cycle

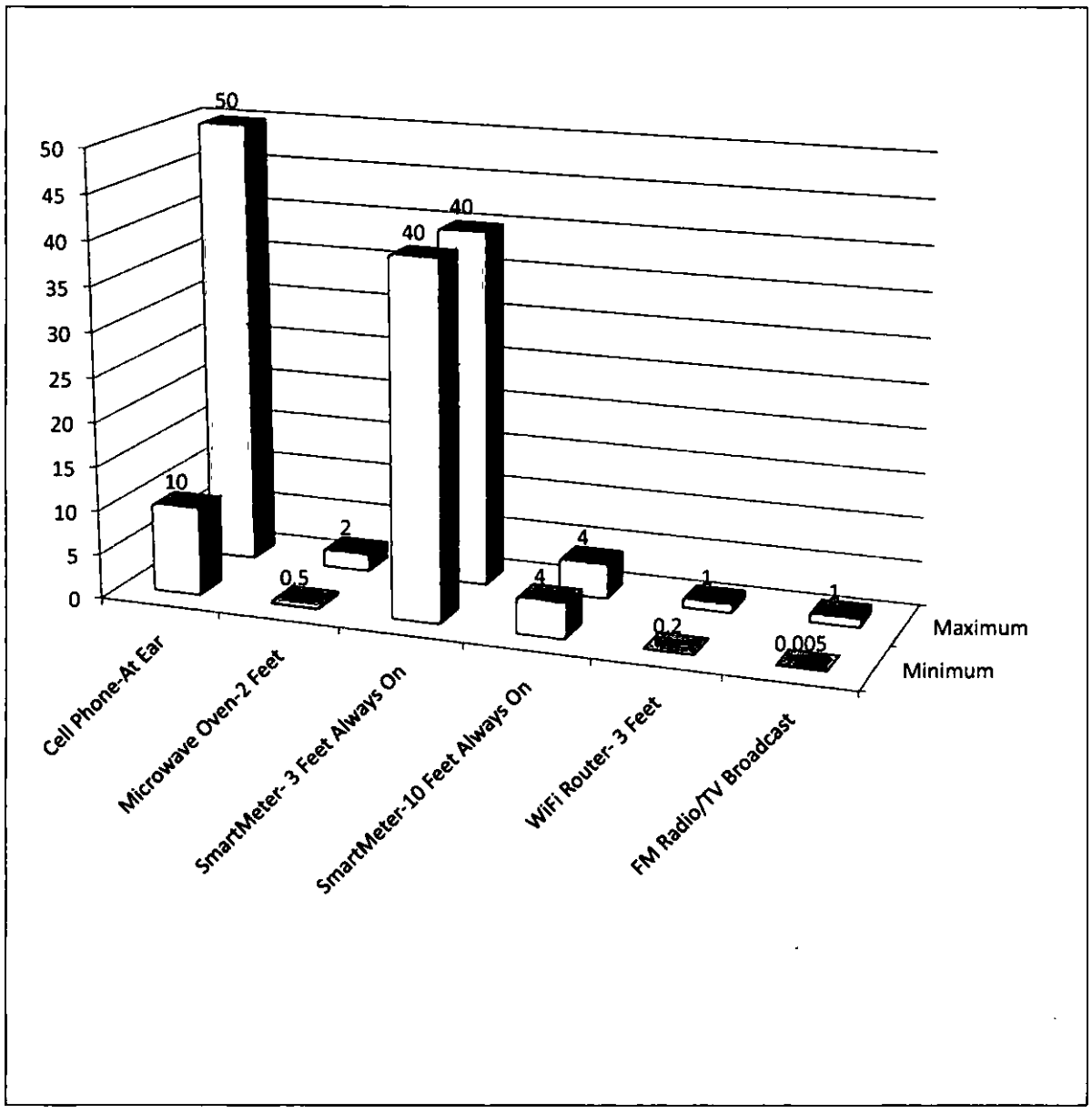

Figure 2. Comparison of Radio-Frequency Levels from Various Sources in $\mu W/cm^2$ over time (corrected only for assumed duty cycle).

