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August 27, 2019

VIA ELECTRONIC FILING

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

Re: Trent Nickels v. PPL Electric Utilities Corporation
Docket No. C-2019-3008215

Dear Secretary Chiavetta:

Enclosed for filing is PPL Electric Utilities Corporation's Motion to Compel in the above-referenced proceeding. Copies will be provided as indicated on the Certificate of Service.

Respectfully submitted,

Devin Ryan

DTR/dmc
Enclosures

cc: Honorable Elizabeth Barnes (*w/enclosures*)
Certificate of Service

CERTIFICATE OF SERVICE

I hereby certify that a true and correct copy of the foregoing has been served upon the following persons, in the manner indicated, in accordance with the requirements of 52 Pa. Code § 1.54 (relating to service by a participant).

VIA FIRST CLASS MAIL

Trent Nickels
135 Marquise Drive
PO Box 251
Tafton, PA 18464

Date: August 27, 2019

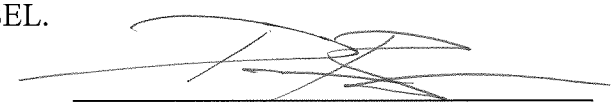
Devin T. Ryan

BEFORE THE
PENNSYLVANIA PUBLIC UTILITY COMMISSION

Trent Nickels,	:	
	:	
Complainant,	:	
	:	
v.	:	Docket No. C-2019-3008215
	:	
PPL Electric Utilities Corporation,	:	
	:	
Respondent.	:	

NOTICE TO PLEAD

YOU ARE HEREBY ADVISED THAT, PURSUANT TO 52 PA. CODE § 5.342(g)(1), YOU MAY FILE A REPLY TO THE ENCLOSED MOTION TO COMPEL WITHIN FIVE (5) DAYS AFTER THE DATE OF SERVICE. YOUR REPLY SHOULD BE FILED WITH THE SECRETARY OF THE PENNSYLVANIA PUBLIC UTILITY COMMISSION, P.O. BOX 3265, HARRISBURG, PA 17105-3265. A COPY OF YOUR REPLY SHOULD ALSO BE SERVED ON THE UNDERSIGNED COUNSEL.



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Date: August 27, 2019

Attorneys for PPL Electric Utilities Corporation

BEFORE THE
PENNSYLVANIA PUBLIC UTILITY COMMISSION

Trent Nickels,	:	
	:	
Complainant,	:	
	:	
v.	:	Docket No. C-2019-3008215
	:	
PPL Electric Utilities Corporation,	:	
	:	
Respondent.	:	

MOTION OF PPL ELECTRIC UTILITIES CORPORATION TO COMPEL RESPONSES TO DISCOVERY PROPOUNDED ON TRENT NICKELS – SET I

TO ADMINISTRATIVE LAW JUDGE ELIZABETH H. BARNES:

Pursuant to 52 Pa. Code §§ 5.342(g) and 5.349(d), PPL Electric Utilities Corporation ("PPL Electric" or the "Company") hereby files this Motion to Compel Responses to Discovery Propounded on Trent Nickels ("Complainant") – Set I. In support of its Motion, PPL Electric states as follows:

I. INTRODUCTION

1. On June 25, 2019, PPL Electric served Interrogatories and Requests for Production of Documents on the Complainant – Set I ("PPL to Complainant Set I") via certified mail. A true and correct copy of PPL to Complainant Set I is attached hereto and marked as **Appendix A**.

2. Pursuant to the Commission's regulations, objections to PPL to Complainant Set I were due on or before July 8, 2019, and responses were due on or before July 18, 2019.[1]

3. The Complainant never served any objections to PPL to Complainant Set I by July 8, 2019.

4. On July 26, 2019, the Complainant served his responses to PPL to Complainant Set I. However, the Complainant refused to provide or did not provide the information and materials requested in PPL to Complainant Set I, Questions 3 and 4. A true and correct copy of the Complainant's responses PPL to Complainant Set I is attached hereto and marked as **Appendix B**.

5. On August 16, 2019, counsel for PPL Electric left the Complainant a detailed voicemail about the deficiencies with the discovery responses.

6. On August 19, 2019, the Complainant's wife called PPL Electric's counsel and stated that she and her husband would try to obtain the materials requested in response to Questions 3 and 4, but she was not sure on when they would be able to do that. Given that the hearing is approximately one month away, counsel for PPL Electric informed her that the Company would proceed with filing the Motion to Compel. However, the Company's counsel also explained that PPL Electric could withdraw the Motion if it receives complete responses to Questions 3 and 4.

7. To date, the Complainant has never sent complete responses to PPL to Complainant Set I, Questions 3 and 4.

[1] Because the discovery was served via certified mail by the United States Postal Service ("USPS"), three days were added to the prescribed period for response. *See* 52 Pa. Code § 1.56(b).

II. MOTION TO COMPEL

8. PPL Electric requests that Administrative Law Judge Elizabeth H. Barnes ("ALJ") compel responses to PPL to Complainant Set I, Questions 3 and 4.

9. Under 52 Pa. Code § 5.321(c), a party is entitled to obtain discovery of any matter not privileged that is relevant to the pending proceeding, or any matter that is reasonably calculated to lead to the discovery of admissible evidence. Discovery is permitted regardless of whether the information sought "relates to the claim or defense of the party seeking discovery or to the claim or defense of another party." *Id.*

10. Objections to interrogatories and requests for production of documents must be served within 10 days of the date the discovery was served. 52 Pa. Code §§ 5.342(e), 5.349(d). Objecting parties remain under an obligation to provide timely answers to interrogatories or subparts of interrogatories to which they did not object. *Id.* § 5.342(f). Further, objections must be contained in a document separate from an answer. *Id.* §§ 5.342(c), 5.349(d).[2]

11. Answers to written interrogatories must "[a]nswer each interrogatory fully and completely unless an objection is made." *Id.* § 5.342(a)(4). Answers must be served within 20 days after service of the interrogatories. *Id.* § 5.342(d).

12. Similarly, a party shall serve a response to a request for documents within 20 days after the service of the request. *Id.* § 5.349(d). The requesting party may move to compel a response to a request for documents with respect to a failure to respond to the request. *See id.*

13. As explained herein, the Complainant has failed to comply with the Commission's discovery rules by failing to provide full and complete responses to PPL to Complainant Set I, Questions 3 and 4 and produce the documents sought by these requests.

[2] The Complainant did not serve objections to discovery. Therefore, Complainant's failure to provide full and complete responses to discovery requests operate, in effect, as untimely objections.

14. For the reasons stated in more detail below, the ALJ should direct the Complainant to answer fully PPL to Complainant Set I, Questions 3 and 4. Moreover, should the ALJ grant PPL Electric's Motion to Compel and the Complainant fail to timely provide full and complete responses to Complainant Set I, Questions 3 and 4, PPL Electric intends to file a Motion for Sanctions pursuant to 52 Pa. Code §§ 5.371(a) and 5.372(a).

A. PPL TO COMPLAINANT SET I, QUESTION 3

15. PPL to Complainant-I-3 requests the following:

> Please state whether you or any member of your household uses a cell phone. If so, please provide the make and model of each cell phone and, for each phone identified, provide 12 months of phone bills or other records of actual cell phone usage.

16. The Complainant's response to PPL to Complainant-I-3 states:

> I will now provide information about household cellphones (response by Beth Nickels).
>
> a. Cellphones – Android ZTE 7.1.1; iPhone 4; iPhone XS Max; iPhone 6 (employer-issued brought home weekends only)
>
> b. Because I realize that any information I provide will become a matter of public record, I decline to provide copies of phone bills in order to protect the privacy of my family members.

17. As explained above, Section 5.342(a)(4) requires a party to fully and completely answer an interrogatory. 52 Pa. Code § 5.342(a)(4). In addition, a party has a duty to amend its prior responses to discovery requests when the information contained therein is incomplete. *Id.* § 5.332(2).

18. The response served by the Complainant was non-responsive and incomplete because he refused to provide the 12 months of phone bills or other records of actual cell phone usage "in order to protect the privacy of [his] family members."

19. However, the deadline to serve any objections was July 8, 2019.

20. The Complainant's response was served on July 26, 2019.

21. Therefore, the Complainant waived his right to object to this interrogatory.

22. Moreover, the information requested about cell phone use is highly relevant to the issues raised in this case.

23. The Complainant claims that he has health concerns about RF fields from AMI meters. As the testimony of PPL Electric's expert witness on RF exposures – Dr. Christopher Davis – will demonstrate, however, the RF exposure received from use of a cell phone (or even standing within 30 feet of another person using a cell phone) is far higher than from an AMI meter.

24. In fact, in the response to Question 3, the Complainant identifies four cell phones.

25. PPL Electric is entitled to: (1) show how the RF exposures from the cell phone use compare to those from the AMI meter; and (2) discover the billing records needed to quantify the amount of time that the Complainant and members of his household choose to use the phone.

26. Finally, to the extent that the Complainant has concerns about the account numbers or payment information from these bills being disclosed, the Complainant can redact them from the bills. PPL Electric simply needs the records to quantify and verify the amount of time that the cell phones are used.

27. Based on the foregoing, the ALJ should direct the Complainant to answer fully PPL to Complainant Set I, Question 3.

B. PPL TO COMPLAINANT SET I, QUESTION 4

28. PPL to Complainant-I-4 requests the following:

> (a) Please state every health condition you claim was caused by a smart meter or will be caused or worsened by the installation of PPL Electric's new smart meter.

(b) Please provide the date that every health condition identified in subpart (a) began.

(c) Please provide copies of all your medical records of every health condition identified in subpart (a).

(d) For each alleged health condition that you do not have medical records for in response to subpart (c), please state whether such condition was diagnosed by a medical professional. If so, please provide the name, address, and telephone number of the medical professional and the date of the diagnosis.

(e) For each of the alleged health conditions identified in subpart (a), please state whether you have been prescribed any therapy or treatment for the condition by a medical professional. If so, please identify the therapy or treatment, provide the name, address, and telephone number of the prescribing medical professional, and provide the date the therapy or treatment was prescribed.

29. The Complainant's response to the question states:

The following health conditions will be worsened by the installation of the new smart meter.

(a) Symptoms of Mast Cell Activation Syndrome and symptoms of electrohypersensitivity will be worsened by the installation of the smart meter (see Item I-1).

(b) Mast Cell Activation Syndrome symptoms began in the mid-1990s. Electrohypersensitivity symptoms began in approximately the year 2000.

(c) Regarding the request for medical records: I decline to provide my wife's private and personal health records. They are protected under HIPPA [sic], and she prefers that they not become a matter of public record (response by Beth Nickels).

(d) For the following health conditions, for which I am not providing medical records:

- Multiple Chemical Sensitivities (diagnosed in 1999 by Roy Kerry, MD; Advanced Integrative Medicine, 160 E. Portersville Road, Portersville, PA 16051; ph# 724-368-2558).

- Mast Cell Activation Syndrome and Peripheral Neuropathy (diagnosed April 19, 2019 by Dr. Anne Maitland, MD; Comprehensive Allergy and Asthma Care; 200 South Broadway Ste: 104, Tarrytown, NY 10591-4004)

(e) For Mast Cell Activation Syndrome, my wife was prescribed the following medications by Dr. Anne Maitland, MD; Comprehensive Allergy and Asthma Care; 200 South Broadway Ste: 104, Tarrytown, NY 10591-4004:

- Montelukast
- Levocetirizine
- Levalbuterol solution for nebulization
- Cromolyn solution for nebulization
- Cyproheptadine
- Clemastine
- Ipratropium bromide

These treatments were prescribed on April 19, 2019 (response by Beth Nickels)

For Multiple Chemical Sensitivities, the primary treatment recommendation was to avoid situations and substances that would trigger her condition. (Roy Kerry, MD; Advanced Integrative Medicine, 160 E. Portersville Road, Portersville, PA 16051; ph# 724-368-3558). The date for these recommendations was 1999 and ongoing (response by Beth Nickels).

30. As explained above, Section 5.342(a)(4) requires a party to fully and completely answer an interrogatory. 52 Pa. Code § 5.342(a)(4). In addition, a party has a duty to amend its prior responses to discovery requests when the information contained therein is incomplete. *Id.* § 5.332(2).

31. The responses served by the Complainant to Question 4 were non-responsive and incomplete.

32. In the response to subpart (c), the Complainant refuses to provide his "wife's private and personal health records" because: (1) they are "protected under HIPPA [sic]"; and (2) his wife "prefers that they not become a matter of public record."

33. However, the deadline to serve any objections was July 8, 2019.

34. The Complainant's response was served on July 26, 2019.

35. Therefore, the Complainant waived his right to object to interrogatory.

36. Moreover, the Complainant alleges that the new AMI meter has caused, contributed to, or exacerbated adverse health effects.

37. Therefore, the Complainant's medical records and conditions are highly relevant to this case.

38. Yet, the Complainant has refused to provide any medical records about the health conditions that have been allegedly caused, contributed to, or exacerbated by the new AMI meter. Although the Complainant has provide information about whether those conditions were diagnosed by a medical professional, there is no way for the Company to investigate and verify these claims because the Complainant refuses to provide the actual medical records.

39. In addition, the Complainant fails to recognize that the Health Insurance Portability and Accountability Act ("HIPAA") does not prohibit the disclosure of medical records in response to a discovery request. Indeed, the U.S. Department of Health and Human Services' HIPAA regulations provide an express exception permitting any "covered entity"[3] to "disclose protected health information in the course of any judicial or administrative proceeding," such as in "response to an order of a court or administrative tribunal" or in "response to a subpoena, discovery request, or other lawful process." 45 C.F.R. § 164.512(e)(1) (emphasis added).[4]

[3] "Covered entity" means a "health plan," a "health care clearinghouse," or a "health care provider who transmits any health information in electronic form in connection with a transaction covered by this subchapter." 45 C.F.R. § 160.103. PPL Electric is not a covered entity.

[4] Certain conditions apply when there is and is not a protective order in place. *See id.*

40. The Complainant should be directed to provide a full and complete response to this interrogatory.

41. Furthermore, Section 5.365(c)(4) of the Commission's regulations states:

> Prior to the issuance of a protective order, a party may not refuse to provide information which the party reasonably believes to be proprietary to a party who agrees to treat the information as if it were covered by a protective order until the presiding officer or the Commission issues the order or determines that issuance of the order would not be appropriate. The party claiming the privilege shall file a petition for protective order under subsection (a) within 14 days of the date the request for information was received.

52 Pa. Code § 5.365(c)(4).

42. Here, concurrent with the filing of the instant Motion, PPL Electric is filing a Motion for Protective Order, which would protect the Complainant's medical records and information from unauthorized public disclosure.

43. PPL Electric hereby agrees to treat such information as confidential as though it were governed by the terms of a protective order. Thus, under Section 5.365(c)(4), the Complainant cannot refuse to provide this information to the Company.

44. For these reasons, the ALJ should direct the Complainant to answer fully PPL to Complainant Set I, Question 4.

III. SANCTIONS

45. Upon the motion of a party, the presiding officer may make an appropriate order for sanctions if a party fails to answer or otherwise respond to a discovery request or refuses to obey an order of the presiding officer respecting discovery. *See* 52 Pa. Code § 5.371(a).

46. In ruling upon a motion for sanctions, the presiding officer may, among other things, issue: (1) "[a]n order that the matters regarding which the questions were asked, the character or description of the thing or land, the contents of the paper, or other designated fact

shall be taken to be established for the purposes of the action in accordance with the claim of the party obtaining the order"; (2) [a]n order refusing to allow the disobedient party to support or oppose designated claims or defenses, or prohibiting the party from introducing in evidence designated documents, things or testimony"; and (3) "[a]n order striking out pleadings or parts thereof, staying further proceedings until the order is obeyed, or entering a judgment against the disobedient party or individual advising the disobedience." *Id.* § 5.372(a)(1)-(3).

47. If the Complainant fails to provide full and complete responses to PPL Electric's discovery requests by September 9, 2019, PPL Electric will be deprived of a reasonable opportunity to prepare for the hearing and respond to the Complainant's claims at the September 23, 2019 hearing.

48. Accordingly, the Company respectfully requests that if the Complainant fails to produce the information and documents related to any of his claims by September 9, 2019, then the Complainant should be barred from litigating the corresponding claim(s).

49. For example, if the Complainant fails to produce the medical records to verify the existence of any claimed medical conditions or issues, then the Complainant would be precluded from litigating claims that the installation of PPL Electric's AMI meter would cause, contribute to, or exacerbate any adverse health effects.

50. Similarly, if the Complainant fails to produce information about the current exposure to RF fields as requested in Question 3, including but not limited to bills showing the extent of cell phone use, then the Complainant would be precluded from litigating claims that PPL Electric's AMI meter exposes him and members of his household to unreasonable levels of RF fields.

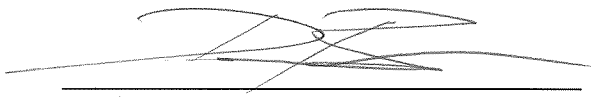
51. Finally, to the extent that this Motion is granted and the Complainant fails to answer fully PPL to Complainant Set I, or otherwise comply with the ALJ's order, PPL Electric intends to file an appropriate motion pursuant to 52 Pa. Code §§ 5.371(a) and 5.372(a) to dismiss the Complaint with prejudice.

52. The Commission has regularly dismissed AMI meter complaints with prejudice due to the complainants' failure to answer discovery in compliance with the presiding administrative law judge's orders granting motions to compel. *See, e.g.*, *Carol Sojda & Carol Lutzkanin v. Metropolitan Edison Co.*, Docket No. C-2017-2638350, pp. 7-8 (Jan. 9, 2019), *adopted*, Docket No. C-2017-2638350 (Order entered Mar. 28, 2019); *Kimberly Beckmann v. Metropolitan Edison Co.*, Docket No. C-2017-2613702, pp. 7-10 (Jan. 31, 2019), *adopted*, Docket No. C-2017-2613702 (Order entered Apr. 11, 2019); *Darlene Stanton v. Pennsylvania Electric Co.*, Docket No. C-2018-3001144, pp. 6-11 (May 10, 2019), *adopted*, Docket No. C-2018-3001144 (Order entered July 11, 2019); *Diana Cook v. West Penn Power Co.*, Docket No. C-2018-3003051, pp. 6-10 (May 1, 2019), *adopted*, Docket No. C-2018-3003051 (Order entered July 11, 2019).

IV. CONCLUSION

For the reasons set forth above, PPL Electric Utilities Corporation respectfully requests that Administrative Law Judge Elizabeth H. Barnes grant this Motion to Compel Responses to Discovery and direct Trent Nickels to answer fully PPL to Complainant Set I, as described above within three (3) days from the date of the order.

Respectfully submitted,



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Phone: 202-737-6302
E-mail: crenner@w-r.com

Date: August 27, 2019

Attorneys for PPL Electric Utilities Corporation

APPENDIX A

Interrogatories and Requests for Production of Documents Propounded by PPL Electric Utilities Corporation on Trent Nickels – Set I

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Devin Ryan

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717-612-6052 Direct
717-731-1981 Direct Fax
File #: 167945

June 25, 2019

VIA CERTIFIED MAIL (7017 1450 0002 3778 0918)

Trent Nickels
135 Marquise Drive
PO Box 251
Tafton, PA 18464

Re: Trent Nickels v. PPL Electric Utilities Corporation
Docket No. C-2019-3008215

Dear Mr. Nickels:

Enclosed are the Interrogatories and Requests for Production of Documents Propounded by PPL Electric Utilities Corporation on Trent Nickels – Set I in the above-referenced proceeding. Copies will be provided as indicated on the Certificate of Service.

Please provide answers to the enclosed discovery within twenty (20) days of the date of service, pursuant to 52 Pa. Code § 5.342.

Sincerely,

Devin Ryan

DTR/jpf
Enclosures

cc: Rosemary Chiavetta, Secretary (*Letter & Certificate of Service Only*)
Certificate of Service

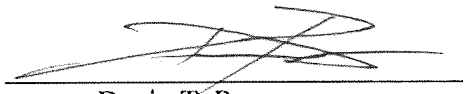
CERTIFICATE OF SERVICE

I hereby certify that a true and correct copy of the foregoing has been served upon the following persons, in the manner indicated, in accordance with the requirements of 52 Pa. Code § 1.54 (relating to service by a participant).

VIA CERTIFIED MAIL

Trent Nickels
135 Marquise Drive
PO Box 251
Tafton, PA 18464

Date: June 25, 2019



Devin T. Ryan

BEFORE THE
PENNSYLVANIA PUBLIC UTILITY COMMISSION

Trent Nickels,	:	
	:	
Complainant	:	
	:	
v.	:	Docket No. C-2019-3008215
	:	
PPL Electric Utilities Corporation,	:	
	:	
Respondent	:	

INTERROGATORIES AND REQUESTS FOR PRODUCTION OF DOCUMENTS PROPOUNDED BY PPL ELECTRIC UTILITIES CORPORATION ON TRENT NICKELS – SET I

Pursuant to 66 Pa.C.S. § 333 and 52 Pa. Code §§ 5.341 *et seq.*, PPL Electric Utilities Corporation ("PPL Electric") propounds the following Interrogatories and Requests for Production of Documents (hereinafter, "discovery requests") on Trent Nickels ("Complainant") – Set I.

INSTRUCTIONS AND DEFINITIONS

1. The "Responding Party," "you," or "your" means the party to which these discovery requests are propounded and/or all attorneys, agents, affiliates, subsidiaries, employees, consultants, members, constituents, and representatives acting on behalf of the Responding Party.

2. "Commission" means the Pennsylvania Public Utility Commission.

3. To "identify" a natural person means to state that person's full name, title or position, employer, last known address, and last known telephone number.

4. To "identify" a business entity means to state the full name of such business, the form of the business, and its location or address.

5. To "identify" a "document" means to provide all of the following information irrespective of whether the document is deemed privileged or subject to any claim of privilege:

a. The title or other means of identification of each such document;

b. The date of each such document;

c. The author, preparer or signer of each such document; and

d. A description of the subject matter of such document sufficient to permit an understanding of its contents and importance to the testimony or position being examined and the present or last known location of the document. The specific nature of the document should also be stated (*e.g.*, letter, business record, memorandum, computer print-out, etc.).

In lieu of "identifying" any document, it shall be deemed a sufficient compliance with these discovery requests to attach a copy of each such document to the answers hereto and reference said document in the particular interrogatory to which the document is responsive.

6. "Document" means the original and all drafts of all written and graphic matter, however produced or reproduced, of any kind or description, whether or not sent or received, and all copies thereof which are different in any way from the original (whether by interlineation, date-stamp, notarization, indication of copies sent or received, or otherwise), including without limitation, any paper, book, account, photograph, blueprint, drawing, sketch, schematic, agreement, contract, memorandum, press release, circular, advertising material, correspondence, letter, telegram, telex, object, report, opinion, investigation, record, transcript, hearing, meeting, study, notation, working paper, summary, intra-office communication, diary, chart, minutes, index sheet, computer software, computer-generated records or files, however stored, check, check stub, delivery ticket, bill of lading, invoice, record or recording or summary of any telephone or other conversation, or of any interview or of any conference, or

any other written, recorded, transcribed, punched, taped, filmed, or graphic matter of which the Responding Party has or has had possession, custody or control, or of which the Responding Party has knowledge.

7. "Communication" means any manner or form of information or message transmission, however produced or reproduced, whether as a document as herein defined, or orally or otherwise, which is made, distributed, or circulated between or among persons, or data storage or processing units.

8. "Date" means the exact day, month, and year, if ascertainable, or if not, the best approximation thereof.

9. Items referred to in the singular include those in the plural, and items referred to in the plural include those in the singular.

10. Items referred to in the masculine include those in the feminine, and items referred to in the feminine include those in the masculine.

11. The answers provided to these discovery requests should first restate the question asked and identify the person(s) supplying the information.