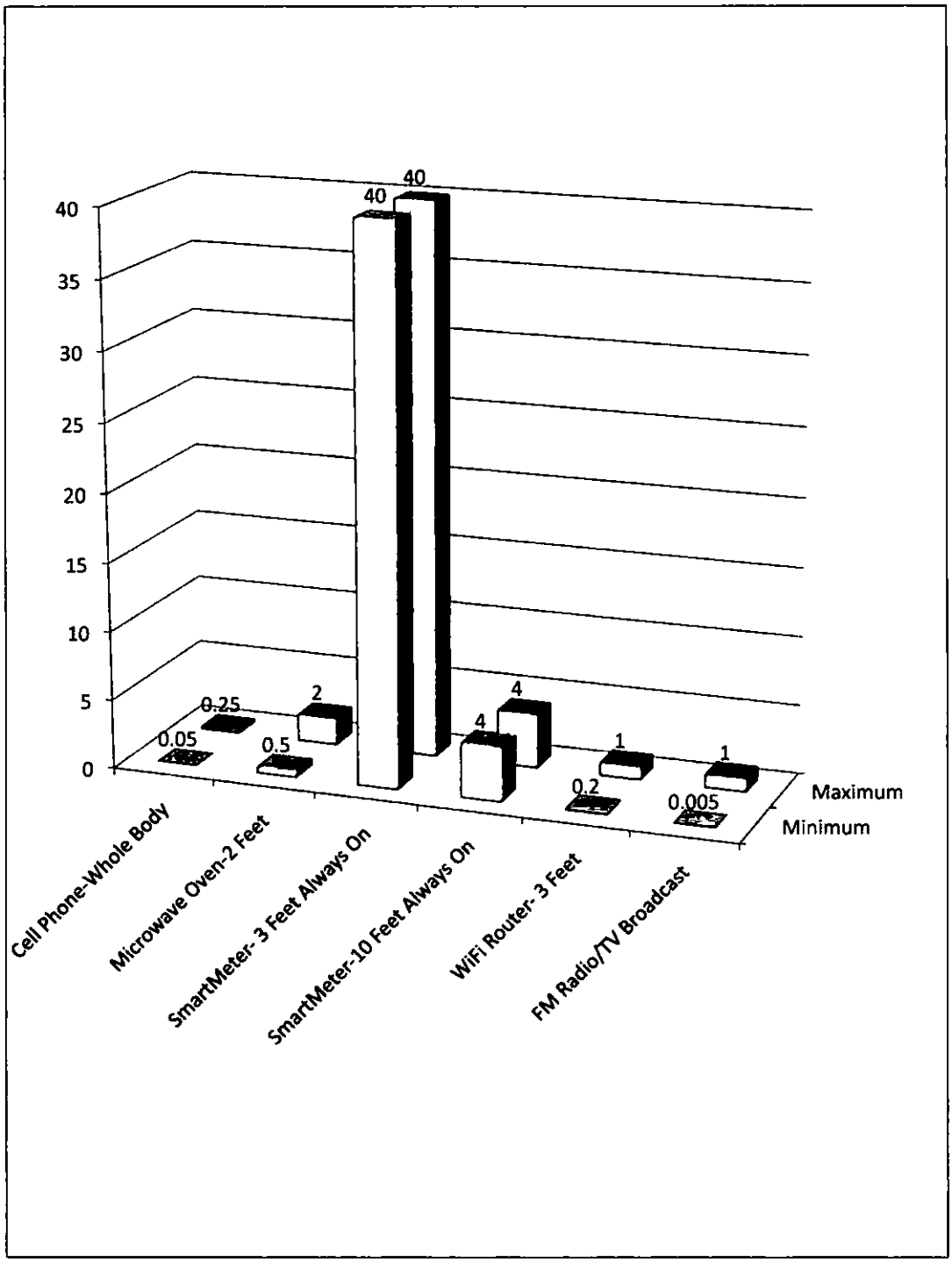

Figure 3. Comparison of Radio-Frequency Levels to the Whole Body from Various Sources in $\mu W/cm^2$ over time [corrected for assumed duty cycle and whole body exposure extrapolated from assumed cell phone dose at ear].