12. In answering these discovery requests, the Responding Party is requested to furnish all information that is available to the Responding Party, including information in the possession of the Responding Party's attorneys, agents, consultants, or investigators, and not merely such information of the Responding Party's own knowledge. If any of the discovery requests cannot be answered in full after exercising due diligence to secure the requested information, please so state and answer to the extent possible, specifying the Responding Party's inability to answer the remainder, and stating whatever information the Responding

Party has concerning the unanswered portions. If the Responding Party's answer is qualified in any particular, please set forth the details of such qualification.

13. If the Responding Party objects to providing any document requested on any ground, identify such document by describing it as set forth in Instruction 5 and state the basis of the objection.

14. If the Responding Party objects to part of a discovery request and refuses to answer that part, state the Responding Party's objection and answer the remaining portion of that discovery request. If the Responding Party objects to the scope or time period of a discovery request and refuses to answer for that scope or time period, state the Responding Party's objection and answer the discovery request for the scope or time period that the Responding Party believes is appropriate.

15. If, in connection with a discovery request, the Responding Party contends that any information, otherwise subject to discovery, is covered by either the attorney-client privilege, the so-called "attorneys' work product doctrine," or any other privilege or doctrine, then specify the general subject matter of the information and the basis to support each such objection.

16. If any information is withheld on grounds of privilege or other protection from disclosure, provide the following information: (a) every person to whom such information has been communicated and from whom such information was learned; (b) the nature and subject matter of the information; and (c) the basis on which the privilege or other protection from disclosure is claimed.

17. As set forth in 52 Pa. Code § 5.342(g), these discovery requests are continuing, and the Responding Party is obliged to change, supplement, and correct all answers given to conform to new or changing information.

18. "Formal Complaint" means the Formal Complaint filed by the Complainant at Docket No. C-2019-3008215.

INTERROGATORIES AND REQUESTS FOR PRODUCTION OF DOCUMENTS PROPOUNDED ON TRENT NICKELS – SET I

PPL to Complainant-I-1

Re: Formal Complaint.

(a) Please explain in detail the reasons why you are challenging the Company's installation of the new smart meter.

(b) Please describe in detail all health concerns, if any, raised by the Company's new smart meter, state the bases for such claims, and provide all documents relied upon by you in your response.

(c) Please describe in detail all safety concerns, if any, raised by the Company's new smart meter, state the bases for such claims, and provide all documents relied upon by you in your response.

(d) Please describe in detail all privacy concerns, if any, raised by the Company's new smart meter, state the bases for such claims, and provide all documents relied upon by you in your response.

(e) Please describe in detail all reasons you believe the Company's new smart meter violates the law.

PPL to Complainant-I-2

Please identify all wireless phones, cellphones, microwaves, wireless routers, wifi networks, tablets, computers, Bluetooth speakers, wireless security systems, smart speakers (*e.g.*, Amazon Echo), garage door openers, baby monitors, and walkie talkies that are contained in or used in the house.

PPL to Complainant-I-3

Please state whether you or any member of your household uses a cell phone. If so, please provide the make and model of each cell phone and, for each phone identified, provide 12 months of phone bills or other records of actual cell phone usage.

PPL to Complainant-I-4

(a) Please state every health condition you claim was caused by a smart meter or will be caused or worsened by the installation of PPL Electric's new smart meter.

(b) Please provide the date that every health condition identified in subpart (a) began.

(c) Please provide copies of all your medical records of every health condition identified in subpart (a).

(d) For each alleged health condition that you do not have medical records for in response to subpart (c), please state whether such condition was diagnosed by a medical professional. If so, please provide the name, address, and telephone number of the medical professional and the date of the diagnosis.

(e) For each of the alleged health conditions identified in subpart (a), please state whether you have been prescribed any therapy or treatment for the condition by a medical professional. If so, please identify the therapy or treatment, provide the name, address, and telephone number of the prescribing medical professional, and provide the date the therapy or treatment was prescribed.

PPL to Complainant-I-5

Please identify each person you plan to call as a fact witness in this proceeding. For each person, please:

(a) Provide the person's name, home and business address, background, and qualifications;

(b) Explain in detail the subject matter(s) on which the witness is expected to testify; and

(c) Provide the source(s) of information relied upon or referenced by the witness.

PPL to Complainant-I-6

Please identify each person you plan to call as an expert witness in this proceeding. For each person, please:

(a) Provide the person's name, home and business address, background, and qualifications;

(b) Explain in detail the subject matter(s) on which the witness is expected to testify;

(c) Provide the source(s) of information relied upon or referenced by the witness; and

(d) Provide a copy of the expert witness's current curriculum vitae.

PPL to Complainant-I-7

Please provide copies of all exhibits you intend to present or utilize at the evidentiary hearing in this proceeding. For each exhibit to be used as part of your direct case, please identify the witness who will be sponsoring the exhibit.

APPENDIX B

Trent Nickels's Answers to PPL Electric Utilities Corporation's Interrogatories and Requests for Production of Documents – Set I

JUL 29 2019

Trent Nickels
P.O. Box 251
Tafton PA 18464

July 26, 2019

VIA CERTIFIED MAIL

Post & Schell
Devin Ryan
17 North Second Street
12th Floor
Harrisburg PA 17101-1601

Re: Trent Nickels v. PPL Electric Utilities Corporation
Docket No. C-2019-3008215

Dear Mr. Ryan,

Enclosed are the responses to the Interrogatories and Request for Production of Documents Propounded by PPL Electric Utilities Corporation on Trent Nickels – Set I in the above-referenced proceeding. Copies will be provided as indicated on the Certificate of Service.

Sincerely,

Trent Nickels

Trent Nickels

TDN/kbn
Enclosures

cc: Rosemary Chiavetta, Secretary (*Letter & Certificate of Service Only*)
Certificate of Service

Responses to Interrogatories and Request for Production of Documents July 26, 2019

Item I – 1

a) My primary reasons for challenging the installation of the smart meter are health-related.

b) I now state in detail my health concerns raised by the Company's new smart meter, the bases for such claims, and the documents relied upon in my response. My wife has been diagnosed with the following conditions, which maker her susceptible to electrohypersensitivity (EHS):

   a. Multiple Chemical Sensitivity
   b. Mast Cell Activation Syndrome (MCAS)
   c. Peripheral Neuropathy

   For the first two conditions, according to her health care providers, electrohypersensitivity (EHS) is a common feature. My wife's sensitivity to electromagnetic fields (EMFs) results in the following symptoms:

      1. Headache
      2. Pressure inside the head
      3. Irritability
      4. Anxiety
      5. Difficulty concentrating
      6. Sensation of burning/tingling/pain on skin
      7. Pain on contact
      8. General malaise
      9. Tinnitus
      10. Earache
      11. Skin rash

   I assert that the non-ionizing radiation emitted by the Smart Meter is likely to cause an increase in the frequency and intensity of her symptoms, making it difficult for her to be comfortable in our home. There is evidence that this type of radiation can cause physical harm, particularly for a customer suffering from electrohypersensitivity (EHS).

   The documents relied upon in my response are the following:

   - Exhibit A: Letter from Elaine Hardy, MS, RN, APN, C @ Holistic Family Healthcare, NJ
   - Exhibit B: Letter from Karin Cseak, DO @ Family Holistic Health, OH
   - Exhibit C: Abstract "A Theoretical Model Based Upon Mast Cells and Histamine to Explain the Recently Proclaimed Sensitivity to Electric and/or Magnetic Fields in Humans"
   - Exhibit D: "Reliable Disease Biomarkers Characterizing and Identifying Electrohypersensitivity and Multiple Chemical sensitivity as Two Etiopathogenic Aspects of a Unique Pathological Disorder"

c) At the present time, I have no safety concerns.

d) At this time, I have no privacy concerns.
e) At this time, I have no concerns about violation of the law.

Item I-2

I will now identify all wireless items contained or used inside the house (response by Beth Nickels)

1. Wireless phones – none
2. Microwave oven GE SpaceMaker– never used
3. Wireless router – LinkSys
4. Wireless modem – Arris
5. Smart speakers – none
6. Garage door opener – none
7. Walkie talkies – Motorola, model #MR350R (radio function only), Audiovox model #GMRS602
8. Baby monitors – none
9. Wireless security system – RING
10. Computers/tablets– HP EliteBook (laptop), refurbished; Gateway 2015, laptop; HP EnergyStar, desktop; MacBook Pro, 17-inch, laptop; Dell Latitude, laptop (employer-issued, brought home weekends only); iPad 3; iPad Pro 2011 (employer-issued, brought home weekends only); Dell Latitude (employer-issued, brought home weekends only)

Item I-3

I will now provide information about household cellphones (response by Beth Nickels).

a. Cellphones – Android ZTE 7.1.1; iPhone 4; iPhone XS Max; iPhone 6 (employer-issued, brought home weekends only)
b. Because I realize that any information I provide will become a matter of public record, I decline to provide copies of phone bills in order to protect the privacy of my family members.

Item I-4

The following health conditions will be worsened by the installation of the new smart meter:

a) Symptoms of Mast Cell Activation Syndrome and symptoms of electrohypersensitivity will be worsened by the installation of the smart meter (see Item I-1).
b) Mast Cell Activation Syndrome symptoms began in the mid-1990s. Electrohypersensitivity symptoms began in approximately the year 2000.
c) Regarding the request for medical records: I decline to provide my wife's private and personal health records. They are protected under HIPPA, and she prefers that they not become a matter of public record (response by Beth Nickels).
d) For the following health conditions, for which I am not providing medical records:

- Multiple Chemical Sensitivities (diagnosed in 1999 by Roy Kerry, MD; Advanced Integrative Medicine, 160 E. Portersville Road, Portersville PA 16051; ph# 724-368-3558).
- Mast Cell Activation Syndrome and Peripheral Neuropathy (diagnosed April 19, 2019 by Dr. Anne Maitland, MD; Comprehensive Allergy and Asthma Care; 200 South Broadway Ste: 104, Tarrytown, NY 10591-4004)

e) For Mast Cell Activation Syndrome, my wife was prescribed the following medications by Dr. Anne Maitland, MD; Comprehensive Allergy and Asthma Care; 200 South Broadway Ste: 104, Tarrytown, NY 10591-4004:
   - Montelukast
   - Levocetirizine
   - Levalbuterol solution for nebulization
   - Cromolyn solution for nebulization
   - Cyproheptadine
   - Clemastine
   - Ipratropium bromide

   These treatments were prescribed on April 19, 2019 (response by Beth Nickels).

   For Multiple Chemical Sensitivities, the primary treatment recommendation was to avoid situations and substances that would trigger her condition. (Roy Kerry, MD; Advanced Integrative Medicine, 160 E. Portersville Road, Portersville PA 16051; ph# 724-368-3558). The date for these recommendations was 1999 and ongoing (response by Beth Nickels).

Item I-5

Regarding a fact witness for this proceeding: I am still collecting information and will provide this information to you at a later date (response by Beth Nickels).

Item I-6

Regarding an expert witness for this proceeding: I am still collecting information and will provide this information to you at a later date (response by Beth Nickels).

Item I-7

I have provided copies of all exhibits I intend to present or utilize at the hearing. At this time, I do not have any witnesses who will be sponsoring any of the exhibits (response by Beth Nickels)

EXHIBIT A

July 18, 2019

Public Utility Commission
400 North Street
Keystone Bldg.
Harrisburg, PA 17120

RE: Beth Nickels, 135 Marquise Drive, PO Box 251, Tafton, PA 18464

To whom it May Concern:

Beth Nickels is a patient under my care, she is being treated for various forms of environmental hypersensitivity, including Electrohypersensitivity (EHS), the electromagnetic field from the Smart Meter would exacerbate her condition.

Electrohypersensitivity or EHS is a physiological condition. It is characterized by neurological and immunological symptoms that noticeably flare or intensify upon, or following expose to:

Electric and magnetic fields (EMF)
One or more of the types of electromagnetic radiation (EMR) found in the modern environment.

Having Electromagnetic Field Sensitivity means experiencing recurring stress or illness when near active EMF sources or emitters of EMR. Symptoms normally diminish with distance from these sources but typically require considerable time to vanish after exposure. The World Health Organization identifies this collection of symptoms and triggers as Electrohypersensitivity, often referred to as Electrosensitivity.

Sincerely,

Elaine Hardy MS, RN, APN-C

Elaine Hardy, MS, RN APN, C

319 Airport Road · Hackettstown, NJ 07840 · Ph: 908-850-0888 · Fax: 908-850-1005
ehardy@holisticfamilyhealthcarepc.com · www.holisticfamilyhealthcarepc.com

... your journey to better health.

EXHIBIT B

Family Holistic Health

Karin Cseak, DO
556 Portage Trail Ext. W.
Cuyahoga Falls, OH 44223
330-923-3060
familyholistichealth@yahoo.com
www.naturalmedicineohio.com

7/18/19

To whom it may concern,

I'm writing as physician for Beth Nickels. I have known and treated Ms. Nickels for 15+ years. Ms. Nickels has a well established history of multiple environmental sensitivities. Her daily health has been affected significantly by her condition. EMFs do seem to have a detrimental effect on her health.

Research into the effects of EMFs on health and disease are limited. However, some data can be found here: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5036708/

Studies on hypersensitive persons in particular, are lacking. The World Health Organization has ongoing studies to further our knowledge in this area. My clinical experience is that certain highly sensitive individuals do report detrimental effects on their health from exposures to EMFs. Ms. Nickels is one such person, and if any accomodations can be made to avoid such exposure, I believe she could benefit.

Please contact me if you require any further information.

Cordially,

Karin Cseak, DO

EXHIBIT C

PubMed

Format: Abstract

Full text links

ELSEVIER

Med Hypotheses. 2000 Apr;54(4):863-71.

A theoretical model based upon mast cells and histamine to explain the recently proclaimed sensitivity to electric and/or magnetic fields in humans.

Gangi S[1], Johansson O.

Author information

Abstract

The relationship between exposure to electromagnetic fields (EMFs) and human health is more and more in focus. This is mainly because of the rapid increasing use of such EMFs within our modern society. Exposure to EMFs has been linked to different cancer forms, e.g. leukemia, brain tumors, neurological diseases, such as Alzheimer's disease, asthma and allergy, and recently to the phenomena of 'electrosupersensitivity' and 'screen dermatitis'. There is an increasing number of reports about cutaneous problems as well as symptoms from internal organs, such as the heart, in people exposed to video display terminals (VDTs). These people suffer from subjective and objective skin and mucosa-related symptoms, such as itch, heat sensation, pain, erythema, papules and pustules. In severe cases, people can not, for instance, use VDTs or artificial light at all, or be close to mobile telephones. Mast cells (MCs), when activated, release a spectrum of mediators, among them histamine, which is involved in a variety of biological effects with clinical relevance, e.g. allergic hypersensitivity, itch, edema, local erythema and many types of dermatoses. From the results of recent studies, it is clear that EMFs affect the MC, and also the dendritic cell, population and may degranulate these cells. The release of inflammatory substances, such as histamine, from MCs in the skin results in a local erythema, edema and sensation of itch and pain, and the release of somatostatin from the dendritic cells may give rise to subjective sensations of on-going inflammation and sensitivity to ordinary light. These are, as mentioned, the common symptoms reported from patients suffering from 'electrosupersensitivity'/'screen dermatitis'. MCs are also present in the heart tissue and their localization is of particular relevance to their function. Data from studies made on interactions of EMFs with the cardiac function have demonstrated that highly interesting changes are present in the heart after exposure to EMFs. One could speculate that the cardiac MCs are responsible for these changes due to degranulation after exposure to EMFs. However, it is still not known how, and through which mechanisms, all these different cells are affected by EMFs. In this article, we present a theoretical model, based upon observations on EMFs and their cellular effects, to explain the proclaimed sensitivity to electric and/or magnetic fields in humans.

PMID: 10659662 DOI: 10.1054/mehy.1999.0923

[Indexed for MEDLINE]

Publication type, MeSH terms, Substance

LinkOut - more resources

EXHIBIT D

Dominique Belpomme, Christine Campagnac and Philippe Irigaray*

Reliable disease biomarkers characterizing and identifying electrohypersensitivity and multiple chemical sensitivity as two etiopathogenic aspects of a unique pathological disorder

DOI 10.1515/reveh-2015-0027
Received September 11, 2015; accepted November 2, 2015

Abstract: Much of the controversy over the causes of electrohypersensitivity (EHS) and multiple chemical sensitivity (MCS) lies in the absence of both recognized clinical criteria and objective biomarkers for widely accepted diagnosis. Since 2009, we have prospectively investigated, clinically and biologically, 1216 consecutive EHS and/or MCS-self reporting cases, in an attempt to answer both questions. We report here our preliminary data, based on 727 evaluable of 839 enrolled cases: 521 (71.6%) were diagnosed with EHS, 52 (7.2%) with MCS, and 154 (21.2%) with both EHS and MCS. Two out of three patients with EHS and/or MCS were female; mean age (years) was 47. As inflammation appears to be a key process resulting from electromagnetic field (EMF) and/or chemical effects on tissues, and histamine release is potentially a major mediator of inflammation, we systematically measured histamine in the blood of patients. Near 40% had a increase in histaminemia (especially when both conditions were present), indicating a chronic inflammatory response can be detected in these patients. Oxidative stress is part of inflammation and is a key contributor to damage and response. Nitrotyrosin, a marker of both peroxynitrite ($ONOO^-$) production and opening of the blood-brain barrier (BBB), was increased in 28% the cases. Protein S100B, another marker of BBB opening was increased in 15%. Circulating autoantibodies against O-myelin were detected in 23%, indicating EHS and MCS may be associated with autoimmune response. Confirming animal experiments showing the increase of Hsp27 and/or Hsp70 chaperone proteins under the influence of EMF, we found increased Hsp27 and/or Hsp70 in 33% of the patients. As most patients reported chronic insomnia and fatigue, we determined the 24 h urine 6-hydroxymelatonin sulfate (6-OHMS)/creatinin ratio and found it was decreased (<0.8) in all investigated cases. Finally, considering the self-reported symptoms of EHS and MCS, we serially measured the brain blood flow (BBF) in the temporal lobes of each case with pulsed cerebral ultrasound computed tomosphygmography. Both disorders were associated with hypoperfusion in the capsulothalamic area, suggesting that the inflammatory process involve the limbic system and the thalamus. Our data strongly suggest that EHS and MCS can be objectively characterized and routinely diagnosed by commercially available simple tests. Both disorders appear to involve inflammation-related hyper-histaminemia, oxidative stress, autoimmune response, capsulothalamic hypoperfusion and BBB opening, and a deficit in melatonin metabolic availability; suggesting a risk of chronic neurodegenerative disease. Finally the common co-occurrence of EHS and MCS strongly suggests a common pathological mechanism.

Keywords: biomarkers; cerebral hypoperfusion; electrohypersensitivity; limbic system; multiple chemical sensitivity.

***Corresponding author:** Philippe Irigaray, PhD, ARTAC, 57-59 rue de la convention, 75015 Paris, Phone: +33 (0)1 45 78 53 54, Fax: +33 (0)1 45 78 53 50, E-mail: philippel.artac@gmail.com; Association for Research and Treatments Against Cancer (ARTAC), F-75015 Paris, France; and European Cancer and Environment Research Institute (ECERI), Brussels, Belgium
Dominique Belpomme: Paris V University Hospital, France; and European Cancer and Environment Research Institute (ECERI), Brussels, Belgium
Christine Campagnac: Hospital Director, seconded from Assistance Publique-Hôpitaux de Paris (AP-HP), Paris, France; and European Cancer and Environment Research Institute (ECERI), Brussels, Belgium

Introduction

In 1962, Randolph first described clinically (1) what is today commonly called multiple chemical sensitivity (MCS) (2): a human pathological disorder that has been identified and defined in 1999 during an international consensus meeting on the basis of the six following criteria: "1. The symptoms are reproducible with [repeated chemical] exposure; 2. The condition is chronic; 3. Low levels of exposure [lower than previously or commonly tolerated] result in manifestations

of the syndrome; 4. The symptoms improve or resolve when the inciting agents are removed; 5. Responses occur to multiple chemically unrelated substances; 6. [Added in 1999]: Symptoms involve multiple organ systems" (3). Although the precise worldwide prevalence of MCS remains unclear, it is expected that due to the vastly increased number of the various chemical products that have been put on the market during the last few decades, MCS is becoming an increasing prevalent pathological disorder (4).

The recent rise of wireless telecommunication worldwide also confronts scientists with the question of whether anthropogenic electromagnetic fields (EMFs) such as emitted by cell phones, wireless internet, and high voltage power lines, can cause adverse health effects as it is the case for chemicals. In 1991 Rea first described what he called electromagnetic field sensitivity (5). Six years later, Bergqvist et al., in a report prepared by a European group of experts for the European Commission coined the term electrohypersensitivity (EHS) to encompass in a unique concept the clinical conditions in which EHS self-reporting patients complain of symptoms they attribute to EMF exposure (6). Since 1998, Santini et al. in France, reported symptoms experienced by users of digital cellular phones and the health risk of people living near cellular phone base stations (7, 8).

In 2004, because of the increasing worldwide prevalence of EHS, the World Health Organization (WHO) organized an international scientific workshop in Prague (Czech Republic) in order to define and characterize EHS. Although not acknowledging EHS as being caused by EMF exposure, the Prague working group defined EHS as "a phenomenon where individuals experience adverse health effects while using or being in the vicinity of devices emanating electric, magnetic, or electromagnetic fields … whatever its cause, EHS is a real and sometimes a debilitating problem for the affected persons" (9). However, following this meeting, WHO proposed to use the alternative term "idiopathic environmental intolerance (IEI) attributed to electromagnetic fields" (IEI – EMF), indicating there is no proven causality between the occurrence of IEI – EMF (formerly EHS) and EMF exposure (9).

In view of the poor knowledge of pathogenesis and etiology of EHS and MCS, most mainstream medical, sanitary and societal bodies maintain there is not sufficient scientific proof to support the concept that clinical symptoms experienced by EHS and/or MCS self-reporting patients are really caused by EMF and/or chemical exposure, respectively. This is particularly the case for EHS patients, for whom in comparison to sham controls, the reproduction of clinical symptoms in the presence of EMFs have globally failed to demonstrate a causal link, in blind or double-blind studies (10).

Moreover, the lack of recognized disease biomarkers objectively characterizing EHS and MCS has resulted in clinical symptoms being dismissed as psychogenic; and/or EHS and MCS are conflated with psychosomatic or psychiatric diseases, and not recognized as true organic disorders caused by the environment (11–16). This is particularly the case for radiofrequency EMF, for which some scientists believe that EHS is an uncertain and confusing concept (17); whereas some others, on the basis of their own clinical experience agree that excessive exposure may cause EHS (5, 18, 19).

Here, we present our own experiences based on the preliminary analysis of a series of 1216 consecutive investigated cases of self-claimed EHS and/or MCS, in the framework of an ongoing prospective clinical study aiming at identifying and characterizing EHS and MCS both clinically and biologically; through the use of biomarkers detected and measured in the peripheral blood and the urine of patients. Our data clearly shows that EHS and MCS should be recognized as genuine somatic pathological entities; that patients with EHS and/or MCS are non-psychosomatic nor psychiatric patients; and probably that EHS and MCS are two etiopathogenic aspects of a single pathological disorder.

Search for reliable disease biomarkers

The identification and measurement of reliable biomarkers is a crucial step for identifying and characterizing diseases. This is *a fortiori* the case for any new pathological entity or clinical syndrome such as MCS, EHS or other environmental intolerance syndrome. However, to our knowledge, such an approach has proven inconclusive for MCS (20) and EHS (21).

We thus searched for characteristic biomarkers and selected a battery of biological tests which could be routinely used clinically in environmental medicine practice for taking care of EHS and/or MCS self-reporting patients.

In addition, due to the reported clinical symptoms, we systematically measured the brain blood flow (BBF) in both cerebral hemispheres of these patients by using echo-doppler of the middle cerebral artery (22) and measured centimeter by centimeter brain pulsatility by using pulsed ultrasound-based cerebral computerized tomosphygmography, which allows centimetric resolution pulsed ultrasound recording of cerebral pulsatility (23–25), to localize more precisely the BBF in the different areas of the two temporal lobes. Our working hypothesis was that under

the influence of environmental factors such as EMFs and/or chemicals, some neuro-inflammation and oxidative stress might occur in the brain, with blood-brain barrier (BBB) disruption as a consequence.

We thus routinely measured the inflammation-associated high-sensitivity C reactive protein (hs-CRP) in the peripheral blood; and levels of vitamin D2-D3, as it has been suggested that low levels of its metabolite, the secosteroid 25 hydroxy-vitamin D (25-D) could be a consequence rather than a cause of inflammatory and/or autoimmune processes (26), and that vitamin D deficiency is associated with abnormal development and functioning of the central nervous system (CNS) (27, 28). Since it has been shown that upon brain injury, degeneration or infection, the inflammatory response may trigger degranulation of mast cells, leading to a massive release of histamine in the blood (29), we systematically measured the levels of histamine in the peripheral blood. In addition, as the best known mast cell degranulation mechanism involve crosslinking of the high affinity surface IgE receptor (30), we also measured total IgE levels in the peripheral blood. It is well known that histamine is a potent mediator of inflammation and is able to increase BBB permeability through oxidative and/or nitrosative stress (31, 32). So we looked for possible oxidative and/or nitrosative stress-related biomarkers of BBB disruption; and identified nitrotyrosine (NTT), because it results from the toxic effects of peroxynitrite ($ONOO^-$) on proteins (33–36). Such a BBB opening marker has also been shown for the calcium-binding protein S100B, produced and released predominantly by peri-vascular astrocytes (37–40). During the inflammatory process, it is well known that cells produce excessive amount of superoxide ($O_2^{\cdot}$) and nitric oxide ($NO^{\cdot}$), and that although $NO^{\cdot}$ is a weak free radical resulting from the action of nitric oxide synthase, its excessive intracellular production is associated with cytotoxic properties because of the formation of extremely reactive nitrogen species such as peroxynitrite. The biochemical reaction in the form of $O_2^{\cdot}+NO^{\cdot}=>ONOO^-$ may thus explain why NTT (which results from oxidative and nitrosative stresses) is associated with BBB disruption (32, 41). Dosage of free NTT and protein-combined NTT as well as protein S100B in the peripheral blood of EHS and/or MCS patients was thus an important element of the battery of biological tests we used.

We also considered that non thermal radiofrequency often is a repetitive stress leading *inter alia* to continuous heat shock protein (HSP) over-expression and release in exposed tissues, particularly in the brain (42–46). HSPs are a family of highly conserved proteins with chaperone functions acting to maintain the structural conformation of cellular proteins. Their over-expression under stress conditions which promotes an inflammatory response is well known (47–49). We thus speculated that the major inducible stress protein HSP70, which has been shown to oppose to neuronal apoptosis (50, 51) and to BBB disruption (51, 52), so eliciting some neuroprotection could be involved as it could be also the case for HSP27 (53, 54). However, under chronic EMF exposure it was reported that, as compared to controls, intracellular HSP70 levels may decline (55). We thus systematically measured HSP70 and HSP27 levels in the peripheral blood of EHS and/or MCS patients in order to try to determine whether these chaperone proteins are a marker of EMF and/or chemicals chronic exposure; as it has been shown for non-thermal EMF exposure in experimental studies (42–46).

Moreover, during oxidative and nitrosative stress proteins may be extensively modified and denatured and so acquire new epitopes which can explain their loss of specificity and biological activity, hence the synthesis of autoantibodies (56, 57). This is the case for EMF exposure which has been shown to alter DNA replication and mitosis and form abnormal proteins (42, 58, 59) and so to produce electro-oxidation-related IgE autoantibodies (60). We consequently hypothesized that under the influence of environmental EMFs and/or chemicals, CNS proteins such as O-myelin may be so denatured that they acquire autoantigenic properties. Consequently we thus systematically searched for and measured autoantibodies against O-myelin in the blood of patients.

Finally, since some effects of EMF exposure have been reported to be mediated by the pineal hormone, melatonin (61), and given the fact that in our series many patients had sleep disturbance, we also systematically measured melatonin metabolism in these patients. However, as measurement of endogenous melatonin in urine is not useful because of its low unmetabolized levels (62), we measured levels of its metabolite 6-hydroxymelatonin sulfate (6-OHMS) and creatinine in 24 h urine, to determine the 6-OHMS/creatinine ratio. Note that since creatinine is excreted in a relatively constant amount in each patients, we used this ratio to reduce the variability of 6-OHMS measurement attributed to urine dilution.

The test battery for identifying and characterizing EHS and MCS is summarized in Table 1. Technical information about the methods we used for carrying out all biological tests and the BBF analysis are summarized as follows:

For the biomarker study, all patients were investigated by using commercially available biochemical tests and values for each patient were compared to the normal reference values obtained from the commercial companies. Sensitivity, specificity and reproducibility of these tests were thus those defined by these companies. Each

Table 1: Disease biomarkers investigated in self-reporting EHS and/or MCS patients with their normal values.

Biomarker	Normal range
High-sensitivity C reactive protein (hs-CRP)	≤3 mg/L
Vitamin D2-D3	≥30 ng/mL
Histamine	≤10 nmol/L
IgE	≤100 UI/mL
Protein S100B	≤0.105 µg/L
Nitrotyrosine (NTT)	≥0.6 µg/L and ≤0.9 µg/mL
Heat shock protein 70 (HSP70)	≤5 ng/mL
Heat shock protein 27 (HSP27)	≤5 ng/mL
Anti-O-myelin autoantibodies	Negative
Hydroxy-melatonin sulfate (6-OHMS)	≥5 ng/L and ≤40 ng/L
6-OHMS/creatinine	≥0.8 and ≤8

assay was performed according to the manufacturer's method. Hs-CRP and 25-OH vitamin D were measured by using an automated immunoassay [Architect CI 4100 (Abbott Laboratories, Abbott Park, Chicago, IL, USA)]; for Histamine measurement we used an ELISA specific test; for protein S100B, a quantitative automated chemiluminescent immunoassays [Liason S100 (DiaSorin Deutschland GmbH, Dietzenbach, Germany)]; for NTT, a competitive ELISA test (Cell Biolabs Inc., San Diego, CA, USA); for anti-O-myelin antibody detection, a Western Blot qualitative analysis (IMMCO Diagnostics, Buffalo, NY, USA); for HSP 27 and HSP 70, specific high sensitivity enzymatic immunoassays (Stressgen Biotechnologies Corporation, San Diego, CA, USA); and for 5-hydroxy-melatonin-sulfate, a urine ELISA test (IBL International GmbH, Hamburg, Germany).

In addition, to these biochemical tests we used a non-invasive ultrasonic cerebral tomosphygmography method that we specifically set-up to investigate the blood flow in the patient temporal lobes and determined for each patient a pulsometric index (PI) that we measured centimeter by centimeter from the cortex to the diencephalic medial area (see Figure 1). This index varies between the territories studied. In this study, PI determination for each cerebral territory in 727 EHS and/or MCS patients was compared to a retrospective series of 141 normal subjects which allowed to establish the normal median reference values of PI (see Figure 2). Finally since our study is still ongoing we did not reported any statistical analysis. This will follow in specific further papers.

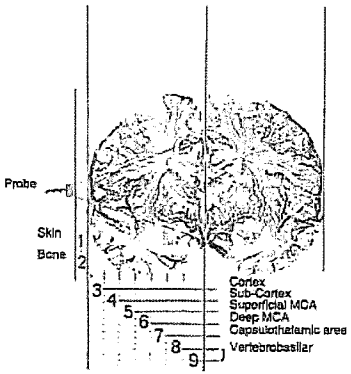


Figure 1: Pulsometric index (PI) obtained by a computerized ultrasonic cerebral tomosphygmography (UCTS) in the different area of temporal lobes.
Data are expressed as mean pulsometric index (PI). PI varies between territories studied: 3+4 correspond to cortico sub-cortical area; 3+4+5, to the superficial area of the middle cerebal artery (MCA); 5+6+7, to the deep area of the MCA; 7, to the capsulothalamic area; 3+4+5+6+7, to the complete area depending of the MCA; 8+9, to the vertebrobasilar area; 3+4+5+6+7+8+9, to the complete temporal lobe.

Search for clinical diagnosis criteria

In 2009, at the time we initiated this prospective cohort study, we were aware there was no available recognized biological markers for defining objectively EHS and MCS; this led us to use clinical criteria as inclusion criteria. For MCS, as already above mentioned, we used the six criteria that had been reported in the 1999 International workshop (3) and for EHS, we used similar criteria. However, as in an unpublished feasibility study we showed that many EHS patients when they are in the vicinity of chemicals may present with olfactory abnormalities consisting in subjective odor disruption; we systematically added a seventh clinical criteria to the six ones already defined during the 1999 consensus meeting on MCS. In order to further characterize clinically MCS and distinguish it from EHS. Accordingly patients with MCS, unlike EHS patients, were characterized not only by the simple odor intolerance, but more specifically by symptoms of mucous inflammation in the nose, the oropharynx and/or the laryngo-trachoo-bronchus tract; manifesting clinically as rhinitis, oropharyngeal dysesthesia or laryngitis and/or bronchospasms, respectively.

To further avoid any confounding pathology, all patients of the present prospective series have been

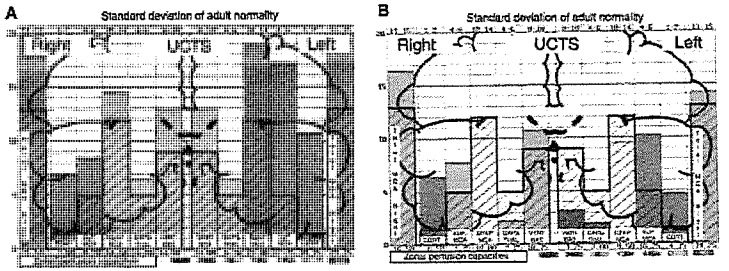


Figure 2: Example of diagrams obtained by using UCTS exploring the global centimetric ultrasound pulsatility in the two temporal lobes of a normal subject (A) and in a EHS self-reporting patient (B).
Measurements are expressed in Pulsometric Index (PI). Note that in A and B mean values of PI in each explored area recorded is from the cortex to the internal part of each temporal lobe; so on the left part of the two diagrams A and B for the right lobe from the left to the right; and on the right part of these diagrams for the left lobe from the right to the left. Note also that in A (normal subject) all values are over the normal median values whereas in B (EHS-self reporting patients) values in the capsulothalamic areas (the fifth and the second column for the right and left temporal lobe, respectively) are under the normal median values.

interviewed face to face at length during their medical consultation and questioned systematically about their past medical history and the type and conditions of occurrence of their clinical symptoms, thanks to the use of a validated pre-established questionnaire. In addition, all patients have been carefully physically examined. Also, before inclusion, all patients were systematically investigated by usual routine blood tests and medical imaging including Brain MRI and/or scanner and carotid echodoppler in order to eliminate any known unrelated CNS pathology.

Finally, based on the above clinical finding for both EHS and MCS patients we used the following inclusion criteria:

1. Absence of known pathology accounting for the observed clinical symptoms;
2. Reproducibility of symptom occurrence under the influence of EMFs and/or multiple chemicals whatever their incriminated source;
3. Regression or disappearance of symptoms in the case of EMF and/or multiple chemical avoidance;
4. Chronic evolution;
5. Symptoms such as headache, superficial and/or deep sensibility abnormalities, skin lesions, sympathetic-nerve dysfunction, reduced cognitive ability including loss of immediate memory and attention and/or concentration deficiencies, insomnia, chronic fatigue and depressive tendency, all main clinical symptoms reported as non-specific symptoms in the scientific literature (13, 19), but which when grouped together may evoke clinically the diagnosis of EHS (data not shown);
6. No serious pre-existing pathology such as atherosclerosis, diabetes, cancer; and/or neurodegenerative or psychiatric diseases which have been associated with EHS and/or MCS in the past or at the inclusion time but would render difficult the interpretation of clinical symptoms and biomarker data (see Section "EHS/MCS as a possible sentinel pathological disorder"); and finally
7. For each patient written informed consent.

Study of this large cohort of patients was not a case-control study neither a randomized study so there was no specific control group.

As depicted in Table 2, on a total of 1216 investigated consecutive cases, 839 are presently analyzed of whom 727 are evaluable, 521 with EHS (71.7%), 52 with MCS (7.1%) and 154 with both EHS and MCS (21.2%), regardless of whether MCS occurred before or after EHS. Only 29 patients, i.e. 3% claimed to suffer from EHS and/or MCS but did not meet the inclusion criteria. In fact most of these patients claimed to be electrohypersensitive. Although many of them were associated with a putative neurologic or psychiatric disorder, EHS could not be clearly established. Also excluded were patients with EHS and/or MCS who were in addition, diagnosed as suffering from heavy pathology evidenced after inclusion, or who were lost to follow-up, or for whom results of the biological investigation were not available at the time of analysis.

Table 2: Summary of the present ongoing prospective clinic-biological study of EHS and/or MCS-self reporting patients.

Patients groups	Total	EHS	MCS	EHS/MCS
Total investigated	1216			
Total presently analyzed	839			
Neither EHS nor MCS	29			
Not evaluable	83			
Evaluable	727	521	52	154
Sex ratio	495 W/232 M	344 W/177 M	34 W/18 M	117 W/37 M
	68%/32%	66%/34%	65%/35%	76%/24%
Mean age	47.9±12.4	48.2±12.9	48.5±10.3	46.7±11.2
Median age [range]*	47 [16–83]	48 [16–83]	47 [31–70]	46 [22–76]

*The range of values is indicated in square brackets, e.g. [minimum-maximum].

Demographic panorama

This large cohort of investigated patients originated from many different European countries, and from other countries worldwide such as the US, Canada, Australia, Russia, China, Middle East and Africa. This allows some estimation the demographic picture of so called EHS and/or MCS patients. The demographic data are depicted in Table 2 and Figure 3.

A noteworthy finding which was observed in many countries is that women appear to be much more susceptible to EHS and/or MCS than men, since in our series two thirds are female, with no difference between EHS and MCS rates. Note however, that the female predominance appears to be more pronounced for patients with both EHS and MCS, where three out of four are female (Table 2).

In this series, median age is about 47 years and does not differ according to EHS, MCS and EHS/MCS diagnosis. As indicated in Figure 3, all age categories are represented and mainly include young and old adults, but it appears that adolescents may be also associated with EHS. This may be due to their excessive use of wireless technology (essentially mobile phones and other devices) at this age. In fact, outside of the present series, we have observed that infants and children could also be suffering from EHS.

Analysis of biochemical markers

Biomarker results are indicated in Tables 3–5 and in Figure 4.

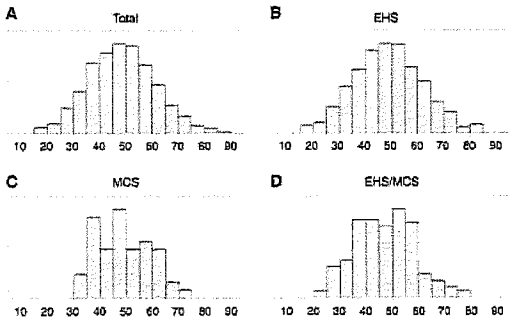


Figure 3: Age categories according to the total number of evaluable patients (A) and to the three EHS (B), MCS (C) and EHS/MCS (D) analyzed groups of patients.

Table 3: High-sensitivity C reactive protein (hs-CRP), immunoglobulin E (IgE), vitamin D2-D3 and histamine in the peripheral blood of EHS and/or MCS self-reporting patients.

Patients groups	EHS	MCS	EHS/MCS
n	521	52	154
hs-CRP	78 (14.97%)	3 (13.46%)	22 (14.28%)
>3 mg/L	[3.27–51.91]	[3.5–10]	[3.27–21.61]
Vitamine D	33 (6.33%)	5 (9.62%)	16 (10.39%)
<10 ng/mL	[4.81–9.70]	[4.80–8.00]	[7.10–9.90]
Vitamine D	300 (57.58%)	25 (48.07%)	92 (59.74%)
≥10 ng/mL and <30 ng/mL	[10.40–29.70]	[10.70–27.90]	[15.00–28.60]
Histamine	182/491 (37%)	16/44 (36.7%)	59/142 (41.5%)
>10 nmol/L	[10.08–360.00]	[10.80–90.00]	[10.10–1797.50]
IgE	115 (22.07%)	8 (15.38%)	38 (24.68%)
>100 UI/mL	[101–1387.60]	[131.10–294.87]	[103.30–1200.00]

Note that for each biomarker the range of values is indicated in square brackets, e.g. [minimum-maximum].

Table 4: Protein S100B and nitrotyrosin (NTT) in the peripheral blood of EHS and/or MCS self-reporting patients.

Patients groups	EHS	MCS	EHS/MCS
n	521	52	154
S100B	73/495 (14.7%)	6/51 (19.7%)	28/142 (10.7%)
>0.105 µg/L	[0.105–2.090]	[0.110–0.500]	[0.110–0.470]
NTT	77/259 (29.7%)	6/29 (26%)	22/76 (28.9%)
>0.9 µg/mL	[0.92–8.20]	[1.10–3.10]	[0.91–3.10]
Increased S100B and/or NTT	133/250 (53.2%)	12/22 (54.5%)	46/73 (63%)
Increased histamine, S100B and/or NTT	220/327 (71.8%)	27/36 (75%)	91/125 (79.1%)

Note that for each marker the range of values is indicated in square brackets, e.g. [minimum-maximum].

Table 5: HSP70 and HSP27 chaperone proteins and anti-O-myelin autoantibodies in the peripheral blood of EHS and/or MCS self-reporting patients.

Patients groups	EHS	MCS	EHS/MCS
n	521	52	154
Hsp 70	91/486 (18.7%)	4/52 (7.7%)	36/142 (7.6%)
>5 ng/mL	[5.90–11.20]	[7.10–7.70]	[5.20–32.20]
Hsp 27	123/476 (25.8%)	6/52 (11.5%)	42/132 (11.5%)
>5 ng/mL	[5.20–11.20]	[5.90–9.20]	[5.10–25.00]
Hsp70 and/or Hsp27	162/487 (33.3%)	9/52 (25%)	56/142 (39.4%)
Anti-O-myelin autoantibodies	109/477 (26.8%)	8/47 (17%)	33/140 (23.4%)

Note that for each marker the range of values is indicated in square brackets, e.g. [minimum–maximum].

High-sensitivity C reactive protein (hs-CRP)

An increase in hs-CRP levels was found globally in 107 patients (14.7% of the cases), and more precisely in 78 patients (15%), seven patients (13.5%) and 22 patients (14.3%), respectively in the three EHS, MCS, EHS/MCS individualized groups (Table 3); suggesting that in such cases, patients were associated with some type of systemic inflammation. We thus systematically looked for unrelated causes of inflammation and/or infection in these patients, but with the exception of three cases, we did not found any. Furthermore, since hs-CRP is considered as a biomarker of age-related cognitive decline or dementia, and more particularly of Alzheimer's disease (63, 64), we systematically searched for Alzheimer's disease in these patients. In two cases, Alzheimer's disease was discovered after inclusion and considered as possibly the results of excessive past EMF exposure (see Section "EHS/MCS as a possible sentinel pathological disorder"). But, because chronologically, Alzheimer's disease appeared to follow the initial occurrence of EHS, we considered that for these two patients, Alzheimer's disease might have been the consequence of EHS rather than simply associated with it. Nevertheless, these two cases were categorized as non-evaluable cases in the present analysis.

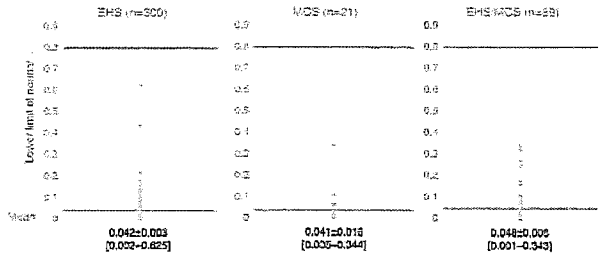


Figure 4: 24 H urine 6-OHMS/creatinine ratio in EHS and/or MCS self-reporting patients.

Vitamin D2–D3

As indicated in Table 3, a profound decrease in the levels of the secosteroid 25-D is found globally in 184 patients (25.3% of the cases), and in 121 patients (23.2%), 12 patients (23.1%) and 51 patients (33.1%) in the three groups, respectively. As already discussed (see Section "Search for reliable disease biomarkers"), these data agree with the concept that decrease in vitamin D2–D3 levels appear to be a consequence rather than a cause of inflammation and so need to be therapeutically normalized.

Histamine

An important finding in our study is the discovery that histamine in the peripheral blood is increased in nearly 40% of the patients and that this increase does not differ between the three groups investigated (Table 3). This finding suggests that histamine is not only a natural clinical biomarker of EHS and MCS, but also may play a crucial role in the pathogenesis of both clinical entities, since it has been shown to be not only a neurotransmitter produced and released by the CNS, but also an inflammatory mediator produced and released by mast cells in many inflammatory processes including neuro-inflammation (see Section "Pathophysiological relevance").

IgE

Levels of circulating IgE were found to be increased in 22%, 15.4% and 24.7% of the three EHS, MCS and MCS/EHS groups, respectively. Since histamine release from mast cells involve the high affinity IgE mast cell surface receptor and IgE (30, 65), we searched for a correlation between histamine and IgE levels in the peripheral blood of the patients. As it will be further discussed, it seemed not to be the case (see Section "Pathophysiological relevance").

Protein S100B

Levels of circulating protein S100B have been found to be globally increased in 107 patients (15.5%), with no differences between the three groups (Table 4). As we will discussed (see Section "Some insight into etiopathogeny") this finding confirms previously reported data showing the glia-derived S100B protein is a biomarker of hypoperfusion-associated brain damage or dysfunction (39, 40, 66–68), and more particularly of neurodegenerative diseases such as Alzheimer's disease (69) and amyotrophic lateral sclerosis (70); but differs from the negative results obtained in non EHS healthy subjects for whom protein S100B levels has been shown to be normal within the 2 h following GSM mobile phone use (71–73).

Nitrotyrosin

Likewise, increased NTT blood levels have been detected globally in 105 patients (29%), with no difference between the three groups. Moreover, as indicated in Table 4, it appears that increased levels of protein S100B and/or NTT can be detected in approximately 55%–60% of the cases.

Since, as previously indicated, protein S100B and NTT could be potential markers of BBB disruption, we consider that such disruption could be evidenced clinically in over 50% of the patients, whatever their EHS and/or MCS clinical presentation.

HSP70 and HSP27

As indicated in Table 5, depending of the group considered, increased levels of the HSP70 and HSP27 chaperone proteins were detected in the peripheral blood in about 7%–19% and about 11%–26% of patients, respectively. Collectively, 25%–40% of the patients were found to be associated with increased levels of HSP70 and/or HSP27, without difference between the 3 so far individualized groups, meaning that HSP70 and HSP27 are circulating biomarkers not only of EMF chronic exposure as it is the case in animal experimental studies (42–46) but also of chemical chronic exposure. HSP70 and HSP27 seem to be more frequent in EHS patients than in MCS patients.

Autoantibodies against O-myelin

As indicated in Table 5, autoantibodies against O-myelin have been detected globally in 17% to nearly 29% of the patients studied with no difference between the three groups, suggesting that in these patients EHS and/or MCS were associated with some type of autoimmune response. Here too, it is more frequent in EHS than in MCS.

Melatonin

6-OHMS and creatinine were measured in the 24 h urine of a number of patients. As indicated in Figure 4, all investigated patients had a decrease in the 6-OHMS/creatinine ratio; suggesting that these patients have decreased antioxidant defenses (74, 75), and so may be at risk of chronic diseases (see Sections "Pathophysiological relevance" and "EHS/MCS as a possible sentinel pathological disorder"). Moreover, this decrease might explain why such patients present sleep disturbance.