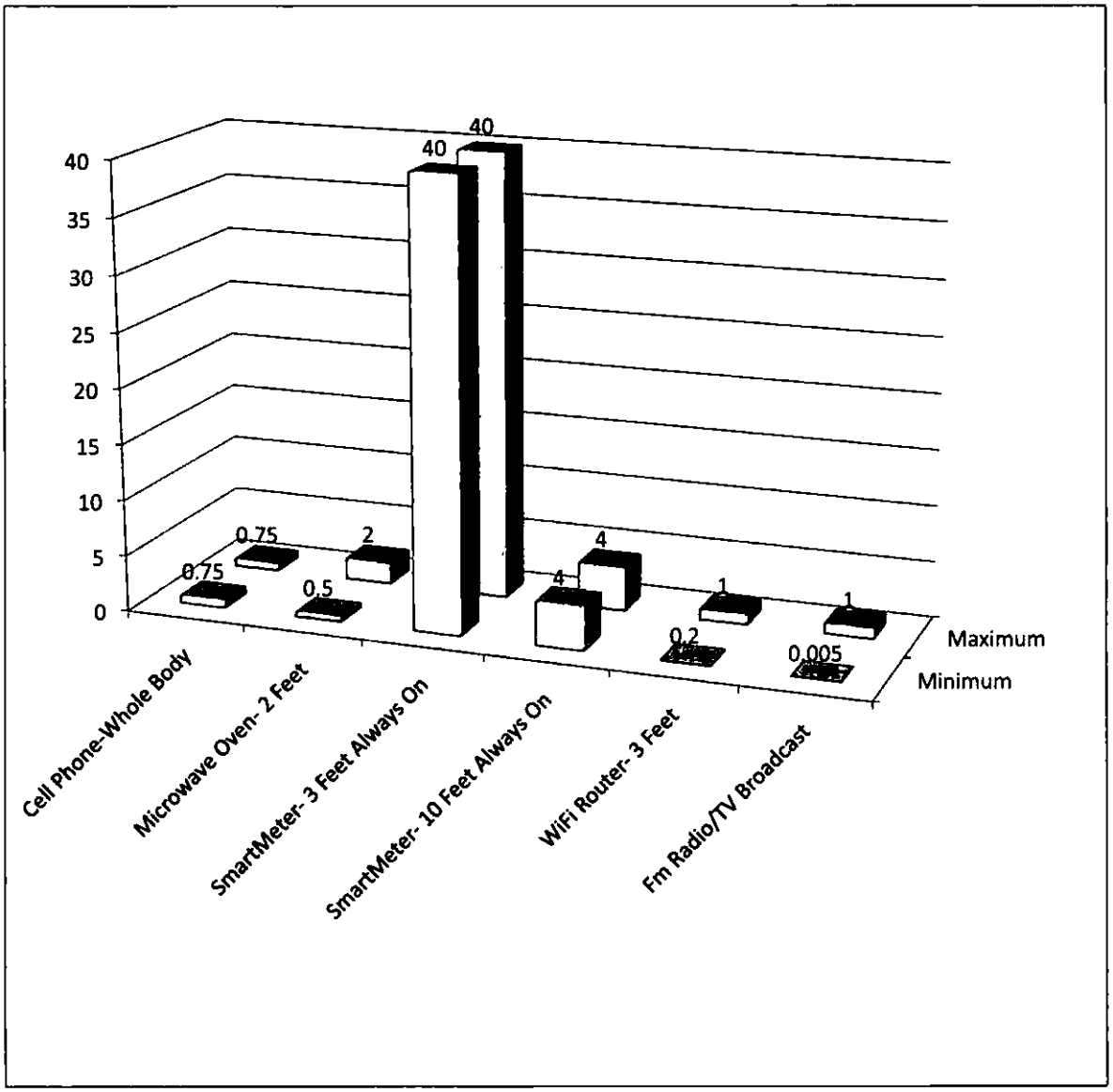

Figure 4. Comparison of Radio-Frequency Levels to the Whole Body from Various Sources in $\mu W/cm^2$ over time [corrected for assumed duty cycle and whole body exposure extrapolated from EPRI/CCST SmartMeter estimated levels at 3 feet].