Clinical forms of EHS and/or MCS without detectable biomarkers

Increase in hs-CRP and vitamin D2–D3 blood levels are non-specific biological parameters. On the other hand, although none of our biomarkers are per se specific (see Section "Some insight into etiopathogeny") the increased serum level of histamine, protein S100B and NTT in the peripheral blood seems more characteristic of EHS and MCS, because of their pathophysiological relevance. However, as indicated in Table 4, increased levels of histamine, protein S100B and/or NTT were found in only 70%–80% of the patients, meaning that in 20%–30% of the cases in our series, EHS and MCS could not be objectively characterized by these biomarkers. However, in such patients in addition, to the clinical picture the objective diagnosis of EHS and/or MCS could still be made based on the abnormal recording of brain pulsed ultrasound computed tomosphygmography.

Pathophysiological relevance

In our study we have shown that EHS and MCS both are associated with the same biological abnormalities. This strongly suggests that both pathological entities share a unique common pathophysiological mechanism.

Since histamine was found to be increased in the peripheral blood of nearly 40% of the patients, this molecule appears to be a key pathogenic mediator, whatever the environmental stressors. Indeed, the fact that histamine levels were not found to be increased in all patients doesn't mean that patients for whom there is no histamine blood level increase have no local histamine production and release in their tissues or at other times. Moreover, we will outline below that histamine is not just a neuro-inflammation mediator. Histamine plays a critical pathophysiological role as a neurotransmitter in the brain. For example neuronal histamine has been shown to be involved in the sleep cycle, motor activity, synaptic plasticity and memory (76–79); all types of neurologic and/or psychologic altered functions or symptoms that we have observed clinically in EHS and/or MCS bearing patients (data not shown). In addition, histamine release from sympathetic nerves can be experimentally induced by nerve stimulation (80) and it has been shown that H1 receptor may play a major role in the regulation of sympathetic nerve activity (81). This may explain why EHS and/or MCS patients may present clinically with some transitory sympathetic-related symptoms such as tachycardia, tachyarrhythmia and/or arterial pressure instability (data not shown) when exposed to EMF and/or chemical stressors (82). Moreover, following ischemic-hypoxic damage, histamine release from nerve endings has been found to be enhanced, possibly contributing to some neuroprotection (83).

However, histamine is also a unique molecule which fulfils all criteria that have been historically established for defining an inflammatory mediator (84). Histamine is mainly produced and stored in perivascular tissue resident mast cells and circulating basophils, and released in inflammatory tissues through established mechanisms predominantly involving cell surface receptors. Regarding histamine release from skin mast cells, the best known degranulation mechanism involves IgE and the high affinity IgE cell surface receptor (30).

In our study, we found elevated levels of circulating IgE in about 20% of the patients, whatever the EHS and/or MCS group considered. However, in such cases, we didn't find any positive correlation between the levels of circulating histamine and the levels of circulating IgE nor the presence of skin lesions. This suggests that skin lesions and circulating histamine level increase in EHS and/or MCS patients are not related to an allergic process.

Also it has been shown that advanced glycation end products (AGEs) can activate mast cells through RAGE, the receptor of AGEs, and may contribute to initiating a vicious circle involving increased AGE formation and ROS production, hence increased low-grade chronic inflammation (85). Similar biological effects may also be obtained with protein S100B which has been shown to engage RAGE in macrophage/microglia and endothelial cells; and so depending of its extracellular concentration, to contribute either to chronic inflammation via NFκB activation or to anti-apoptotic effects and trophic protection in the course of pathological conditions such as brain insult or diabetes (86). Since AGEs have been shown to be involved in diabetes mellitus (87) although all included patients had no diabetes type II at inclusion time, we systematically search for a possible occurrence of diabetes type II in EHS and/or MCS patients during the follow up of this study, but with the exception of two cases, all patients were free from diabetes.

Predominantly found at host/environment interfaces such as skin, respiratory and gastrointestinal tracts (88) and closely associated with blood vessels, mast cells play a crucial sentinel role in host defense (89). Consequently, more precise investigations remain to be done in EHS and/or MCS patients to determine what mast cell-associated tissue histamine release come from.

However, since brain mast cells have been shown to be critical regulators of the pathogenesis of CNS diseases including stroke, traumatic injury and neurodegenerative diseases (83, 90) (see also Section "EHS/MCS as a possible sentinel pathological disorder") we systematically looked for brain pathological alterations in EHS and/or MCS patients. Routine cerebral MRI and/or scanner as well as carotid echography were critically considered to be normal in all evaluable cases. We thus measured the BBF-related pulsatility in the patient hemispheres by using echodoppler of the middle cerebral artery, and found that resistance index and systolic and diastolic velocity indexes were associated with cerebral hypoperfusion in one or the two hemisphere in 50.5% of the cases, whatever the patient group considered (data not shown). More precisely, by using pulsed ultrasound computed tomosphygmography, we found that in comparison to normal subjects, cerebral pulsatility in EHS and /or MCS patients was decreased or even completely abolished in one or the two temporal lobes (Figure 2), suggesting that BBF might be specifically decreased or abolished in this brain area. We found that this abnormality, although being not specific, was so frequently observed in these patients that it may represent a typical brain alteration similar to that found in Alzheimer's disease and other neurodegenerative diseases (see Section "EHS/MCS as a possible sentinel pathological disorder"). This finding therefore, strongly suggests that brain could be the main target of environmental EMFs and/or chemicals in EHS and/or MCS patients, and that both cerebral hypoperfusion and subsequent histamine release whatever its neuronal or mast cell origin could be main contributing factors to BBB disruption. Furthermore, we found that cerebral blood pulsatility was quasi-constantly decreased in the capsulothalamic area of the temporal lobes, which includes the *limbic system* and the *thalamus*, and so correspond to particularly vulnerable areas to environmental stressors in the brain.

Confirming this capsulothalamic hypothesis, it has been shown that experimentally-induced brain ischemia-hypoxia can increase BBB permeability (91–94) and that hippocampal pathology arising after chronic hypoperfusion can give rise to cognitive impairment and more particularly memory deficit (95), a pathophysiological mechanism that supports both the key role of cerebral hypoperfusion/hypoxia in neurodegenerative diseases such as Alzheimer's disease (96) and our clinical observation of frequent cognitive defects in EHS and/or MCS patients. How cerebral hypoperfusion/hypoxia may arise from the neuro-inflammation process remains however, unclear. Cerebral blood flow restriction and consequently impaired oxygen supply may occur due to local oedematous swelling, artery and/or capillary vasoconstriction and/or increased BBB permeability induced by histamine or other neuro-inflammation mediators (97, 98). While hypoxia itself rather than ischemia can induce histamine release (99). In addition, less efficient oxygen utilization due to mitochondrial uncoupling may be associated with impaired oxygen supply (100). As a consequence of hypoxia and impairment of mitochondrial functioning, reduced sensorial excitability, hence transitory loss of

motor, sensory and cognitive function may occur during EHS and/or MCS processes; but this loss of function may progress to permanence and universality in the case of chronic neurodegenerative diseases (97, 101).

Under the influence of environmental stressors, not only mast cells (102, 103), but also microglia cells and astrocytes (31, 104–106) play a crucial role in BBB disruption. Indeed the resident CNS tissue macrophages glial cells such as microglia cells and astrocytes, and the resident CNS mast cells are probably the first cells to respond to any neuro-inflammatory stimuli. In addition, it has been shown that tachykinin peptides such as substance P. can trigger microglial activation and subsequent release of proinflammatory molecules, thereby contributing in addition, to mast cells to the development of microglia-mediated inflammation and BBB break down (107–109). It is indeed well known that under the influence of neuro-inflammatory stressors, such as EMF and particularly during mobile device (GSM) prolonged exposure, microglia cells can migrate to the site of injury, proliferate and recruit astrocytes (110), what is commonly called gliosis – a first cellular neuro-inflammation response which produces and releases $NO^{\bullet}$, ROS and inflammatory mediators (105, 111). Moreover, astrocytes express histamine receptors (112) which after activation can trigger release of cytokines, which are themselves able to induce histamine release through mast cell degranulation in positive feedback loop (113). Finally, our finding of both cerebral hypoperfusion and histamine release, supports previous data according to which BBB disruption is obtained more efficiently when these two factors are combined (91).

At a molecular level it has been evidenced that histamine and other neuro-inflammation mediators induce oxidative and nitrosative stress and so change the molecular composition and functional state of the BBB endothelial tight junctions, hence increasing permeability of the BBB (32, 104, 114, 115). As a consequence of this process circulating inflammatory cells may thus transmigrate into the CNS and so amplify the neuro-inflammation response (116, 117). Note that such oxidative/nitrosative stress-induced BBB disruption has not only been evidenced as a consequence of chronic cerebral hypoperfusion (118) but also proved to occur under the influence of EMF exposure at non thermal as well as thermal levels in several animal studies (104, 119–122).

Melatonin suppression as a consequence of EMF exposure has been experimentally evidenced both in animals and humans (123–125). We found that 6-OHMS 24 h-urine excretion was decreased in all the investigated cases, whatever the EHS and/or MCS patient group considered. Although this finding suggests that melatonin production might have been decreased in these patients, EMF exposure have been reported to be incapable of altering melatonin synthesis and secretion (126). So an alternative plausible explanation is that decrease in 6-OHMS excretion may reflect decreased melatonin metabolic availability, due to an increased uptake and utilization of melatonin as a free radical scavenger (127, 128). Such reduction in melatonin bioavailability may thus contribute to decrease host defence mechanisms and may account for the fact that patients submitted to prolonged and intensive EMF exposure may be at risk of neurodegenerative diseases and cancer (129), particularly of breast cancer (130) (see Section "EHS/MCS as a possible sentinel pathological disorder").

The development of the oxidative/nitrosative stress-related autoimmune response may also contribute to weaken the protective effect of the chaperone proteins HSP70 and HSP27 (131) as has been evidenced for example in stroke patients (132). Indeed the role of histamine in modulating the immune system (133), the disturbance of the immune system by EMFs (134) and the progressive increase in oxidative and nitrosative stress as long as chronic exposure to EMFs and/or chemicals persists may explain why the physiological defence mechanisms of these patients may finally collapse.

On the basis of our data we therefore, propose the following pathophysiological model of co-MCS/EHS exposure: 1) Under the influence of EMFs and/or chemicals a cerebral hypoperfusion/hypoxia-related neuro-inflammation may occur; 2) Due to the release of histamine and other mediators BBB disruption and permeability increase may be induced through resulting oxidative and/or nitrosative stress; 3) Circulating inflammatory cells could then enter the brain to initiate a vicious circle which may considerably amplify the neuro-inflammation process; and finally 4) Because of oxidative and nitrosative stress and subsequent decreased melatonin bioavailability and autoimmune response, physiological defence mechanisms are weakened making EHS and/or MCS patients potentially at risk of chronic neurodegenerative diseases and cancer.

Part of this model has been proposed separately for histamine release from mast cells in EHS (135) and for the NO/NOOH- nitrosative stress cycle in MCS (136). Our proposed EHS/MCS common pathogenic model is summarized in Figure 5.

Some insight into etiopathogeny

Certainly this study does not prove a causal link between EMFs and EHS, or between chemicals and MCS, but it does strengthen the evidence for such a possibility. To our knowledge this is the first time that EHS and/or MCS have been objectively characterized by the use of several different types of biomarkers and in a large prospective series of patients. This finding should avoid the frequent erroneous interpretation that EHS and/or MCS patients are psychosomatic patients (11–14, 17, 137) and so strongly suggests that EHS and MCS are genuine somatic pathological entities. Furthermore, our study revealed that with the exception of the two cases of Alzheimer's disease which were detected soon after inclusion, and several other cases of neurodegenerative diseases which were diagnosed during the follow-up (these cases have been considered as non-evaluable cases) (see Section "EHS/MCS as a possible sentinel pathological disorder"), all EHS and/or MCS patients had no detectable psychiatric disease.

As previously mentioned we should however, note that none of the biomarkers so far identified in our study are specific of EHS and/or MCS. This is the case for histamine which is known to be increased in the serum of patients with typical migraine (138–140) and/or allergy (30) and for HSP70 and HSP27 which has been shown to be increased in several neurodegenerative diseases (141, 142); and for protein S100B which acts normally as a physiological intracellular regulator and extracellular signal and so has been shown to be expressed and released not only by damaged CNS cells such as glial cells and neurons, but also by different non CNS cells such as chondrocytes, adipocytes, melanocytes, myofibers and other non CNS cells (67, 86, 143). This explains why the detection of increased levels of protein S100B in the serum of patients does not mean they are necessarily EHS and/or MCS patients. Other pathological disorders such as neurodegenerative diseases, psychiatric diseases such as bipolar disorder or cancer (70, 144, 145) may be indeed also concerned by such S100B protein levels. Likewise NTT is not only a general marker of inflammation but also more particularly a marker of atherosclerosis (146). Increased levels of NTT are thus also non-specific. As already indicated (see Section "Search for reliable disease biomarkers") we therefore, paid attention for excluding from our series all cases associated with neuropsychiatric diseases and/or other serious pathologies such as atherosclerosis and type 2 diabetes in order to eliminate any confounding factors.

Unlike the reported negative result of histamine increase in MCS patients (147), we found increased histamine levels globally in about 40% of MCS, EHS or MCS/EHS patients. Since it has been shown that increased histamine levels may in fact appear only when MCS patients are submitted to environmental stressors such as volatile organic compounds (VOC) (147), we thus wonder whether the 60% of patients in our series who were not associated with detectable increased histamine levels may in fact be patients who were not exposed to environmental EMF and/or chemical stressors just before histamine measurement. Such interpretation may also involve the fact that in our series we detected increased S100B protein levels in only 15% of the patients, since the increased levels of protein S100B following brain injury are fleeting (39, 40, 66–68).

However, since EHS and MCS share similar biological abnormalities and so may share a common pathophysiological mechanism (see Section "Analysis of biochemical markers"), these two so far clinically individualized entities may represent two etiopathogenic aspects of a unique common pathological disorder. Arguments in support are the following: 1. EHS and MCS are associated with a similar symptomatic clinical picture; 2. Both entities share identical biological abnormalities including histamine release, oxidative and nitrosative stress, and BBB opening; 3. Both entities are characterized by a similar BBF decrease, and this cerebral hypoperfusion take place in the majority of cases predominantly in the same areas, i.e. mostly in the temporal lobes, more precisely in the capsulothalamic area; 4. either EHS or MCS occur first; 5. Using the same therapeutic protocol, similar positive clinical results can be obtained in both cases (data not shown).

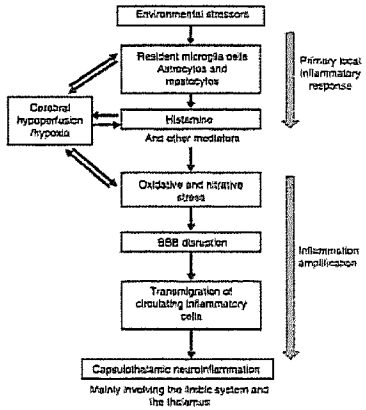


Figure 5: Proposed hypothetic EHS/MCS common pathogenic model based on EHS/MCS induced-neuroinflammation, cerebral hypoperfusion, histamine release, oxidative/nitrosative stress and BBB disruption.

Because EHS and MCS were historically identified clinically and distinguished from each other on the basis of individual potentially environmental stressors, some confusion has emerged. That is, unlike EHS and/or MCS which are still considered as subjective entities because of a lack of etiological substratum, many other internationally recognized diseases were medically characterized before discovery of their etiopathological mechanisms. In fact, the acknowledgment of EHS and MCS as resulting from environmental causes oppose to powerful socioeconomic interests and may explain why they are still not recognized as genuine pathological disorders by national or international bodies and health institutions (137).

Moreover, it is well known that diseases are multifactorial and this may explain why current research failed to attribute a causal origin to EHS and/or MCS. Case-control epidemiologic studies and provocation studies, globally have failed to demonstrate a causal link between EMF and EHS (13, 137), as it may also be the case for chemicals and MCS. These negative results however, do not exclude the possibility of a causal link, as observational studies are difficult to conduct and objective inclusion/exclusion criteria and endpoint evaluation criteria were not clearly defined because of a lack of objective reliable biomarkers. Moreover, if we accept the concept that EHS and/or MCS are part of a common multifactorial disease, clearly those findings may also have been biased by multiple related or unrelated confounding exposure factors and so may have been associated with a reduction of signal-to-noise ratio, thereby obscuring evidence of a possible causal link. Moreover, black box epidemiology and provocation studies focus on risk factors without satisfactory understanding pathogenesis.

There are in fact several arguments for a causal role of EMFs and/or multiple chemicals in the genesis of the so far individualized EHS/MCS pathological disorder: 1. Self-reporting occurrence of clinical symptoms depending on electromagnetic and/or chemical sources, 2. Efficient removing or lessening of clinical symptoms in EHS patients and/or MCS patients in case of avoidance of EMFs and/or chemicals, respectively (19); 3. Appearance of biological abnormalities (positive detection of biomarkers) when patients are exposed to electromagnetic and/or chemical sources, and regression or disappearance of these biological abnormalities (normalization of biomarkers) when patients are withdrawn from electromagnetic and/or chemical sources, a finding that confirm objectively self-reporting patient symptoms (data not shown); 4. a possible common underlying pathophysiological mechanism involving oxidative and/or nitrosative stress-associated neuro-inflammation and BBB opening (see Sections "Demographic panorama" and "Analysis of biochemical markers"); and finally 5. Identical or similar biological abnormalities detected in humans as compared to those evidenced experimentally in animals submitted to EMF and/or chemicals exposure. Although our data account for clinical symptoms and biological abnormalities associated with an intolerance syndrome and highlight its pathogenesis, they do not account for susceptibility and more particularly, hypersensitivity which in addition, to intolerance both characterize EHS and MCS. Virtually all diseases result from the interaction of genes and the environment, hence the concept of genetic susceptibility via constitutive genes which can further the pathogenic role of environmental stressors (148). Theoretically such susceptibility could explain why some subjects are particularly suffering from EHS and/or MCS and not others. A genetic predisposition including gene variants of drug-metabolizing enzymes has been reported for MCS (149–151) but this has not been confirmed (152, 153), suggesting that to define MCS biologically, redox state and cytokine profiling should be considered instead (153).

Our data reveal that women are more susceptible than men to EHS or MCS and this susceptibility concerns both EHS and MCS (see Section "Search for clinical diagnosis criteria"). This suggests some still undetermined sex-related genetic susceptibility. To our knowledge there is no reported study on genetic predisposition in EHS patients. As magnetosomes are detectable in the human brain and meninges (pia mater and dura mater) (154), and because some EMF-related biological effects are achieved through magneto-reception (155), we speculated that some type of innate genetic predisposition to EHS might result from the presence of a high number of magnetosomes in the brain and meninges of susceptible patients. This may reveal to be true particularly for non-thermal EMFs (156). Other hypothesis may include acquired susceptibility through epigenetic mechanisms related to EHS and/or MCS prolonged exposure and some biological synergistic potential between EMF exposure and low dose organic or inorganic chemical contamination (157, 158). This may be particularly the case for heavy metals which, as for EMF, have been shown to release proinflammatory cytokines (159, 160).

It is worthy of note that metallic dental alloys are associated with release of heavy metals such as mercury, lead and cadmium into oral cavity (161, 162) and so may contribute to EHS (158). It has been shown that EMFs such as GSM frequencies emitted from mobile phone may induce or accelerate the mercury vapor release from dental amalgam (163) and consequently may contribute not only to EHS but also to MCS (164).

An intriguing unknown pathophysiological mechanism referred to as sensitivity-related illness (SRI) (4) or as toxicant-induced loss of tolerance (TILT) (165) has been put forward in order to account for the fact that patients with EHS and/or MCS cannot tolerate weak intensity of EMFs and/or low concentration of chemicals. We define acquisition of such a hypersensitivity state with two criteria: 1. Decrease in the tolerance threshold for EMFs or chemicals; and 2. Extension of this decreased tolerance threshold to the whole electromagnetic spectrum or to multiple structurally unrelated chemicals, as disease progress. Although our data may suggest a role of the limbic system and the thalamus, to our knowledge no clear pathophysiological explanation of this intriguing brain-related hypersensitivity condition has yet been given.

EHS/MCS as a possible sentinel pathological disorder

The BBB protects the brain against potentially harmful toxic chemicals which may have contaminated the blood and thereby is currently regarded as a physiological structure that plays a crucial role in maintaining brain homeostasis (166–169). However, the BBB cannot protect the brain against EMFs (170). This may explain why EMFs are probably a major stressor associated with BBB disruption and brain inflammation, and why oxidative stress and more particularly oxidative/nitrosative stress-induced BBB breakdown may be causally involved in neurodegenerative diseases (171, 172), such as Alzheimer's disease (AD) (173–176), Parkinson's disease (PD) (177), multiple sclerosis (MS) (178), Huntington's disease and amyotrophic lateral sclerosis (70) and even possibly psychiatric diseases such as schizophrenia, autism and bipolar disorder (179–182).

Since the first reports on EMF exposure-related BBB disruption (119, 183) conflicting data have emerged (122) leading to search for new tests for evidencing BBB disruption in EHS and/or MCS patients. BBB permeability imaging (184) in addition, to search of peripheral biomarkers could be helpful. Using protein S100B and NTT as biomarkers our data tend to show that BBB opening could be detected in 55%–60% of patients; but this result does not mean the remaining cases could not have been associated with BBB opening we were unable to detect.

There is indeed compelling evidence that chronic neuro-inflammation is a long lasting and potentially self-perpetuating process including an initially long-standing release of inflammatory mediators, leading to increased oxidative and nitrosative stress. This process may thus persist long after the initial environmental trigger and consequently can contribute to neurodegeneration through free radical attack on neural cells (185). This is particularly the case in AD and PD for which toxicity of free radicals have been demonstrated to contribute to protein and DNA injury, inflammation, tissue damage and subsequent neuronal degeneration and apoptosis (175, 176, 183, 185–187).

We have shown that patients with EHS and/or MCS often have cerebral hypoperfusion and histamine release, two factors that in addition, to the production of autoantibodies have been evidenced to occur in AD (173, 174) and PD (188–192); hence contributing to neuro-inflammation and BBB dysfunction. Moreover, several studies have shown that prolonged occupational exposures to low or extremely low frequency EMFs are associated with AD (193–196) and such a link has recently been confirmed in a meta-analysis based on more than twenty epidemiological studies (197). Although it has been shown in a single study that long term high frequency EMF exposure could protect against and even reverse cognitive impairment in mice bearing a so called animal equivalent of AD (198), there is currently no scientific reason to believe that in humans prolonged radiofrequency EMF exposure as it is the case with excessive cell phone and/or mobile phone use will be not also causally related to AD occurrence (199). Moreover, it has been shown that neurodegenerative diseases are in fact multifactorial and that, as it has been hypothesized, ferrimagnetic metals in food chain may contribute to initiate these neurodegenerative diseases under the influence of EMF exposure (200).

Typically AD starts with mild memory deficits, primarily affecting short term memory and gradually progresses to loss of retrospective memory and dementia. An important finding in our still ongoing study is that most of EHS and/or MCS patients had decreased cognitive ability manifested by loss of immediate memory and attention and concentration deficiency (see Sections "Search for reliable disease biomarkers" and "Analysis of biochemical markers"). Since EHS and/or MCS pathogenesis appears to be associated with brain pathophysiological abnormalities similar to that occurring in neurodegenerative disorders, a question is whether EHS and/or MCS are either a pre-neurodegenerative state or an unrelated pathological disorder whose environmental causal origin might however, be similar to that of neurodegenerative diseases. Nevertheless, whichever these two possible etiopathologic alternatives, EHS and/or MCS might be considered as some type of environmental sentinel pathological disorder.

It is worthy of note that in our series, in addition, to the two cases of AD, which were diagnosed a few months after inclusion, another case of AD and two cases of PD were discovered in association with EHS during the patient follow up. Moreover, at inclusion time we excluded two cases of AD, two cases of PD, three cases of multiple sclerosis,

and one case of Huntington disease, which were found to be associated with EHS. In addition, we excluded seven EHS or EHS/MCS cases because they were associated with previous or simultaneous carcinoma: breast carcinoma (3 cases), brain tumor (2 cases) and lymphoma (1 case). We also excluded three MCS cases because they were associated with lymphoma (1 case) and thyroid endocrinopathy (2 cases).

Certainly long term longitudinal analysis and replication of this ongoing prospective study will be necessary to establish whether EHS and/or MCS could be related to neurodegenerative disease and/or cancer, and thus may announce or reflect occurrence of these pathologies.

The growing worldwide health problem

Whatever the causal origin of EHS and/or MCS, there is compelling evidence that EHS and/or MCS self-reporting patients constitute an unsolved, large and growing health problem worldwide.

As far as EHS is concerned, about 1%–10% of the investigated population, e.g. 5% in Switzerland (13), 5% in Ireland, 9% in Sweden, 9% in Germany and 11% in England are presently estimated to be EHS self-reporting persons (201). Given the seven billion persons worldwide using cordless and/or mobile phone it is expected these percentages may increase in the 50 next years. However, because at the time these estimations were made there was no objective criteria for identifying EHS (21), these data require confirmation by more objective investigations.

By using the battery of biomarkers we have investigated in this study it now seems possible to objectively characterize and identify EHS and MCS. Although termed "idiopathic", IEI has been defined as abnormal responses possibly triggered by exposure to organic chemicals and/or metals. It is believed that in addition, to MCS several pathological disorders such as fibromyalgia and chronic fatigue syndrome, because they may share a similar environment-related intolerance condition, could be part of IEI. We have shown multiple lines of evidence that EHS and MCS share a similar pathogenesis and so might be the same pathological disorder whatever their putative causal stressors. This strongly reinforces the concept that both EHS and MCS must be part of the so called IEI syndrome.

Since the WHO publication in 1993 on EMFs (202), much progress have been made in the identification and understanding of EMF effects on the organism, while EHS has still not been clearly characterized and acknowledged by WHO.

Present research vainly focus on the causal role of EMFs and chemicals as possible triggers of EHS and MCS, respectively and not enough on the actually unmet health care needs at a socioeconomic and public health setting for persons with environmental sensitivity (203), as it is particularly the case for EHS and/or MCS persons.

We therefore, strongly propose that whatever their proofs for their causal origins, EHS and MCS should clearly be added to the next version of the WHO International classification of diseases (ICD) on the basis on their clinical and pathological description; as has been the case for many other diseases.

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Review

Microwave frequency electromagnetic fields (EMFs) produce widespread neuropsychiatric effects including depression

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ABSTRACT

Non-thermal microwave/lower frequency electromagnetic fields (EMFs) act via voltage-gated calcium channel (VGCC) activation. Calcium channel blockers block EMF effects and several types of additional evidence confirm this mechanism. Low intensity microwave EMFs have been proposed to produce neuropsychiatric effects, sometimes called microwave syndrome, and the focus of this review is whether these are indeed well documented and consistent with the known mechanism(s) of action of such EMFs. VGCCs occur in very high densities throughout the nervous system and have near universal roles in release of neurotransmitters and neuroendocrine hormones. Soviet and Western literature shows that much of the impact of non-thermal microwave exposures in experimental animals occurs in the brain and peripheral nervous system, such that nervous system histology and function show diverse and substantial changes. These may be generated through roles of VGCC activation, producing excessive neurotransmitter/neuroendocrine release as well as oxidative/nitrosative stress and other responses. Excessive VGCC activity has been shown from genetic polymorphism studies to have roles in producing neuropsychiatric changes in humans. Two U.S. government reports from the 1970s to 1980s provide evidence for many neuropsychiatric effects of non-thermal microwave EMFs, based on occupational exposure studies. 18 more recent epidemiological studies, provide substantial evidence that microwave EMFs from cell/mobile phone base stations, excessive cell/mobile phone usage and from wireless smart meters can each produce similar patterns of neuropsychiatric effects, with several of these studies showing clear dose–response relationships. Lesser evidence from 6 additional studies suggests that short wave, radio station, occupational and digital TV antenna exposures may produce similar neuropsychiatric effects. Among the more commonly reported changes are sleep disturbance/insomnia, headache, depression/depressive symptoms, fatigue/tiredness, dysesthesia, concentration/attention dysfunction, memory changes, dizziness, irritability, loss of appetite/body weight, restlessness/anxiety, nausea, skin burning/tingling/dermographism and EEG changes. In summary, then, the mechanism of action of microwave EMFs, the role of the VGCCs in the brain, the impact of non-thermal EMFs on the brain, extensive epidemiological studies performed over the past 50 years, and five criteria testing for causality, all collectively show that various non-thermal microwave EMF exposures produce diverse neuropsychiatric effects.

Contents

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Chemicals having roles:

Calcium(2+)
Nitric oxide (NO)
Oxido nitrite (peroxynitrite)

1. Introduction

Microwave syndrome (Hocking, 2001; Johnson Liakouris, 1998), a combination of various neuropsychiatric symptoms originally described in persons with occupational exposures to microwave frequency EMFs, has been disputed largely because of the lack of an apparent mechanism for generating these symptoms. It is reported to often include such symptoms as fatigue, headache, insomnia, dysesthesia (impaired sensation), irritability, lack of concentration and other symptoms (Hocking, 2001; Johnson Liakouris, 1998). Similar but more extensive combinations of symptoms have been reported following occupational exposures in two U.S. government reports from the 1970s/1980s (Naval Medical Research Institute Research Report, 1971; Raines, 1981) and following environmental exposures as described in two more recent reviews (Khurana et al., 2010; Levitt and Lai, 2010).

The goal here is not just to review the epidemiology, however, but more importantly to consider the issue of possible physiological mechanism(s). Hennekens and Buring (1989), on p. 40 in their textbook Epidemiology in Medicine state "The belief in the existence of a cause and effect relationship is enhanced if there is a known or postulated biologic mechanism by which the exposure might reasonably alter risk of developing disease." It is of critical importance therefore to assess possible biological mechanism before considering the epidemiological evidence.

Accordingly, this paper considers the mechanism by which low intensity microwave EMFs impact the cells of our bodies, how that mechanism may be predicted to impact the nervous system, evidence for such impact from experimental animal studies, genetic polymorphism evidence for that mechanism acting in humans to produce neuropsychiatric effects and finally, the epidemiological evidence for such effects in human populations with repeated low level microwave EMF exposure. Consideration of each of these types of evidence influences the overall interpretation presented in this paper.

2. Microwave/lower frequency EMFs act to activate voltage-gated calcium channels

In 24 different studies reviewed earlier (Pall, 2013) and two additional studies (Li et al., 2014; Lisi et al., 2006), microwave and lower frequency low intensity EMF effects were blocked or greatly lowered by calcium channel blockers, agents thought to be specific for blocking voltage-gated calcium channels (VGCCs). In these 26 studies, a total of 5 distinct types of channel blockers were used, with each type having a distinct structure and binding to a distinct site, such that it is essentially certain that these must be acting by blocking VGCCs, which is their only known common property. In each of these 26 studies, each of the responses studied, were blocked or greatly lowered by calcium channel blockers, showing that VGCC activation has roles in producing a wide variety of EMF effects. There is a large literature on changes in calcium fluxes and in calcium signaling following microwave EMF exposure (partially reviewed in Walleczek, 1992; Adey, 1993); each of these, including calcium efflux changes, can be explained as being due to VGCC activation, again suggesting a widespread role of VGCC activation in producing biological responses to EMFs. Pilla (2012) showed that pulsed microwave field exposure, produced an almost instantaneous increase in calcium/calmodulin-dependent nitric oxide (NO) signaling, providing strong evidence that these fields can produce an almost instantaneous VGCC activation. It is likely, that these EMFs act directly on the voltage sensor of the VGCCs to produce VGCC activation (Pall, 2015) with the voltage sensor being exquisitely sensitive to these EMFs because of its physical properties and location in the plasma membrane.

EMFs have been proposed to act to produce a wide variety of responses in the cell, via downstream effects of VGCC activation (Pall, 2013, 2014, 2015), including elevated intracellular calcium [Ca2+]i, excessive calcium and nitric oxide signaling and also excessive peroxynitrite, free radicals and oxidative stress.

VGCC activation has been shown to have a universal or near-universal role in the release of neurotransmitters in the brain and also in the release of hormones by neuroendocrine cells (Berridge, 1998; Dunlap et al., 1995; Wheeler et al., 1994), with such release being produced by calcium signaling. There are high densities of diverse VGCCs occurring in neurons throughout the nervous system. Both the high VGCC density and their function in neurotransmitter and neuroendocrine release throughout the nervous system suggests that the nervous system is likely to be highly sensitive to low intensity EMFs.

3. Genetic polymorphism studies

Genetic polymorphism studies are powerful tools for looking at the roles of specific proteins in human populations. In Table 1, a series of genetic polymorphism studies have been performed that show that an allele producing increased expression of the gene encoding the channel of the main L-type VGCC in the brain, produces diverse neuropsychiatric effects. These studies clearly show that excess L-type VGCC activity can cause neuropsychiatric effects. They also predict, therefore, that increased VGCC activity produced by microwave EMFs may be able to also produce widespread neuropsychiatric effects.

4. Histological and functional changes in central nervous system (CNS) and peripheral nervous system (PNS) in animals exposed to microwave EMFs

The most extensive literature on histological and functional changes in animals is from the Soviet literature from the 1950s/1960s with additional Western literature from the same time period. Both Soviet and non-Soviet literature were reviewed in an English language Publication by Tolgskaya and Gordon (1973). This publication is, therefore, the main focus of this section. That publication was divided into thermal and non-thermal exposure studies, with the non-thermal studies which occupy the majority of the text (pp. 53–137) being of sole interest here.

Table 1
Influence of genetic polymorphism of the CACNA1C in producing diverse neuropsychiatric effects.

Citation	Genetic polymorphism	Changes produced by allele of gene
Bhat et al. (2012)	Polymorphism producing increased expression of CACNA1C L-type VGCC subunit	Review: The polymorphism is associated with increased susceptibility to bipolar disorder, "depression, schizophrenia, autism spectrum disorders, as well as changes in brain function and structure in control subjects who have no diagnosable psychiatric illness."
Bigos et al. (2010)	Polymorphism producing increased expression of CACNA1C L-type VGCC subunit	Associated with increases in both bipolar disorder and schizophrenia
Krug et al. (2010)	Polymorphism producing increased expression of CACNA1C L-type VGCC subunit	Negatively influences language production on a semantic level
Krug et al. (2014)	Polymorphism producing increased expression of CACNA1C L-type VGCC subunit	Influences episodic memory and retrieval
Soeiro-de-Souza et al. (2012)	Polymorphism producing increased expression of CACNA1C L-type VGCC subunit	Produces impaired facial emotion recognition
Tesli et al. (2013)	Polymorphism producing increased expression of CACNA1C L-type VGCC subunit	Produces increased activation of the amygdala during emotional processing
Thimm et al. (2011)	Polymorphism producing increased expression of CACNA1C L-type VGCC subunit	Associated with attention deficits including alerting, orienting and executive control of attention

These were all derived from the Tolgskaya and Gordon (1973) review and page numbers listed are page numbers from that document. All refer to changes produced by non-thermal exposures in the nervous system of experimental animals, with most being in rats.

This discussion scrolls down through Table 2.

The majority of the histological changes seen in these mostly rodent studies, are seen in the nervous system, despite its being less than 2% of the rodent cell mass. There are statements made that the nervous system, both central and peripheral, is the most highly sensitive tissue to these non-thermal microwave and lower frequency EMFs. Following the nervous system in sensitivity are the myocardium and the testis; myocardial cells are known to have very high densities of VGCCs with especially high densities in the pacemaker cells and the testis is known to have high densities specifically of the T-type VGCCs. Pulsed EMFs are more active in producing histological changes in the brain than are non-pulsed fields, in two studies reviewed; there is a much larger literature showing that in most cases pulsed fields are more biologically active (Pall, 2015; Pangopoulos et al., 2013; Belyaev, 2015).

A wide variety of brain and peripheral nervous system tissues show histological changes following non-thermal exposures. Among the important tissues impacted are the hypothalamus and pituitary gland, where both show similar patterns of changes in neuroendocrine activities. There is an initial increase in neuroendocrine activity (this may be produced directly by VGCC stimulation of secretion), followed over time by "exhaustion" of neuroendocrine activity (this may be produced by tissue damage produced from long term intracellular calcium [Ca2+]i elevation).

There are widespread histological changes produced in neuronal and neuroendocrine tissues. These were repeatedly reported to be largely reversible on cessation of EMF exposure. They become, however, irreversible when exposure is extended in time. There are changes in EEG activity, which may be an easily measurable monitor of neurological damage.

In a summary statement, Tolgskaya and Gordon (1973) state, "This does not confirm the view, so widely held in the past among Soviet investigators and still maintained to a large extent even at the present time in the West, that the action of microwaves is entirely thermal."

While there were many studies of brain impact of non-thermal EMFs performed in the 1950s/60s that make the information content of Tolgskaya and Gordon (1973) quite high, there is also a substantial recent literature on brain effects of non-thermal microwave EMF exposures (see, for example: Ammari et al., 2008a,b; Bas et al., 2009; Brillaud et al., 2007; Carballo-Quintás et al., 2011; Eberhardt et al., 2008; Dasdag et al., 2009, 2012; Grafström et al., 2008; Kumlin et al., 2007; López-Martín et al., 2006; Mausset-Bonnefont et al., 2004; Odaci et al., 2008; Ragbetli et al., 2010; Salford et al., 2003; Sonmez et al., 2010).

5. Older epidemiological reviews and other related studies

Two U.S. Government reports each listed many apparent neuropsychiatric effects of microwave/radiofrequency EMFs and a third recognized the role of non-thermal effects on our bodies, but had only a little consideration of neuropsychiatric effects.

The earliest to these was a Naval Medical Research Institute (NMRI) Research Report (1971) which listed 40 apparent neuropsychiatric changes produced by non-thermal exposures including: 5 central/peripheral nervous system (NS) changes, 9 CNS effects, 4 autonomic system effects, 17 psychological disorders, 4 behavioral changes and 2 misc. effects. This NMRI report also provided a supplementary document listing over 2300 citations documenting these and other effects of microwave exposures in humans and in animals.

The Raines (1981) NASA report reviewed extensive literature based on occupational exposures to non-thermal microwave EMFs, with that literature coming from U.S., Western European and Eastern European studies. There are no obvious differences in the literature coming from these different regions. Based on multiple studies, Raines (1981) reports 19 neuropsychiatric effects to be associated with occupational microwave/radiofrequency EMFs.

The Bolen (1994) report put out by the Rome Laboratory of the U.S. Air Force, acknowledged the role of non-thermal effects of microwave EMFs on humans. This report states in the Conclusion section that "Experimental evidence has shown that exposure to low intensity radiation can have a profound effect on biological processes. The nonthermal effects of RF/MW radiation exposure are becoming important measures of biological interaction of EM fields." Clearly Bolen (1994) rejects the claim that only thermal effects occur. Bolen (1994) discusses a specific non-thermal neuropsychiatric effect, where anesthetized animals are awakened when the head is irradiated with microwave EMFs. This suggests a similar mechanism to that acting in humans where such EMFs produce insomnia (see below).

6. Specific epidemiological studies on neuropsychiatric effects of microwave EMFs

There are 26 different epidemiological studies described in Table 3. Although 4 of these only studied a single neuropsychiatric effect, 22 of these each provide substantial evidence for the pattern described in the earlier U.S. reports, that a wide range of

Table 2
Histological and functional changes in brain function in animals following exposure to non-thermal microwave EMFs.

Observations including page numbers	Comment from Author
The majority of the histological changes seen following non-thermal exposures, occurred in the nervous system, despite its being only about 2% of the tissue mass in rodents; this suggests that the nervous system is highly sensitive to such exposures. Elsewhere (pp. 129, 136), it is suggested that the nervous system is the most sensitive tissue, followed by the heart and the testis, among all of the tissues of the body. The most severe histological changes produced by these non-thermal EMF exposures occur in the nervous system (pp. 136).	High CNS sensitivity to EMFs is predicted by the high density of VGCCs that occur in neurons throughout the nervous system, plus the VGCC role in neurotransmitter and neuroendocrine release.
Pulsed fields were more active than non-pulsed fields in producing histological changes (pp. 71, 97).	Pulsed fields have often been found to be more biologically active than are non-pulsed fields in many different studies from many countries (Pall, 2015; Pangopoulos et al., 2013; Belyaev, 2015).
Nervous system regions impacted by non-thermal microwave and lower frequency fields include: cortex, diencephalon including the hypothalamus and thalamus, hippocampus, autonomic ganglia, sensory fibers, pituitary gland including neurohypophysis.	
Neuroendocrine changes seem to undergo change over increased time of exposure. Neurosecretion in the hypothalamus and in the pituitary each go through a complex sequence over time, where EMF exposure initially produces increased hormone secretion but where over time, the neurosecretory cells become "exhausted", leading to lowered secretion and in some cases cell death (pp. 77–96).	Elevated [Ca2+]i stimulates hormone secretion. However when such elevated [Ca2+]i occurs over extended time periods it is highly damaging to the cell, leading in some cases to apoptosis; thus this time course of action should not be surprising.
Histological changes include boutons/argyrophilia, smaller neurons, vacuole formation in neuroendocrine cells, bead-like thickening along dendrites (pp. 66, 70, 71, 73, 97, 98, 100, 111, 115–117, 121–125). Spines near the ends of dendrites become deformed and with still more sessions of irradiation, disappeared entirely (p. 70). Sensory neurons, following exposures, developed changes characteristic of irritation, with "marked tortuosity of the nerve fibers." Many histological changes are seen in the hypothalamic cells (pp. 87–92) as their neuroendocrine function becomes impacted. Histological changes were found even with exposures that produced no apparent functional changes.	
Many histological and functional changes are reported to initially be reversible, following cessation of exposure, but progressively become irreversible with longer exposure. (pp. 64, 72, 74). Paralleling the development of irreversibility, it is found that "Repeated exposure leads to gradual increase in severity of observed changes." ... including "increasingly severe disturbance of conditioned reflex activity in the animals, changes in responses of animals particularly sensitive to acoustic stimulation..." (p. 104).	If this is also true in humans, then claims that there cannot be non-thermal effects, claims which act to prolong exposures, may be causing irreversible damage to many humans.
EEG changes (pp. 55, 60, 102), including seizure activity following sensory provocation.	Lai (1997) has an extensive review of EEG changes in animals following non-thermal microwave EMF exposures
Neurodegeneration is reported in a number of places in this review (pp. 72, 83, 117).	
Synaptic connections in regions of the brain are disrupted (pp. 65–74, 97, 113, 121, 136), and at the extreme, some neurons are completely asynaptic (p. 73).	Synaptic connections are known to be disrupted in autism; could this suggest that autism may be generated by EMF exposure? No doubt, we need much more evidence on this.
"after prolonged and repeated irradiation with low-intensity centimeter waves, with no elevation of the body temperature and when the animal's condition remained satisfactory, changes were nevertheless found in the sensory fibers of the skin and viscera in the form of irritation phenomena. These findings concur with the view in the literature that the receptor system as a whole and, in particular its preterminal portions are highly sensitive." p. 76. This description is similar to what is reported to occur in electromagnetic hypersensitivity (EHS). Other such studies are described and include cumulative changes over time, that may also explain changes reported in EHS (pp. 75, 99, 100, 104).	One wonders whether almost 60 years ago, the Soviet literature may have already described a possible animal model for EHS. None is known to exist today, and because of that, EHS studies are severely constrained. Clearly one needs to be skeptical about this interpretation, but it is of great importance that this be further studied.

neuropsychiatric effects are produced by exposure to various non-thermal microwave frequency EMFs. Perhaps the most important of these 26 is the Santini et al. (2003) study of people living near cell phone base stations.

There are three recent studies on the generation of headache during or shortly following long mobile phone calls (listed under Chu et al., 2011 in Table 3). The timing of development of these headaches and the finding that they occur on the ipsilateral side of the head, the side receiving much higher EMF exposure during the call, both argue strongly that these headaches are caused by the long mobile phone calls. Such causality was concluded earlier by Frey (1998) based on earlier studies and is now still more strongly documented.

7. Criteria for assessing causality in epidemiological studies

It is important to consider the different criteria that allow one to judge whether a cause and effect relationship is justified by the studies listed in Table 3 and the individual studies cited in Raines (1981). There are five such criteria that should be considered in making that judgment (see pp. 39–43 in Hennekens and Buring, 1989):

Strength of Association: Is there a strong correlation between exposure and the neuropsychiatric symptoms? There clearly is for several studies cited in Raines (1981). One example is the Dwyer and Leeper (1978) study (see Table 3) where there is a large increase in symptoms and where that increase is greater with longer occupational exposure. Another example is the Lerner (1980) study of 1300 microwave workers, where workers with relatively low exposure levels had an approximate doubling of neurological complaints and where those with substantially higher exposure levels had an approximate tripling of neurological complaints over controls. Sadcikova (1974) found that 7 of 8 neuropsychiatric symptoms studied, showed a statistically significant rise in prevalence with longer occupational exposure (see Table 3). Sadcikova (1974), also found that microwave workers had increases of 3 to over 10-fold in: feeling of heaviness in the head; tiredness; irritability; sleepiness; partial loss of memory; and skin sensitivity. There is also a strong association where important new exposures occur – this is clearly the case with all of the studies of people living near cell/mobile phone base

Table 3
Neuropsychiatric symptoms apparently produced by exposure to various electromagnetic fields.