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ENCLOSURE (4)

Electromagnetic and Radiofrequency Fields Effect on Human Health

For over 50 years, the American Academy of Environmental Medicine (AAEM) has been studying and treating the effects of the environment on human health. In the last 20 years, our physicians began seeing patients who reported that electric power lines, televisions and other electrical devices caused a wide variety of symptoms. By the mid 1990's, it became clear that patients were adversely affected by electromagnetic fields and becoming more electrically sensitive. In the last five years with the advent of wireless devices, there has been a massive increase in radiofrequency (RF) exposure from wireless devices as well as reports of hypersensitivity and diseases related to electromagnetic field and RF exposure. Multiple studies correlate RF exposure with diseases such as cancer, neurological disease, reproductive disorders, immune dysfunction, and electromagnetic hypersensitivity.

The electromagnetic wave spectrum is divided into ionizing radiation such as ultraviolet and X-rays and non-ionizing radiation such as radiofrequency (RF), which includes WiFi, cell phones, and Smart Meter wireless communication. It has long been recognized that ionizing radiation can have a negative impact on health. However, the effects of non-ionizing radiation on human health recently have been seen. Discussions and research of non-ionizing radiation effects centers around thermal and non-thermal effects. According to the FCC and other regulatory agencies, only thermal effects are relevant regarding health implications and consequently, exposure limits are based on thermal effects only.[1]

While it was practical to regulate thermal bioeffects, it was also stated that non-thermal effects are not well understood and no conclusive scientific evidence points to non-thermal based negative health effects.[1] Further arguments are made with respect to RF exposure from WiFi, cell towers and smart meters that due to distance, exposure to these wavelengths are negligible.[2] However, many in vitro, in vivo and epidemiological studies demonstrate that significant harmful biological effects occur from non-thermal RF exposure and satisfy Hill's criteria of causality.[3] Genetic damage, reproductive defects, cancer, neurological degeneration and nervous system dysfunction, immune system dysfunction, cognitive effects, protein and peptide damage, kidney damage, and developmental effects have all been reported in the peer-reviewed scientific literature.

Genotoxic effects from RF exposure, including studies of non-thermal levels of exposure, consistently and specifically show chromosomal instability, altered gene expression, gene mutations, DNA fragmentation

and DNA structural breaks.[4-11] A statistically significant dose response effect was demonstrated by Maschevich et al. , who reported a linear increase in aneuploidy as a function of the Specific Absorption Rate(SAR) of RF exposure.[11] Genotoxic effects are documented to occur in neurons, blood lymphocytes, sperm, red blood cells, epithelial cells, hematopoietic tissue, lung cells and bone marrow. Adverse developmental effects due to non-thermal RF exposure have been shown with decreased litter size in mice from RF exposure well below safety standards.[12] The World Health Organization has classified RF emissions as a group 2 B carcinogen.[13] Cellular telephone use in rural areas was also shown to be associated with an increased risk for malignant brain tumors. [14]

The fact that RF exposure causes neurological damage has been documented repeatedly. Increased blood-brain barrier permeability and oxidative damage, which are associated with brain cancer and neurodegenerative diseases, have been found.[1,7,15-17] Nittby et al. demonstrated a statistically significant dose-response effect between non-thermal RF exposure and occurrence of albumin leak across the blood-brain barrier.[15] Changes associated with degenerative neurological diseases such as Alzheimer's, Parkinson's and Amyotrophic Lateral Sclerosis (ALS) have been reported.[1,16] Other neurological and cognitive disorders such as headaches, dizziness, tremors, decreased memory and attention, autonomic nervous system dysfunction, decreased reaction times, sleep disturbances and visual disruption have been reported to be statistically significant in multiple epidemiological studies with RF exposure occurring non-locally.[18-21]

Nephrotoxic effects from RF exposure also have been reported. A dose response effect was observed by Ingole and Ghosh in which RF exposure resulted in mild to extensive degenerative changes in chick embryo kidneys based on duration of RF exposure.[21] RF emissions have also been shown to cause isomeric changes in amino acids that can result in nephrotoxicity as well as hepatotoxicity.[25]

Electromagnetic field (EMF) hypersensitivity has been documented in controlled and double blind studies with exposure to various EMF frequencies. Rea et al. demonstrated that under double blind placebo controlled conditions, 100% of subjects showed reproducible reactions to that frequency to which they were most sensitive.[26] Pulsed electromagnetic frequencies were shown to consistently provoke neurological symptoms in a blinded subject while exposure to continuous frequencies did not.[26]