Citation	EMF exposure	Apparent neuropsychiatric symptoms
Abdel-Rassoul et al. (2007)	Living near mobile phone base station	Significant increases in neuropsychiatric complaints included: headache, memory changes, dizziness, tremors, depressive symptoms, sleep disturbance; attributed to effects of EMFs on the human nervous system.
Al-Khlaiwi and Meo (2004)	Mobile phone use	Higher prevalence of fatigue, headache, dizziness, tension and sleep disturbance; the authors conclude that mobile phone use is a risk factor for developing these symptoms.
Altpeter et al. (2000)	Short-wave broadcasting tower, ranging from 6.1 to 21.8 MHz	Sleep disruption shown to occur, correlated with exposures and apparent increase over time; short term suppression of melatonin shown, based on melatonin increases during a 3 day period when the tower was turned off.
Bortkiewicz et al. (2004)	Living near cell phone base station EMFs	Sleep disturbance, irritability, depression, blurred vision, concentration difficulties, nausea, lack of appetite, headache, vertigo.
Bortkiewicz et al. (2012)	Living near mobile phone base stations	Dose response relationships for sleep disturbance, irritability, depression, blurred vision, concentration difficulties, nausea, lack of appetite.
Chu et al. (2011), also Chia et al. (2000), Oftedal et al. (2000)	Mobile phone use	Headache during prolonged mobile phone use or within an hour following such use, with pain occurring on the ipsilateral side of the head; similar observations obtained in each of the 3 studies in column 1; see also Frey (1998).
Conrad (2013)	Smart meter EMF exposure	14 common new symptoms (both severe and moderate) among those exposed and symptomatic, 13 apparent neuropsychiatric: Insomnia, tinnitus, pressure in the head, concentration difficulty, headaches, memory problems, agitation, dizziness, fatigue, skin tingling/burning, involuntary muscle contractions, eye/vision problems, numbness; These ranged in prevalence from 63% to 19% of those experiencing symptoms, such that most symptomatic people experienced multiple symptoms.
Dasdag et al. (1992)	People working in MW broadcasting or at a television transmitter station	These groups suffered from headache, fatigue, irritability, stress, sleepiness, loss of appetite, loss of hearing.
Dwyer and Leeper (1978)	People working in radiofrequency EMFs	Headache, eyestrain, dizziness, disturbed sleep, daytime sleepiness, moodiness, mental depression, memory impairment, muscle and/or cardiac pain, breathing difficulties, increased perspiration, difficulty with sex life.
Eger and Jahn (2010)	Living near mobile phone base station	Neuropsychiatric symptoms, with most showing dose–response relationships: depression; headache; cerebral symptoms; dizziness; disorders of optical and acoustic sensory systems; sleep disturbance; skin changes; with the exception of dizziness, all of these had $p < 0.001$.
Johnson Liakouris (1998)	Study of personnel in U.S. embassy in Moscow exposed to microwave EMFs	Statistically significant increases in neurological (peripheral nerves and ganglia), dermographism (skin responses), irritability, depression, loss of appetite, concentration difficulties, peripheral ganglia and nerve dysfunction.
Khan (2008)	Excessive mobile phone use	Complaints of headache, fatigue, impaired concentration, memory disturbance, sleeplessness, hearing problems.
Kolodynski and Kolodinska (1996)	Children living near a Radio Location Station, Latvia	Memory dysfunction, attention dysfunction, lowered motor function, slowed reaction time, lowered neuromuscular endurance.
Lamech (2014)	Exposure to wireless smart meter radiation in Victoria, Australia	The most frequent symptoms to develop after smart meter radiation exposure were insomnia, headache, tinnitus, fatigue, cognitive disturbances, dysesthesias (abnormal sensation), dizziness.
Navarro et al. (2003)	Living near cell phone base station	Statistically significant dose response relationships for fatigue, irritability, headache, nausea, loss of appetite, sleep disorder, depressive tendency, feeling of discomfort, difficulty of concentration, loss of memory, visual disorder & dizziness.
Oberfeld et al. (2004)	Living near cell phone base station	Statistically significant dose–response relationships for headache, fatigue, irritability, loss of appetite, visual disorder, nausea, sleeping disorders, dizziness, poor concentration, memory loss.
Otto et al. (1994)	Occupational exposure of 25 workers to either UHF television broadcasting (10) or to 1062 kHz medium wave broadcasting (15)	10 neuropsychiatric changes were assessed, all showing statistically significant changes compared with controls: Somatization*, obsessive compulsivity*, interpersonal sensitivity, depression, anxiety*, hostility*, phobic anxiety*, paranoid ideation, psychoticism*, sleeping disturbance. * $p < 0.001$.
Sadcikova (1974)	Occupational exposure to microwave radiation, including at <.07 mW/cm^2	Heaviness in head*, fatigue*, irritability*, sleepiness, memory loss*, cardiac pain*, dermographism (skin sensitivity)*, hyperhidrosis* * significant increase with time of exposure.
Salama and Abou El Naga (2004)	High cell (mobile) phone use	Most common effects were headache, ear ache, sense of fatigue, sleep disturbance, concentration difficulty, face burning sensation. The first three of these had very high statistical significance for correlation with extent of cell phone use.
Santini et al. (2003)	Living near cell phone base stations	Each of the following neuropsychiatric symptoms showed statistical significant dose–response relationships: nausea, loss of appetite, visual disturbance, irritability, depressive tendencies, lowered libido, headache, sleep disturbance, feeling of discomfort, fatigue.
Schüz et al. (2009)	Mobile phone use	Found a small, statistically significant increase in migraine and vertigo. Also found an apparent lowered occurrence of Alzheimer's, other dementia, Parkinson's and epilepsy – these latter were interpreted as being due to perhaps early symptoms of the developing diseases lowering probability of acquiring a mobile phone.
Söderqvist et al. (2008)	Use of mobile phone among adolescents	Increased mobile phone use was associated with increases in tiredness, stress, headache, anxiety, concentration difficulties and sleep disturbances.
Thomée et al. (2011)	High mobile phone use	High mobile phone use was associated with statistically significant rises in stress and sleep disturbance, with somewhat weaker association with depression.
Waldmann-Selsam et al. (2009)	Digital TV signaling	Constant headaches, pressure in head, drowsiness, sleep problems, tightness in chest, shortness of breadth, depressive mood, total apathy, loss of empathy, burning skin, inner burning, leg weakness, pain in limbs, stabbing pain in various organs, weight increase.

stations, listed in Table 3 and also with the two studies of people who become exposed to radiation from smart meters. The studies listed in Table 3 under Chu et al. (2011) (see also Chia et al., 2000; Oftedal et al., 2000) are of a special type. Here people making very long (over 1 h) cell/mobile phone calls develop headaches an hour or more following the initiation of the long call. So these occur within a specific time range following initiation of these long calls, such that headache would only occur very infrequently in that time frame by chance. So here again, there is a strong association. While there is no question that many of these studies show high strength of association, it is also clear that it is becoming progressively more difficult to do these studies. As exposures become almost universal in countries around the world, it is getting difficult if not impossible to find good negative controls. There may be a similar problem in doing animal studies, such that it may be necessary to raise animals in Faraday cages in order to avoid exposures that would otherwise occur as a consequence of our near ubiquitous EMFs.

Biological credibility is extremely strong here, with three aspects of the biology predicting that these low intensity fields cause widespread neuropsychiatric effects. This was discussed above and is reconsidered in the following section.

Consistency within the different epidemiological studies and with other types of studies. The epidemiological studies listed in Table 3 and also those showing neuropsychiatric effects that were cited in Raines (1981) have been performed in many different countries with different cultures. They have been performed in multiple countries in Western Europe, Eastern Europe, the Middle East and in East Asia, as well as in the U.S. and Australia. They are, therefore, not limited to one or two cultural contexts. This is deemed, therefore, an important indicator of causality. We also have a surprising consistency of apparent neuropsychiatric effects of different fields, including various occupational exposures and exposures to cell/mobile phone base stations, exposure to the phones themselves, exposure to smart meter pulses, and other EMFs (see Table 3). Pulsation patterns, frequencies and exact intensities may produce various biological responses (Pall, 2015; Pangopoulos et al., 2013; Belyaev, 2015) so it is a bit surprising that we have as much consistency as we do have across different types of exposures. We also have consistency with the biology discussed in the previous section. Because elevated VGCC activity produced by genetic polymorphism (Table 1) produces diverse neuropsychiatric effects, it is not surprising that elevation of VGCC activity produced by microwave EMF exposure apparently also produces diverse neuropsychiatric effects. Similarly because non-thermal EMF exposures produce widespread changes in brain structure and function in animals (Tolgskaya and Gordon, 1973), it is not surprising that the neuropsychiatric symptoms, which are produced as a consequence of brain dysfunction are produced by such EMFs.

Time sequence: It is clear that the all of these effects follow exposure in the various studies that have been published. In some studies, it is also clear that longer occupational exposure times produce increased symptom prevalence. These include Dwyer and Leeper (1978) and Baranski and Edelwejn (1975). These observations all support a causal relationship between exposure to EMF and the development of neuropsychiatric symptoms.

Dose–response relationship: It is assumed, here, that biological effects have a positive correlation with the intensity of the apparent causal stressor. This is not necessarily true of EMF effects, because it has been shown that there are "window effects" where specific intensities have larger biological effects, than *do either lower or higher intensities* (Pall, 2015; Pangopoulos et al., 2013; Belyaev, 2015). Nevertheless, where different intensities were studied in these epidemiological studies, they do show the dose–response relationship assumed here including Altpeter et al. (2000), Dwyer and Leeper (1978), Eger and Jahn (2010), Lerner (1980), Navarro et al. (2003), Oberfeld et al. (2004), Salama and Abou El Naga (2004), Santini et al. (2003) and Thomée et al. (2011). Thus these data do fit well to the assumed dose–response relationship, found in most causal roles. The Altpeter et al. (2000) study showed a special type of evidence for causality: during a 3-day period when the broadcasting tower was turned off, the melatonin levels recovered to near-normal levels. The studies of headache occurrence on prolonged cell/mobile phone calls (typically well over one hour) listed under Chu et al. (2011) in Table 3 also suggest the assumed dose–response relationship (see also Chia et al., 2000; Oftedal et al., 2000 and earlier citations listed in Frey, 1998). Because such headaches only occur with prolonged cell/mobile phone calls, these studies also provide evidence for a dose–response relationship because low doses are ineffective. Furthermore these same studies provide evidence for such a dose–response relationship from another type of observation. Because the headaches occur predominantly on the ipsilateral side of the head which receives much higher EMF exposure intensity, rather than on the contralateral side of the head, which receives much lower intensities, this provides an additional type of evidence for the predicted dose–response relationship.

While the evidence is convincing that the various neuropsychiatric apparent consequences of microwave EMF exposure are in fact caused by such exposures, there may be somewhat more controversy about another EMF-neuropsychiatric linkage. Havas et al. (2010) have reported a similar list of neuropsychiatric symptoms in electromagnetic hypersensitivity (EHS) patients. They found that each of the following symptoms were common in EHS: poor short term memory; difficulty of concentration; eye problems; sleep disorder; feeling unwell; headache; dizziness; tinnitus; chronic fatigue; tremors; body pain; difficulty speaking; tingling sensation in feet or hands; difficulty writing; difficulty walking; migraine. The similarity of these symptoms to the most commonly found symptoms following non-thermal microwave EMF exposures (Table 3), suggests that EHS is a genuine sensitivity to EMFs. In the bottom row in Table 2, sensitivities were found in rodent studies following non-thermal exposure that suggest a possible animal model for the study of EHS. Each of these EHS-related issues needs to be followed up experimentally.

8. Discussion and conclusions

In the previous section, each of the five criteria for assessing whether an epidemiological association is causal, were considered. Those five are (Hennekens and Buring, 1989): (1) strength of association; (2) biological credibility; (3) consistency; (4) time sequence; (5) dose–response relationship. Each of these five provide strong support for causality such that the combination of all five provides compelling evidence for causality. Low-intensity microwave frequency EMFs do cause diverse neuropsychiatric symptoms. While each of these five is important here, the one that is most important is the criterion of biological credibility.

Three related sets of biological observations each predict that low-intensity microwave EMFs produce widespread neuropsychiatric effects:

1. Such EMFs act via activation of VGCCs, acting through the VGCC voltage sensor which is predicted to be exquisitely sensitive to these EMFs (Pall, 2015). VGCCs occur in high densities throughout the nervous system and have essential roles throughout the nervous system in releasing neurotransmitters and neuroendocrine hormones. These properties predict, therefore, that these low intensity non-thermal microwave EMFs cause widespread changes in the nervous system, causing, in turn, diverse neuropsychiatric effects.

2. Elevated VGCC activity, produced by an allele of the CACNA1C gene which encodes the channel of the main L-type VGCC in the brain, produces various neuropsychiatric effects (Table 1). This predicts, that low intensity non-thermal microwave frequency EMFs which also produce elevated L-type and other VGCC activity, therefore produce widespread neuropsychiatric effects.
3. Studies reviewed in the Tolgskaya and Gordon, 1973 publication (Table 2) have shown that the cells of the mammalian nervous system show high sensitivity to various non-thermal microwave and lower frequency EMFs, being apparently more sensitive than any other organ in the body of rodents. These studies predict that the human nervous system is likely to be similarly sensitive to these EMFs, predicting, therefore, widespread neuropsychiatric effects in humans.

We not only have biological credibility but also more importantly, each of these distinct but interrelated biological considerations predicts that low-intensity, non-thermal microwave EMFs produce widespread neuropsychiatric effects. That common prediction is verified by extensive data summarized in citations provided by the Naval Medical Research Institute Research Report (June 1971), data provided by The Raines (1981) NASA report, and by 26 epidemiological studies summarized in Table 3.

The most commonly reported neuropsychiatric symptoms from these studies are summarized in Table 4.

A total of 22 different studies described in Table 3 were used for data for this table, but not 4 others that only assessed a single neuropsychiatric end point. The Altpeter study which only assessed sleep disturbance/melatonin depletion and the three studies listed under Chu et al. which only assessed headache occurrence following long cell phone calls, listed in Table 3 were not included. Because many of the studies only assessed from 3 to 7 specific symptoms, it is not surprising that the numbers of studies reporting a specific symptom fall far below 22. Where several symptom descriptions were included under one heading, such as dysesthesia, if a study had more than one of these symptom descriptions, it was only counted once.

All the symptoms listed in Table 4 should be considered established parts of microwave syndrome (Hocking, 2001; Johnson Liakouris, 1998). Even if the statistical significance in each study was of the lowest statistical significance ($p < .05$) one would expect only 1 positive study to occur at random out of the 22 studies included here. Because many individual symptoms were not surveyed in many individual studies, the expectation is substantially lower than that. Each of these, having shown positive results in 5 or more studies are highly unlikely, therefore, to have occurred by chance. Stong statistical significance is also seen for individual neuropsychiatric effects reported to have $p < 0.001$ in the Eger and Jahn (2010) and Oto et al. (1994) studies (see Table 3).

Table 4
Commonly reported neuropsychiatric symptoms following microwave EMF exposure.

Symptom(s)	Numbers of studies reporting
Sleep disturbance/insomnia	17
Headache	14
Fatigue/tiredness	11
Depression/depressive symptoms	10
Dysesthesia (vision/hearing/olfactory dysfunction)	10
Concentration/attention/cognitive dysfunction	10
Dizziness/vertigo	9
Memory changes	8
Restlessness/tension/anxiety/stress/agitation/feeling of discomfort	8
Irritability	7
Loss of appetite/body weight	6
Skin tingling/burning/inflammation/dermographism	6
Nausea	5

EEG changes may well be part of microwave syndrome, as well. While none of the studies described in Table 3 measured EEGs, six studies of human occupational exposure cited in the Raines (1981) showed EEG changes (Baranski and Edelwejn, 1975; Bise, 1978; Dumanskij and Shandala, 1974; Lerner, 1980; Sheppard and Eisenbud, 1977). Murbach et al. (2014) cited 10 human studies in support of their statement that "the most consistently reported effects (of mobile phone use) in various studies conducted by different laboratories are changes in the electroencephalogram (EEG) power spectrum." Three recent studies (Lustenberger et al., 2013; Schmid et al., 2012a,b) and several earlier studies cited in Wagner et al. (1998) have each shown EEG changes in sleeping humans exposed to non-thermal pulsed microwave fields. Two recent studies showed EEG changes in persons exposed to Wi-Fi fields (Maganioti et al., 2010; Papageorgiou et al., 2011). Lai (1997) described 8 animal studies showing changes in EEG patterns in animals exposed to non-thermal EMFs and three additional animal studies were described in Tolgskaya and Gordon (1973). With the exception of the 6 studies cited in the second sentence in this paragraph, all of these are direct experimental studies which are not, therefore, susceptible to the questions of causality that can be raised about epidemiological studies. It is the author's view that future studies should consider studying EEG changes as an objectively measurable assessment of brain physiology and that before and after increased exposure studies should be considered when a new EMF source is to be introduced into human populations. While such studies must be done carefully, given the complexity of EEGs, even very small numbers of individuals may produce highly statistically significant results in well designed studies analyzed with paired *t*-tests.

One of the citations from the previous paragraph, Bise (1978) reviewed earlier studies of low level microwave frequency exposures in humans and concluded that such EMFs produced the following neuropsychiatric effects: headache, fatigue, irritability, dizziness, loss of appetite, sleepiness, sweating, difficulty of concentration, memory loss, depression, emotional instability, dermographism, tremor, hallucinations and insomnia. The strong similarity of this list from 37 years ago and the list in Table 4 should be noted. The Bise (1978) list is based on occupational exposure studies whereas the current list in Table 4 is based primarily on EMF exposures from cell/mobile phone base stations, from heavy cell phone usage and from smart meters, *three types of exposures that did not exist in 1978*. The strong similarity between the Bise (1978) list and the current one 37 years later alone produces a compelling argument that the 11 neuropsychiatric effects found on both lists are caused by exposure to multiple types of low-intensity microwave EMFs.

The pattern of evidence is compelling in support of the earlier statement of Levitt and Lai (2010) that "the primary questions now involve specific exposure parameters, not the reality of complaints or attempts to attribute such complaints to psychosomatic causes, malingering or beliefs in paranormal phenomena."

We can barely imagine how the combinations of neuropsychiatric effects, including those in Table 4, will influence human behavior and social interactions, now that the majority of the human populations on earth are exposed to ever increasing intensities and diversity of microwave frequency EMFs. You may recall that three of the occupational exposure studies cited in (Raines, 1981 showed increasing prevalence of neuropsychiatric symptoms with years of exposure to consistent patterns of EMF exposure intensities (Dwyer and Leeper, 1978; Sadcikova, 1974;

Baranski and Edelwejn, 1975). With ever increasing exposures in human populations, we have no idea what the consequences of these ever increasing exposures will be.

Conflict of interest

The author declares no conflict of interest.

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EXHIBIT F

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American Academy of Environmental Medicine

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.[9-11] 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.[4,7,15-17] 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.[4,20] 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.[24] 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.[23] Pulsed electromagnetic frequencies were shown to consistently provoke neurological symptoms in a blinded subject while exposure to continuous frequencies did not.[25]

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.[26,28,29] 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.[26,27,30] 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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Review

Effects of electromagnetic fields exposure on the antioxidant defense system

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ABSTRACT

Technological devices have become essential components of daily life. However, their deleterious effects on the body, particularly on the nervous system, are well known. Electromagnetic fields (EMF) have various chemical effects, including causing deterioration in large molecules in cells and imbalance in ionic equilibrium. Despite being essential for life, oxygen molecules can lead to the generation of hazardous by-products, known as reactive oxygen species (ROS), during biological reactions. These reactive oxygen species can damage cellular components such as proteins, lipids and DNA. Antioxidant defense systems exist in order to keep free radical formation under control and to prevent their harmful effects on the biological system. Free radical formation can take place in various ways, including ultraviolet light, drugs, lipid oxidation, immunological reactions, radiation, stress, smoking, alcohol and biochemical redox reactions. Oxidative stress occurs if the antioxidant defense system is unable to prevent the harmful effects of free radicals. Several studies have reported that exposure to EMF results in oxidative stress in many tissues of the body. Exposure to EMF is known to increase free radical concentrations and traceability and can affect the radical couple recombination. The purpose of this review was to highlight the impact of oxidative stress on antioxidant systems.

1. Introduction

Electromagnetic fields (EMF) are emitted by many natural and man-made sources that play important roles in daily life. More than 3 billion people across the world are exposed to EMF every day [1]. Lifetime exposure to EMF is becoming the subject of significant scientific investigation since it has the potential to cause crucial changes and deleterious effects in biological systems. The biological impacts of EMF can be classified as thermal and non-thermal. Thermal effects are associated with the heat created by EMFs in a certain area. This mechanism occurs via an alteration in temperature deriving from radiofrequency (RF) fields. It is possible that every interaction between RF fields and living tissues causes an energy transfer resulting in a rise in temperature. The skin and other superficial tissues usually absorb the non-thermal radiations emitted by mobile phones; this causes the insignificant increase of temperature of the brain or other organs in the body [2]. Nonthermal mechanisms are those that are not directly associated with this temperature change but rather to some other changes in the tissues in association with the amount of energy absorbed [3,4]. Studies on the health effects of RF energy from communication systems have revealed that non-thermal effects should also be discussed. The fact that the possible biophysical mechanisms of RF-EMF interaction with living cells have not yet been fully elucidated is one of the reasons for these discussions [4]. A significant part of many studies concerning EMF have investigated the "non-thermal" effects of RF on biological tissues [5,6]. It has been observed that this effect is mediated by generation of reactive oxygen species (ROS) [7]. ROS are involved in various cellular functions. They can be essential or extremely toxic to cellular homeostasis [8]. Their cytotoxic effects derive from peroxidation of membrane phospholipids. This creates a change in the conductivity of the membrane and loss of membrane integrity [9]. Exposure to EMF has been observed to cause increased free radical production in the cellular environment. Living organisms have anti-oxidative mechanisms, such as glutathione (GSH), glutathione peroxidase (GPx), catalase (CAT), and superoxide dismutase (SOD), in order to alleviate the damage caused by ROS and their products [10]. This defense mechanism acts by sup-

Abbreviations: EMF, electromagnetic fields; RF, radiofrequency; ROS, reactive oxygen species; GSH, glutathione; GPx, glutathione peroxidase; GR, glutathione reductase; GST, glutathione S-transferase; CAT, catalase; SOD, superoxide dismutase; HSP, heat shock protein; EMF/RFR, electromagnetic frequency and radiofrequency exposures; ELF-EMFs, exposure to extremely low frequency; MEL, melatonin; FA, folic acid; MDA, malondialdehyde.

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pressing or impairing the chain reaction triggered by ROS. In this case, antioxidant defense mechanisms are impaired by being subjected to an agent that causes overproduction of ROS, including EMF, thus resulting in oxidative stress [11,12]. Studies in recent years have reported that free radicals play a major role in the mechanism behind many diseases, such as diabetes and cancer [13–15]. However, there is still much uncertainty on the subject, and several questions remain to be answered.

This review evaluated the effect of exposure to EMF on biological tissues by concentrating on alterations in several antioxidant enzyme activities and different parameters of oxidation.

2. Electromagnetic field effects

A wide spectrum of electromagnetic waves are today emitted by radar, communication equipment, mobile phone base stations, high voltage lines, radio and television transmitters, substations, and electrical equipment at home and work, in addition to many electrical systems in the environment [16]. The Global System for Mobile Communications (GSM, 850–900 MHz and 1850–1990 MHz) is currently the most extensive system for mobile telecommunications worldwide [17,18]. The mobile phone models (1800 MHz –2200 MHz), laptops (1000 MHz–3600 MHz) and wireless networks in use today function with high frequency (2.45 GHz) microwave radiation [19]. In parallel to technological developments in this century, technological devices are becoming ever more important in daily life. However, despite making life easier, they may also cause a number of health problems. In particular, the average age of beginning mobile phone use has decreased rapidly to elementary school age, and durations of exposure to EMF are also increasing. One study reported that extremely low exposure to EMF from mobile phones may cause health problems [20]. Several studies have reported findings such as stress, headache, tiredness, anxiety, decreased learning potential, impairment in cognitive functions and poor concentration in case of exposure to microwave radiation emitted from mobile phones [2,21,22]. EMFs influence metabolic processes in the human body and exert various biological effects on cells through a range of mechanisms. EMF disrupts the chemical structures of tissue since a high degree electromagnetic energy absorption can change the electric current in the body [23]. As a result of this exposure, the functions of organs are affected. Electric fields exert an oscillatory force on every free ion on the both sides of the plasma membrane and cause them to cross it. This movement of ions causes deterioration in the ion channels on the membrane, biochemical changes in the membrane and consequently impairment of all cellular functions [24].

Exposure to EMFs can damage biological tissues by inducing changes, which can be explained in terms of thermal or non-thermal mechanisms [25]. Thermal effects can occur with the conversion and absorption of heat by the body's electromagnetic energy. Increased body temperature is stabilized and alleviated by blood circulation. Although non-thermal effects do not raise the body temperature sufficiently to impair the structure of tissues, their effects can still be seen as an increase in free radical production in tissues [3]. EMFs, no matter where they occur in the frequency spectrum, are reported to causes a rise in levels of oxygen free radicals in an experimental environment in plants and humans [26].

3. EMF-related oxidative stress and effects on tissue

Free radicals are reactive molecules produced during the conversion of foods into energy through oxygen. The formation of free radicals is an oxidation reaction that occurs on an oxygen basis. [27]. Since oxygen is essential for survival, the formation of free radicals cannot be avoided. However, factors including ionizing and non-ionizing radiation alter the transcription and translation of genes such as JUN, HSP 70 and MYC, via the epidermal growth factor receptor EGFR-ras, leading to the generation of ROS [28,29] and resulting in the overproduction of ROS in tissues [30].

The Fenton reaction is a catalytic process that converts hydrogen peroxide, a product of mitochondrial oxidative respiration, into a highly toxic hydroxyl free radical. Some studies have suggested that EMF is another mechanism through the Fenton reaction, suggesting that it promotes free radical activity in cells [31,32]. Although some researchers have reported that ROS perform beneficial function, a high degree of ROS production may cause cellular damage, resulting in a range of diseases. These radicals react with various biomolecules, including DNA (Fig. 1). Namely, the energy of free radicals is not enough, and for this reason they behave like robbers who seize energy from other cells and rob a person to satisfy themselves [33]. Many studies have suggested that EMF may trigger the formation of reactive oxygen species in exposed cells in vitro [34–37] and in vivo [7,31,38]. The initial stage of the ROS production in the presence of RF is controlled by the NADPH oxidase enzyme located in the plasma membrane. Consequently, ROS activate matrix metalloproteases, thereby initiating intracellular signaling cascades to warn the nucleus of the presence of external stimulation. These changes in transcription and protein expression are observed after RF exposure [39]. Kazemi et al. investigated the effect of exposure to 900-MHz on the induction of oxidative stress and the level of intracellular ROS in human mononuclear cells. Excessive elevation in ROS levels is an important cause of oxidative damage in lipids and proteins and nucleic acids. It therefore causes changes in enzyme activity and gene expression, eventually leading to various diseases, including sleep disorder, arthrosclerosis, loss of appetite, diabetes, dizziness, rheumatoid arthritis, cardiovascular disease, nausea and stroke [40–42]. In addition, degradation of the pro-oxidant-antioxidant balance due to an uncontrolled increase in ROS may also result in lipid peroxidation. Lipid peroxidation is the process in which cell membranes are rapidly destroyed due to the oxidation of components of phospholipids containing unsaturated fatty acids. By continuing this reaction, lipid peroxides (-CO, H) accumulate in the membrane, and transform polyunsaturated fatty acids into biologically active substances [43]. Consequently, lipid peroxidation leads to significant damage in the cells, such as disturbances in membrane transport, structural changes, cell membrane fluidity, damage to protein receptors in membrane structures, and changes in the activity of cell membrane enzymes [44]. Hoyto et al. demonstrated significant induction of lipid peroxidation after exposure to EMF in the mouse SH-SY5Y cell and L929 fibroblast cells [45]. Epidemiological studies have also suggested that oxidative damage to lipids in blood vessel walls may be a significant contributor to the development of atherosclerosis [46–48].

Studies generally focus on the brain, since cell phones are held close to the head during use. There is considerable evidence that EMF can affect neural functions in the human brain [50]. The relation between EMF and neurological disorders can be explained in terms of the heat shock response [51]. The heat shock protein (HSP) response is generally concerned with heat shock, exposure to heavy metals and environmental insults such as EMF. Generally, HSP is a marker in cells under stress. Living organisms generate stress proteins in order to survive environmental stressors. The heat shock response is regarded as a general response to a wide variety of stresses, such as oxidative stress [52]. In humans and other mammals, many environmental stimuli causes ultraviolet radiation [53], ionizing radiation [54] and laser radiation [55] are caused by cellular stresses and alter Hsp90 and 70 levels. Non-ionizing radiation also causes HSP changes in various tissues, including the brain [56], myocardium [57], testis [5] and skin [58]. Studies have described these findings as an adaptation or readjustment of cellular stress

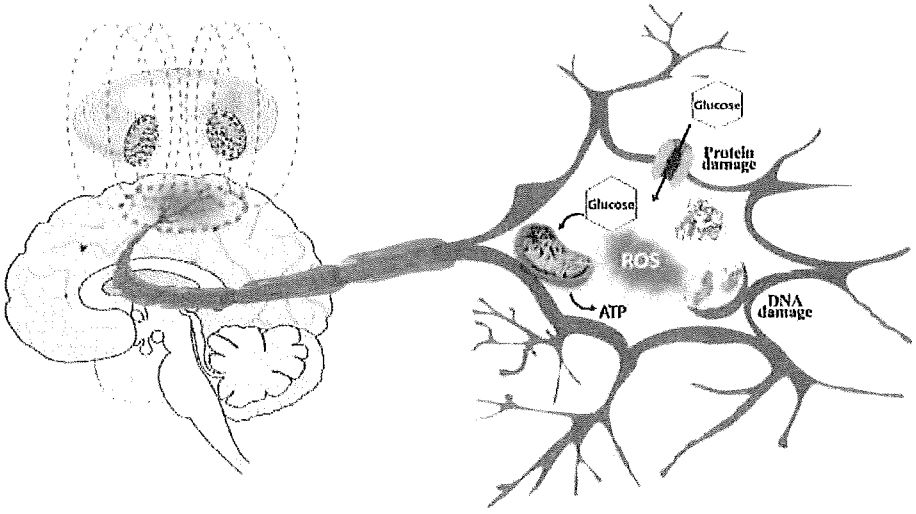

Fig 1. Reactive oxygen species generated by the effects of exposure to EMF can damage various cellular structures in neurons of the central nervous system [49].

proteins before preparing the cellular machinery for an adequate environmental change. Small, transitory readjustments of the circuits may thus decisively influence overall stress tolerance [59,60].

Low frequency (0–300 Hz) and RF (10 MHz–300 GHz) EMF has also been reported to alter the permeability of the blood–brain barrier [61–63]. At the same time, these changes in the blood-brain barrier may lead to excess accumulation of heavy metals and specifically of iron in the brain. This effect may trigger several neuronal disorders [64,65]. Some studies have reported that DNA damage and blood–brain barrier disruption is connected, and that autism spectrum conditions are associated with EMF exposure. The disruption of fertility and reproduction associated with EMF/RFR may also be related to the increasing incidence of autism spectrum conditions [66–68].

Oxidative stress plays an important role in DNA damage process, general and specific gene expression and cell apoptosis. The brain has a high metabolic rate, making it more prone to damage by ROS and oxidative damage compared to other organs [69]. Excessive amounts of ROS in tissues may lead to necrosis, the death of neurons and neuronal damage in brain tissue, as well as to neurological disorders such as Alzheimer's disease, spinal cord injury, multiple sclerosis, and epilepsy [70] (Fig. 2). Several studies have observed neuronal damage and cellular losses caused by exposure to EMF in many regions of the brain, including the cortex, basal ganglia, hippocampus and cerebellum [71–75]. One epidemiological study determined an association between amyotrophic lateral sclerosis and exposure to high intensity EMF, but no correlation was observed with other neurodegenerative diseases [76]. Rubin et al. noted that the pain level of headache may increase during exposure but decreased immediately when exposure ceased [77]. Haynal and Regli suggested that exposure to extremely low frequency (ELF)-EMF may be linked to amyotrophic lateral sclerosis, a fatal neurodegenerative disorder [78]. Maskey et al. investigated the effects on the brain of 835-MHz over different exposure times and observed a significant loss of pyramidal cells in the CA1 region of the hippocampus [79]. Another case control study by Villeneuve et al. reported a 5.3-fold increased risk of one brain cancer type, glioblastoma, in individuals exposed to EMF, but no increased risk for other brain cancers [80].

Some studies have shown that microwave exposure failed to induce a detectable genotoxic effect by itself, and have reported interference with DNA-repair mechanisms [82–85]. Oxidative damage in DNA occurs as a result of interaction between free radicals and DNA, with the addition of bases or abstractions of hydrogen atoms from sugar moiety. Modified nucleotides emerge as products of damage (8-OH-dG) when DNA is modified by the oxidative damage caused by reactive oxygen molecules [86]. These products are markers of oxidative stress measured using analytical methods [87,88]. Agarwal and Saleh and Aitken et al. have reported that ROS may have harmful effects on sperm DNA and other biomolecules, proteins and lipids, consequently leading to male infertility [89,90].

At the same time, men carrying phones in their pocket or on their belt and therefore, most of adverse effects of the EMF are seen in reproductive organs. Sepehrimanesh et al. showed that exposure to RF-EMF produces increases in testicular proteins in adults that are related to carcinogenic risk and reproductive damage [6]. Neuroendocrine changes caused by EMFs are a key factor in changing hormone functions [91]. Eroğlu et al. stated that exposure to cell phone radiation reduces the motility and changes the morphology of isolated sperm cells. They also discussed the effects of EMFs on female infertility [92]. Goldhaber et al. reported a significant increase in fetal abnormalities and spontaneous abortions in pregnant women exposed to EMF [93]. Many of these effects may occur due to hormonal changes [94,95].

Studies on the effects of EMF on tissues discussed here are set out in Tables 1 and 2.

4. The antioxidant defense system and EMF

Antioxidant defense systems have developed in organisms to control the formation of free radicals and to prevent the harmful effects of these molecules [122]. These antioxidants reduce or impair the damage mechanism of ROS via their free radical scavenging activities [123]. Two major mechanisms have been identified for antioxidants [124]. The first is a mechanism of chain disruption in which the primary antioxidant releases an electron to the free radical found in the systems. The second mechanism includes elimination of the initiators of species of ROS/reactive nitrogen (secondary antioxidants) by suppressing chain-initiating catalysts. Antioxidants may also impact on biological systems by various mechanisms involving electron releasing, metal ion chelation, co-antioxidants, or by maintaining the expression of genes [125]. If these antioxidant defense mechanisms are impaired through exposure to an agent that causes the overproduction of ROS, including EMF, antioxidants may not be sufficient or free radical formation may increase to such an extent that it overpowers the defense

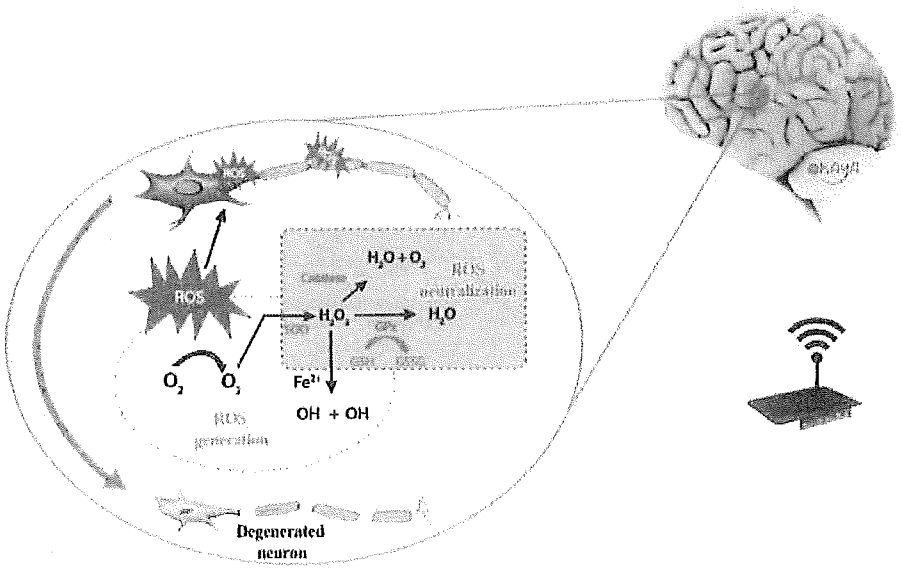

Fig. 2. The role of EMF emitted from several devices, depicting an increase in the generation of ROS and consequent oxidative stress in the central nervous system resulting from the inability of the antioxidant defense system to cope with this increase in ROS [81].

capabilities of antioxidants [10]. This is known as oxidative stress. EMFs can initiate various biochemical and physiological changes, including oxidative stress, in the systems of various species. Several studies in the literature show that plasma membrane receptors are possible targets for field interactions [126,127].

Generally, antioxidants have been divided into exogenous groups (carotene, C, and vitamin E), and endogenous groups (melatonin (MEL)), SOD, GSH-Px, CAT, including; protein (MEL), vitamins (vitamin C), trace elements (Mg, Se), complexes of compound, hydrophilic (ascorbic acid, urate, flavonoids) and hydrophobic (β-carotene, α-tocopherol) substances, with direct impacts (SOD, CAT), and indirect effects (vitamin E). Substances with functions concerning the membrane (vitamin A and E, β- carotene), circulation (vitamin C, amino acids and polyphenols), cytosol (co-enzyme Q10) are classified as antioxidants [122,128].

4.1. Glutathione

Glutathione (GSH) is an endogenous antioxidant and an important cellular defense agent against oxidative damage. GSH reacts with the free radicals in the cell and reduces the entry of hydrogen peroxides [129]. GSH also prevents the oxidization of sulfhydryl groups in the protein structure. GSH levels in tissues are often used as a marker for measuring radical damage. It acts as a substrate for antioxidant enzymes that causes resistance to radical-induced damage, behaving like a radical scavenger. GSH is especially important for the activity of glutathione peroxidase (GSH-Px), glutathione reductase (GR) and glutathione-S-transferase (GST). In the oxidative stress process, levels of GSH decrease, while glutathione disulfide increases. In this case, accumulation of hydrogen peroxide (H_2O_2) is scavenged by the effects of reductase and glutathione peroxidase (GSH-Px). GSH-Px is also an important enzyme, which prevents damage to phagocytic cells caused by free radicals. A decrease in GSH-Px activity leads to the accumulation of hydrogen peroxide and to cell damage. GSH-Px also prevents the initiation of lipid peroxidation [65]. EMF emitted by cellular phones is known to be related to a decreased level of GSH in brain tissue and blood [97]. However, a decreased level of blood GSH may possibly be explained by an elevated oxidation rate and use of GSH during the elimination of lipid and other peroxides [130]. Awad and Hassan investigated the brains of rats exposed to 900-MHz EMF from mobile phones for 1 h/day for one week. They observed an increase in lipid peroxidation after exposure to mobile phones [131]. Aydin and Akar studied the effect of 900-MHz EMF for 2 h/day for 45 days on lymphoid organs in immature and mature rats. They reported that CAT and GPx activities decreased significantly compared to a control group. Similarly, an increase in lipid peroxidation and a concomitant demolition in GSH levels were seen in all lymphoid organs after EMF exposure, suggesting that increased levels of lipid peroxidation may have been a consequence of depleted GSH stores [132]. Luo et al. investigated that the whether the protective effects of LSPCs performed by oral gavage on oxidative stress injury induced by ELF-EMF exposure. According the results, GST activity was significantly decreased in the ELF-EMF group when compared with the control group. They found that LSPCs could effectively prohibit oxidative stress damage induced by ELF-EMF exposure, it may be related to the ability to remove free radicals and induce antioxidant enzyme activity [132]. Singh et al. investigated the biochemical mechanism of the interaction of 900-MHz mobile phone EMF with root formation in mung bean hypocotyls. The obtained results showed up regulation of the activities of antioxidant enzymes such as CAT and GR, which protect against oxidative damage induced by EMF [133]. Sepehrimanesh et al. studied that effect of 900-MHz electromagnetic field (EMF) exposure on rat serum and testes antioxidant enzyme levels. They observed that after 30 days exposure both SOD and GPx activities decreased in the long-time EMF exposure group [134]. In the other study RF-EMF exposure caused increase antioxidant stress response via increase of CAT and GR activity it lead to the generation of lipid and protein oxidative damage [135].

4.2. Catalase

CAT is a common enzyme present in organisms exposed to oxygen, such as vegetables, fruits and animals. It catalyzes the reaction that degrades hydrogen peroxide to water and oxygen. It is a crucial enzyme in the protection of the cell against oxidative damage caused by ROS. CAT exerts its peroxidase activity in vivo. It can also catalyze the reaction of oxidation, by hydrogen peroxide, of numerous metabolites and toxins, not excluding formaldehyde, formic acid, phenols, acetaldehyde and alcohols. Its basic function is to remove hydrogen peroxide and peroxide ROOH in molecular oxygen in order to prevent irreversible damage to the membranes

Table 1
Some experimental studies on the oxidative effects of EMF.

Reference	Biological endpoint	Results
Ghodbane et al. [96]	Kidney	In the study investigated that whether Static magnetic fields induces oxidative stress and apoptosis in rat tissues and to evaluate the possible protector effect of selenium (Se) and vitamin E (vit E) supplementation. In the results have been shown exposure to SMF induced oxidative stress in kidney that will be able prevented by treatment with Se or vit E.
Meral et al. [97]	Brain	890–915-MHz EMF emitted by cellular phones may generate oxidative stress. MDA levels increased and GSH level and CAT enzyme activity decreased, while vitamin A, E and D3 levels remained unchanged in the brain tissue of guinea pigs
Misa-Agustiño et al. [98]	Thymus	The thymus tissue exhibited several morphological changes, including increased distribution of blood vessels along with the appearance of red blood cells and hemorrhagic reticuloepithelial cells
Balci et al. [99]	Cornea and lens	To investigate the adverse effects of mobile-phone on the antioxidant balance in corneal and lens tissues and to observe any protective effects of vitamin C in this setting. The results of this study suggest that mobile telephone radiation leads to oxidative stress in corneal and lens tissues and that antioxidants such as vitamin C can help to prevent these effects.
Bodera et al. [100]	Antioxidant capacity of blood	EMF exposure at 1800 MHz significantly reduced antioxidant capacity in both healthy animals and those with paw inflammation
Ozorak et al. [101]	Kidney and testis	In the present study was investigated that the effects of both Wi-Fi and 900 and 1800 MHz EMF on oxidative stress and trace element levels in the kidney and testis of growing rats from pregnancy to 6 weeks of age. It has been observed Wi-Fi and mobile phone-induced EMR may cause precocious puberty and oxidative kidney and testis injury in growing rats.
Ozgur et al. [102]	Liver and kidney	RF exposure is reported to induce lipid peroxidation, accompanied by decreased activity of superoxide dismutase (SOD), myeloperoxidase (MPO) and glutathione peroxidase (GSH-Px), in various organs, such as guinea pig liver and rat kidney
İkinci et al. [103]	Spinal cord	The aim of this study was therefore to investigate changes in the spinal cords of male rat pups exposed to the effect of 900 MHz EMF. The study results showed that MDA and GSH levels in EMFG increased significantly while CAT and SOD levels decreased following application of 900-MHz EMF pathological changes may occur in the spinal cords of male rats following exposure to 900 MHz.
Gurler et al. [104]	Brain	In the study has been investigated that the oxidative damage and protective effect of garlic on rats exposed to low level of EMF at 2.45 GHz MWR. It may be concluded that EMF increases the DNA damage in both brain tissues and plasma of the rats whereas it increases protein oxidation only in plasma. It may also be argued that the use of garlic decreases these effects.
Türedi et al. [105]	Bladder	In the study investigated the effect on male rat bladder tissues of exposure to 900 MHz EMF applied on postnatal days 22–59, inclusive. In bladder tissue, degeneration in the transitional epithelium and stromal irregularity and an increase in cells tending to apoptosis were observed in EMFG.
Yan et al. [106]	Sperm	Rats exposed to 6 hours of daily cellular phone emissions for 18 weeks exhibited a significantly higher incidence of sperm cell death than control group rats.
Rajkovic et al. [107]	Thyroid gland	After significant morphophysiological changes caused by ELF-EMF exposure, the thyroid gland recovered morphologically, but not physiologically, during the investigated repair period.
Deniz et al. [108]	Kidney	In the results was observed the 900-MHz EMR cause to kidney damage and FA may exhibit a protective effect against the adverse effects of EMR exposure in terms of the total number of glomeruli.
Wang et al. [109]	Blood-testicle Barrier	In the study investigated the effect of electromagnetic pulse (EMP) exposure on cerebral micro vascular permeability in rats. It has been shown that exposure to 200 and 400 pulses (1 Hz) of EMP at 200 kV/m can increase the permeability of the blood-testicle barrier in mice
Avendaño et al. [110]	Sperm	Four-hour EMF exposure ex vivo to a wireless internet-connected laptop caused a significant decrease in progressive sperm motility and an increase in sperm DNA fragmentation
Narayanan et al. [111]	Human semen	RF exposure for one month induced oxidative stress in the rat brain, but the magnitude differed in the various regions studied, and RF-induced oxidative stress may be one underlying causes of the behavioral deficits seen in rats after RF exposure
Hanci [112]	Spleen and thymus	900 MHz EMF applied to spleen and thymus tissue caused significant histopathological changes at the TEM and LM levels

[136]. EMF is known to impact on biological systems by increasing ROS, which causes oxidative stress by altering the CAT levels of tissues [137–139]. Odaci et al. observed a decrease in CAT levels in an EMF-exposed group. Exposure to EMF during the prenatal period also caused oxidative stress in developing rat embryos. This oxidative stress persisted through postnatal day 21 [140]. Vuokko et al. reported that EMF exposure led to depression of antioxidant systems because of raised lipid peroxidation and generation of free radicals [141]. Mobile phones triggered oxidative damage in the living cell by increasing the levels of xanthine oxidase and carbonyl group activity and reducing CAT activity. Treatment with MEL significantly prevents oxidative damage in the brain [142]. Özgüner et al. reported that EMF exposure leads to renal tissue damage by raising nitric oxide and malondialdehyde (MDA) levels [143].

Table 2
Some clinical studies of the oxidative effects of EMF.

Reference	Biological endpoint	Results
Lantow et al. [113]	Monocytes and lymphocytes	No significant ROS generation was measured in human cell lines exposed to 1800 MHz.
Baohong et al. [114]	Human blood lymphocytes	RF exposure for 1.5 and 4 h did not significantly exacerbate human lymphocyte DNA damage, but may reduce and increase DNA damage in human lymphocytes induced by ultraviolet C at 1.5 and 4 h incubation.
Ansarihadipour et al. [115]	Human blood proteins	EMF exacerbated oxidative damage to plasma proteins as well as conformational changes in Hb.
Wu et al. [35]	Human epithelial lens cells	RF at 4W/kg for 24 h significantly increased intracellular ROS and DNA damage.
Belyaev et al. [116]	Human blood lymphocytes	Decreased background levels of p53 binding protein 1 foci and may indicate a reduced accessibility of 53BP1 to antibodies because of stress-induced chromatin condensation.
Agarwal et al. [117]	Human ejaculated semen	900 MHz EMF emitted by mobile phones may cause oxidative stress in human semen.
Lewicka et al. [118]	Human blood platelets (in vivo)	The largest increase in ROS concentration vs. a control sample was observed after exposure to EMF of 220 V/m intensity for 60 min. The enzymatic activity of SOD-1 also decreased.
Lu et al. [119]	Human peripheral blood mononuclear cells	Cell apoptosis can be induced in human peripheral blood mononuclear cells by 900-MHz GSM radiofrequency electromagnetic field at a specific absorption rate of 0.4W/kg when exposure exceed 2 h.
De Iuliis et al. [120]	Human spermatozoa (in vitro)	Highly significant relationships were observed between SAR, the oxidative DNA damage bio-marker, 8-OH-dG, and DNA fragmentation after RF exposure.
Yao et al. [37]	Human lens epithelial cells	DNA damage was significantly increased by comet assay at 3 and 4 W/kg, whereas double strand breaks by histone variant foci were significantly increased only at 4 W/kg, while increased ROS levels were detected in the 3 and 4 W/kg groups.
Sefidbakht et al. [121]	Human embryonic kidney cells	Results showed that an increase in the activity of luciferase after 60 min of continuous exposure may be associated with a decrease in ROS levels caused by activation of the oxidative response.

4.3. Superoxide dismutase

SOD is an enzyme that catalyzes the reaction in which the toxic superoxide (O_2-) radical is partitioned into molecular oxygen (O_2) or hydrogen peroxide (H_2O_2). Superoxide is generated as a by-product as a result of the oxygen metabolism, leading to several types of damage to cells. Three forms of SOD can be encountered in humans; SOD_1 is present in the cytoplasm, SOD_2 in the mitochondria, and SOD_3 in the extracellular compartment. SOD is present in the cytosol and mitochondria and inactivates the existing superoxide radicals, as well as protecting cells from the harmful effects of the superoxide radicals [144]. Research has shown that the rat brain is susceptible to the effects of exposure to ELF-EMF. Decreased CAT and SOD activity results in after exposure suggested that EMF might change the antioxidant levels of the brain [145]. Gambari et al. reported that 50-day exposure to EMF causes oxidative stress by increasing MDA levels and reducing SOD activity, and observed that treatment with vitamin E prevented oxidative stress and lipid peroxidation in the substantia nigra [146]. Another study reported decreased antioxidant enzyme levels and increased levels of ROS in the kidneys of rats exposed to 900-MHz EMF for 30 min/day for 1 month [143].