Although these studies clearly show causality and disprove the claim that health effects from RF exposure are uncertain, there is another mechanism that proves electromagnetic frequencies, including radiofrequencies, can negatively impact human health. Government agencies and industry set safety

standards based on the narrow scope of Newtonian or "classical" physics reasoning that the effects of atoms and molecules are confined in space and time. This model supports the theory that a mechanical force acts on a physical object and thus, long-range exposure to EMF and RF cannot have an impact on health if no significant heating occurs. However, this is an incomplete model. A quantum physics model is necessary to fully understand and appreciate how and why EMF and RF fields are harmful to humans.[26,27] In quantum physics and quantum field theory, matter can behave as a particle or as a wave with wave-like properties. Matter and electromagnetic fields encompass quantum fields that fluctuate in space and time. These interactions can have long-range effects which cannot be shielded, are non-linear and by their quantum nature have uncertainty. Living systems, including the human body, interact with the magnetic vector potential component of an electromagnetic field such as the field near a toroidal coil.[25,28,29] The magnetic vector potential is the coupling pathway between biological systems and electromagnetic fields.[26,27] Once a patient's specific threshold of intensity has been exceeded, it is the frequency which triggers the patient's reactions.

Long range EMF or RF forces can act over large distances setting a biological system oscillating in phase with the frequency of the electromagnetic field so it adapts with consequences to other body systems. This also may produce an electromagnetic frequency imprint into the living system that can be long lasting.[26,27,40] Research using objective instrumentation has shown that even passive resonant circuits can imprint a frequency into water and biological systems.[31] These quantum electrodynamic effects do exist and may explain the adverse health effects seen with EMF and RF exposure. These EMF and RF quantum field effects have not been adequately studied and are not fully understood regarding human health.

Because of the well documented studies showing adverse effects on health and the not fully understood quantum field effect, AAEM calls for exercising precaution with regard to EMF, RF and general frequency exposure. In an era when all society relies on the benefits of electronics, we must find ideas and technologies that do not disturb bodily function. It is clear that the human body uses electricity from the chemical bond to the nerve impulse and obviously this orderly sequence can be disturbed by an individual-specific electromagnetic frequency environment. Neighbors and whole communities are already exercising precaution, demanding abstention from wireless in their homes and businesses.

Furthermore, the AAEM asks for:

- An immediate caution on Smart Meter installation due to potentially harmful RF exposure.

- Accommodation for health considerations regarding EMF and RF exposure, including exposure to wireless Smart Meter technology.
- Independent studies to further understand the health effects from EMF and RF exposure.
- Recognition that electromagnetic hypersensitivity is a growing problem worldwide.
- Understanding and control of this electrical environmental bombardment for the protection of society.
- Consideration and independent research regarding the quantum effects of EMF and RF on human health.
- Use of safer technology, including for Smart Meters, such as hard-wiring, fiber optics or other non-harmful methods of data transmission.

Submitted by: Amy L. Dean, DO, William J. Rea, MD, Cyril W. Smith, PhD, Alvis L. Barrier, MD

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December 6, 2011

[redacted]

Dear [redacted]

Thank you for contacting me to express your concerns about smart meters. I appreciate hearing from you.

As you may know, provisions within the 2005 Energy Policy Act allow for consumers to opt out of smart meter programs that are run at the state level. Florida consumers can opt out of these programs by contacting the appropriate authorities. As this is a state issue, I suggest that you contact Florida Power and Light at (305) 552-2950 and the Florida Public Service Commission at 1(800) 342-3552 and request that you be added to the smart meter opt out list.

Thank you again, [redacted], for contacting me. I appreciate having the benefit of your views. It is an honor to serve you in Congress. For more information on my work in Congress, to sign up to receive my E-newsletter, or to participate in telephone town hall meetings, please visit my website, http://www.posey.house.gov, or call my office at (321) 632-1776. If I may be of service to you in the future, please do not hesitate to contact me.

Sincerely,

Bill

Bill Posey
Member of Congress

ENCLOSURE (5)

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