5. Antioxidants alleviate the potential risks of EMF exposure

When applied antioxidant supplemented with EMF exposure, improved the hydrophilic, lipophilic and enzymatic antioxidant blood capacity and partially compensated for these changes [147,148]. Vitamin E (tocopherol) is one of the most important such antioxidants. Compounds of vitamin E, including alpha, beta, gamma and delta tocopherols, are soluble in lipid. Vitamin E is stored in the liver and has many functions. Its main antioxidant function is to prevent lipid peroxidation [149]. Several studies have shown the beneficial effects of vitamin E observed by reducing alteration in antioxidant capacity against the harmful effects of EMF [150,151]. Ghambari et al. observed that exposure to 3-MT EMF led to oxidative stress by reducing SOD activity and reported that treatment with vitamin E prevents the lipid peroxidation in the substantia nigra [146]. Mohammadnejad et al. studied ultrastructural changes in the thymus after exposure to EMF and investigated the protective effects of vitamin E in preventing these change. Their results demonstrated that exposure to EMF caused damage to the immune system and that vitamin E consumption can prevent ultrastructural alteration in tissue [152].

Vitamin B9 (folic acid and folate) is crucial for several functions in the human body, ranging from the production of nucleotides to homocysteine remethylation. In humans, folate is required for the body to make or repair DNA, and to methylate DNA, in addition to its function as a cofactor in various biological reactions. Moreover, this vitamin possesses antioxidant features [153]. It is especifically crucial during periods involving quick cell division and cellular growth. Folic acid (FA) is particularly required in pregnancy and for infant brain development. It is also necessary for the formation of new cells [154]. Our previous study revealed that FA prevented the adverse effect of exposure to EMF by preventing reductions in cell numbers in the cerebellum and brain. Kivrak observed that EMF triggered oxidative damage by increasing the levels of CAT activity and reducing GPx activity. They also noticed that oxidative damage in the brain was significantly prevented by FA therapy [75] (Fig. 3).

MEL is a hormone secreted by the pineal gland and that is also known as N-acetyl-5-methoxy tryptamin. It functions as a first line of defense against oxidative stress [155]. This hormone acts together with other antioxidants such as CAT, SOD and GPx to increase the effectiveness of each antioxidant. As a free radical scavenger, it possesses amphiphilic properties and can easily cross cell membranes and the blood-brain barrier [156–158]. Previous studies have shown that MEL exhibits a protective effect against EMF-induced oxidative stress [159–161]. Koc et al. showed that MEL reduced neuronal damage in the hippocampus induced by 900-MHz EMF. Ozguner et al. showed that exposure to 900-MHz EMF led to mild skin alterations [162]. Ulubay et al. stated that exposure to 900-MHz EMF in the rat kidney during the prenatal period results not only in an increase in total kidney volume, but also in decreased numbers of glomeruli. The application of MEL was found to prevent the negative effects of EMF on the kidneys [148].

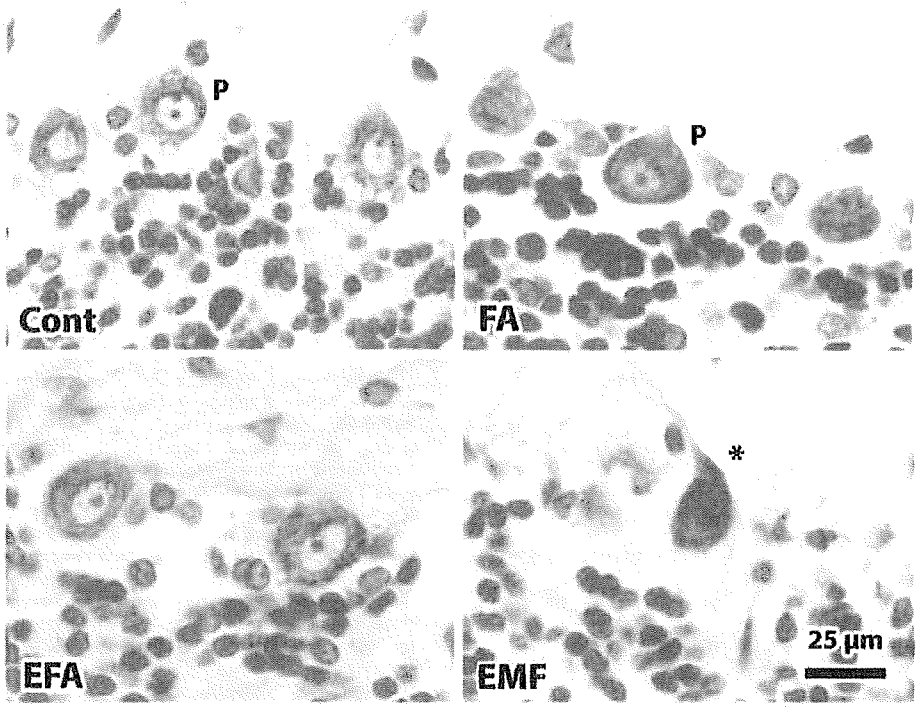

Fig. 3. Images of cerebellar tissues from the control (Cont), EMF exposure, FA and EMF + FA (EFA) groups. The letter P indicates healthy Purkinje cells in the Cont and FA groups. Necrosis of Purkinje cells is indicated with a star in the EMF group [72].

Lai and Singh demonstrated that MEL prevents EMF-induced DNA damage resulting from free radical generation in rat brain cells [31].

6. Conclusion

The biological effect of exposure to EMF is a subject of particular research interest. The results of the recent studies not only clearly demonstrate that EMF exposure triggers oxidative stress in various tissues, but also that it causes significant changes in levels of blood antioxidant markers. Fatigue, headache, decreased learning ability, and cognitive impairment are among the symptoms caused by EMF. The human body should therefore be protected against exposure to EMF because of the risks this can entail. As reported in many studies, people may use various antioxidants such as vitamin E, MEL and FA to prevent the potential adverse effects of exposure to EMF.

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EXHIBIT H

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Wi-Fi is an important threat to human health☆

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ABSTRACT

Repeated Wi-Fi studies show that Wi-Fi causes oxidative stress, sperm/testicular damage, neuropsychiatric effects including EEG changes, apoptosis, cellular DNA damage, endocrine changes, and calcium overload. Each of these effects are also caused by exposures to other microwave frequency EMFs, with each such effect being documented in from 10 to 16 reviews. Therefore, each of these seven EMF effects are established effects of Wi-Fi and of other microwave frequency EMFs. Each of these seven is also produced by downstream effects of the main action of such EMFs, voltage-gated calcium channel (VGCC) activation. While VGCC activation via EMF interaction with the VGCC voltage sensor seems to be the predominant mechanism of action of EMFs, other mechanisms appear to have minor roles. Minor roles include activation of other voltage-gated ion channels, calcium cyclotron resonance and the geomagnetic magnetoreception mechanism. Five properties of non-thermal EMF effects are discussed. These are that pulsed EMFs are, in most cases, more active than are non-pulsed EMFs; artificial EMFs are polarized and such polarized EMFs are much more active than non-polarized EMFs; dose-response curves are non-linear and non-monotone; EMF effects are often cumulative; and EMFs may impact young people more than adults. These general findings and data presented earlier on Wi-Fi effects were used to assess the Foster and Moulder (F&M) review of Wi-Fi. The F&M study claimed that there were seven important studies of Wi-Fi that each showed no effect. However, none of these were Wi-Fi studies, with each differing from genuine Wi-Fi in three distinct ways. F&M could, at most conclude that there was no statistically significant evidence of an effect. The tiny numbers studied in each of these seven F&M-linked studies show that each of them lack power to make any substantive conclusions. In conclusion, there are seven repeatedly found Wi-Fi effects which have also been shown to be caused by other similar EMF exposures. Each of the seven should be considered, therefore, as established effects of Wi-Fi.

1. Introduction

Wi-Fi (also known as WiFi or WLAN) is a wireless network involving at least one Wi-Fi antenna connected to the internet and a series of computers, laptops and/or other wireless devices communicating wirelessly with the Wi-Fi antenna. In this way, each such wireless communication device can communicate wirelessly with the internet. All the studies reviewed here were of Wi-Fi using the 2.4 GHz band, although there is also a 5 GHz band reserved for possible Wi-Fi use.

Telecommunications industry-linked individuals and groups have claimed that there are no and cannot possibly be any health impacts of Wi-Fi (Foster and Moulder, 2013; Berezow and Bloom, 2017). However with Wi-Fi exposures becoming more and more common and with many of our exposures being without our consent, there is much concern about possible Wi-Fi health effects. This paper is not focused on anecdotal reports but rather on 23 controlled, scientific studies of such health-related effects in animals, cells including human cells in culture and in human beings (Table 1).

Each of the effects reported above in from 2 to 11 studies, have an extensive literature for their occurrence in response to various other non-thermal microwave frequency EMFs, discussed in detail below. These include (see Table 1) findings that Wi-Fi exposures produce impacts on the testis leading to lowered male fertility; oxidative stress; apoptosis (a process that has an important causal role in neurodegenerative disease); cellular DNA damage (a process causing cancer and germ line mutations); neuropsychiatric changes including EEG changes; hormonal changes.

The discussion here focuses on those Wi-Fi effects which have been found by multiple Wi-Fi studies and have been previously confirmed by non-thermal exposures to other microwave frequency EMFs. The 1971/72 U.S. Office of Naval Medical Research study (Glaser, 1971) reported the following changes related to testis or sperm: 1. Decreased testosterone leading to lowered testis size. 2. Histological changes in testicular epithelial structure. 3. Gross testicular histological changes. 4.

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Table 1
Summary of health impacts of Wi-Fi EMF exposures.

Citation(s)	Health Effects
Atasoy et al. (2013); Ozorak et al. (2013); Ayaşlı et al. (2013); Çiftçi et al. (2015); Tok et al. (2014); Çiğ and Nazıroğlu (2015); Ghazizadeh and Nazıroğlu (2014); Yüksel et al. (2016); Oltman et al. (2017a, 2017b); Topsakal et al. (2017)	Oxidative stress, in some studies effects lowered by antioxidants
Atasoy et al. (2013); Shokri et al. (2015); Dasdag et al. (2015); Avendaño et al. (2012); Yildirim et al. (2015); Ozorak et al. (2013); Oni et al. (2011); Akdag et al. (2016)	Sperm/testicular damage, male infertility
Pamparopoulou et al. (2011); Maganioti et al. (2010); Oltman et al. (2017a, 2017b); Hassanshahi et al. (2017)	Neuropsychiatric changes including EEG; prenatal Wi-Fi leads to post-natal neural development, increased cholinesterase; decreased special learning; Wi-Fi led to greatly lowered ability to distinguish familiar from novel objects, changes in GABA and cholinergic transmission
Shokri et al. (2015); Dasdag et al. (2015); Çiğ and Nazıroğlu (2015); Topsakal et al. (2017)	Apoptosis (programmed cell death), elevated apoptotic markers
Avendaño et al. (2012); Atasoy et al. (2013); Akdag et al. (2016)	Cellular DNA damage
Salti et al. (2015); Yüksel et al. (2016); Topsakal et al. (2017)	Endocrine changes incl.: Catecholamines, pancreatic endocrine dysfunction, prolactin, progesterone and estrogen
Çiğ and Nazıroğlu (2015); Ghazizadeh and Nazıroğlu (2014)	Calcium overload
Ayaşlı et al. (2013)	Melatonin lowering; sleep disruption
Oltman et al. (2017a)	MicroRNA expression (brain)
Oltman et al. (2017a)	Abnormal postnatal development
Çiftçi et al. (2015)	Disrupts development of teeth
Salti et al. (2015)	Cardiac changes, blood pressure disruption; erythrocyte damage
Lee et al. (2014)	Growth stimulation of adipose stem cells (role in obesity?)

Decreased spermatogenesis. Glaser (1971) also reported a total of 35 neurological/neuropsychiatric effects of non-thermal EMF exposures, including 9 central nervous system effects, 4 autonomic system effects, 17 psychological disorders, 4 behavioral changes and EEG changes. It also reported 7 types of chromosomal aberrations several of which are known to be caused by chromosomal double stranded DNA breaks, 8 types of endocrine changes, and cell death (what we now call apoptosis). Glaser (1971) also provided over 1000 different citations each reporting various types of non-thermal microwave frequency EMF effects. Consequently, the existence of 5 types of Wi-Fi effects, each supported by multiple Wi-Fi studies were already well-supported *as general non-thermal EMF effects* back in 1971, 47 years ago: effects on the testis and sperm production, neurological/neuropsychiatric effects, endocrine effects, attacks on cellular DNA and increased apoptosis/cell death.

The 146 page review published by Tolgskaya and Gordon (1973) found that in studies of histological changes in rodents, the three most sensitive organs in the body to non-thermal microwave EMFs were the nervous system (including the brain), followed closely by the heart and the testis. They also reported changes in neuroendocrine tissues and increased cell death in multiple tissues. Thus those pre-1973 rodent studies already showed that other EMFs caused 4 of the repeated, recently documented Wi-Fi effects: changes in testis structure/function, neurological effects, increased cell death (possibly via apoptosis) and endocrine effects. Findings from our longer list of EMF reviews of non-thermal effects are summarized in Table 2.

Each of the 7 Wi-Fi effects found in 2–11 studies (Table 1), have also been found to be caused by other microwave frequency EMFs, in a much larger literature (Table 2). From 10 to 16 reviews extensively document each of these seven effects as general microwave frequency effects (Table 2). These are, therefore, general effects produced by such EMFs. Each of these 7 repeatedly found Wi-Fi effects should, therefore, be considered established Wi-Fi effects. The author is not aware of any genuine Wi-Fi studies on these 7 effects that reported no statistically significant evidence of effect.

Each of these 7 is very serious: Oxidative stress has causal roles in most chronic human diseases; cellular DNA damage can cause cancer, thus producing a partial explanation for EMF cancer causation; because such DNA damage occurs in sperm cells (Atasoy et al., 2013; Avendaño et al., 2012; Akdag et al., 2016; Adams et al., 2014; Liu et al., 2014; Asghari et al., 2016), such damage is highly likely to produce mutations that impact future generations; calcium overload is highly likely to be the cause of each of these various other effects, as discussed below; apoptosis has central roles in neurodegenerative diseases; the neuropsychiatric effects are almost certainly caused by the impact of EMFs on brain structure which is extensively documented and, in my opinion, produces many impacts (Pall, 2016b). A recent meta-analysis shows major lowering of sperm counts and sperm quality in many countries around the world, with declines of over 50% in all advanced technology countries (Levine et al., 2017). The senior author of this study suggested that this effect alone may lead to human extinction (No authors listed, 2017). Given the major impact of EMF exposures on sperm count and quality in human and in animal studies, the pattern of evidence on male fertility is very worrying.

One thing needs to be clarified, here, however. In the two studies on calcium overload following Wi-Fi exposure, such overload was measured a substantial time period following exposure. Overload was shown to be caused, to a substantial effect, by increased TRPV1 receptor activity (Çiğ and Nazıroğlu, 2015; Ghazizadeh and Nazıroğlu, 2014). The TRPV1 receptor is known to be activated by oxidative stress. It is my view, discussed in detail below, that there is a central mechanism that acts to produce excessive intracellular calcium immediately following EMF exposure and that the oxidative stress/TRPV1 activation is secondary.

We have then, major impacts of non-thermal EMF exposures on both of the most important intercellular regulatory systems in the body, the nervous system and the endocrine systems. We have major impacts on what may be the most important intracellular regulatory system, the calcium regulatory system. And we also have non-thermal EMFs attacking the DNA of our cells, putting our biological inheritance at great risk. As living organisms, EMFs attack each of the most important functions that go to the heart of our human complexities.

Despite all of these clear and important, non-thermal effects, and the fact that there was substantial evidence for many of them already known before 1973, our current U.S. and international safety guidelines are still based on considering only thermal effects.

2. Wi-Fi and other wireless communication EMFs are pulsed, leading to larger biological impacts; These EMFs are also polarized, also producing larger effects; Dose response curves are often both non-linear and non-monotone

There are three patterns of EMF action, each of which is very important and each of which is almost universally ignored by the

Table 2
Reviews of Non-thermal Effects of Microwave Frequency EMFs Similar to Those Found in Multiple Wi-Fi Studies.

Non-thermal effects	Citations
Cellular DNA damage	Glaser (1971); Yakymenko et al. (1999); Aitken and De Iuliis (2007); Hardell and Sage (2008); Hazmi et al. (2008); Phillips et al. (2009); Roeliger (2009); Makker et al. (2009); Yakymenko and Sidorik (2010); Batista Napotnik et al (2010); Yakymenko et al. (2011); Pall (2013, 2015b); Asghari et al. (2016); Pall (2018)
Changes in testis structure, lowered sperm count/ quality	Glaser (1971); Tolgskaya and Gordon (1973); Aitken and De Iuliis (2007); Hazmi et al. (2008); Desai et al. (2009); Gye and Park (2012); Nazıroğlu et al. (2013); Carpenter (2013); Adams et al. (2014); Liu et al. (2014); Houston et al. (2016); La Vignera et al. (2012); Makker et al. (2009)
Neurological/neuropsychiatric effects	Glaser (1971); Tolgskaya and Gordon (1973); Raines (1981); Lai (1994); Grigor'ev (1996); Hardell and Sage (2008); Makker et al. (2009); Khurana et al. (2010); Levitt and Lai (2010); Consales et al. (2012); Carpenter (2013); Pall (2016b); Belyaev et al. (2016); Sangün et al. (2016); Kaplan et al. (2016)
Apoptosis/cell death	Glaser (1971); Tolgskaya and Gordon (1973); Raines (1981); Yakymenko et al. (1999); Batista Napotnik et al. (2010); Yakymenko and Sidorik (2010); Pall (2013, 2016b); Asghari et al. (2016); Sangün et al. (2016)
Calcium overload	Adey (1981, 1988); Walleczek (1992); Yakymenko et al. (1999); Gye and Park (2012); Pall (2013, 2015a, 2015b, 2016a, 2016b)); Asghari et al. (2016)
Endocrine effects	Glaser (1971); Tolgskaya and Gordon (1973); Raines (1981); Hardell and Sage (2008); Gye and Park (2012); Hardell and Sage (2008); Makker et al. (2009); Pall (2015b); Sangün et al. (2016); Asghari et al. (2016)
Oxidative stress, free radical damage	Raines (1981); Houston et al. (2016); Hardell and Sage (2008); Hazmi et al. (2008); Desai et al. (2009); Yakymenko and Sidorik (2010); Yakymenko et al. (2011); Consales et al. (2012); La Vignera et al. (2012); Nazıroğlu et al. (2013); Yakymenko et al. (2015); Pall (2013, 2018); Dasdag and Akdag (2016); Wang and Zhang (2017)

telecommunications industry and industry-linked organizations. The most extensively reviewed of these is that pulsed EMFs are usually much more biologically active than are non-pulsed (also known as continuous wave) EMFs of identical frequency and similar average intensity (Osipov, 1965; Pollack and Healer, 1967; Creighton et al., 1987; Grigor'ev, 1996; Belyaev, 2005, 2015; Markov, 2007; Van Boxem et al., 2014; Pall, 2015b; Panagopoulos et al., 2015b). This pattern of action is particularly important because all wireless communication devices, including Wi-Fi (Panagopoulos et al., 2015b; Maret, 2015) communicate via pulsations and are likely to be particularly dangerous as consequence of this. Panagopoulos et al., 2015b have argued that the more pulsed they are, the more damaging EMFs will be and while this may still be questioned, it may well be a roughly applicable generalization.

It is also true that artificial EMFs are polarized and this makes artificial EMFs particularly dangerous (Belyaev, 2005, 2015; Panagopoulos et al., 2015a). Polarized EMFs put much larger forces of electrically charged chemical groups than do non-polarized EMFs (Panagopoulos et al., 2015a), an observation that is relevant to the main mechanism of EMF action in living cells discussed below.

It has often been found that there are windows of exposure where specific intensity ranges produce maximum biological effects, which drop off going to both lower or higher intensities (Belyaev, 2005, 2015; Pall, 2015b). It can be seen from this that dose-response curves are often both non-linear and non-monotone whereas industry linked groups often assume a linear and therefore monotone dose-response curve.

3. EMF effects are often cumulative and irreversible

One question that has been raised about the effects of these low-intensity EMFs producing biological effects is are they cumulative? I am aware of three different types of evidence for cumulative effects. Three of the human occupational exposure studies from the 1970's reviewed in Raines (1981), showed that effects increased substantially with increasing time of exposure to a particular type and intensity of EMF.

The impacts of such EMFs on animal brains were reviewed in Tolgskaya and Gordon (1973) and discussed in Pall (2016b). Initially exposures over period of 1–2 months produced relatively modest changes in structure of the brain and the neurons and when exposures ceased, most of the structural changes disappeared – that is the changes were largely reversible. However more months of exposure produced much more severe impacts on brain and neuronal structure and these were irreversible (Tolgskaya and Gordon, 1973; Pall, 2016b).

Magras and Xenos (1997) put pairs of young mice into cages on the ground at two locations each with somewhat different exposures within an antenna park. The exposure levels at both sites were well within safety guidelines, so if the safety guidelines have any biological relevance, there should have been no apparent effects. It takes about 30 days for mice to go through gestation. At the higher level exposure, the pairs produced one litter of lower than normal size, and a second litter with lowered numbers of progeny; after that they were completely sterile or had extremely low fertility (Magras and Xenos, 1997). At the other site, the mating pairs produced four litters, with decreasing numbers of progeny over time followed by complete sterility. In both groups, the mating and possible subsequent gestatation for the fifth possible litter were performed under conditions of no EMF exposure, but the fertility effects were not reversed; therefore fertility effects may become irreversible, suggesting a similar pattern to the brain related effects of EMFs. It should be noted that Özorak el al (2013) showed that Wi-Fi exposure impacted animal reproduction and that (Atasoy et al., 2013; Shokri et al., 2015; Dasdag et al., 2015; Avendano et al., 2012; Yildiring et al., 2015; Oni et al., 2011; Akdag et al., 2016) suggest this as well from the Wi-Fi impacts on testis structure and sperm production.

Mutation accumulation produced by cellular DNA damage is likely to be both cumulative and irreversible, as well, because later mutations are highly unlikely to reverse previously occurring mutations.

We have therefore reason to think that such effects as brain damage to animal brains, neuropsychiatric effects in humans, reproductive dysfunction in mice and mutational effects, are each cumulative. Those same effects may be completely or largely irreversible. One thing that this should tell us is that the short-term Wi-Fi studies shown in Table 1 may *greatly underestimate* the damage Wi-Fi may do over much longer time periods. Given the fact that Wi-Fi has been placed in most schools, hotels, restaurants, coffee shops, commercial aircraft and airports as well as in many homes and that Wi-Fi hot spots are becoming increasingly common in cities around the world, we should expect massive cumulative Wi-Fi effects in many people. A second tentative inference is that false assurances of safety on the part of industry are likely to lead to much more severe effects on people exposed to Wi-Fi or other EMFs; rather than leading them to protect themselves or their children by avoiding exposures or demanding that others stop non-voluntary exposures, they are likely to avoid protective changes or be prevented from doing such protective changes. A third inference is that these effects may be among the more difficult ones for us to attribute to EMF exposure. We are much more aware of effects that occur rapidly than those that take months or years before they become readily apparent.

4. Wi-Fi and other EMFs may be particularly damaging to young people

Most arguments that have been made that microwave frequency EMFs may be much more damaging to young children have centered on the much smaller skulls and skull thickness in young children, increasing the exposure of their brains to EMFs (Gandhi and Kang, 2001; Gandhi et al., 2012). However there are other arguments to be made. EMFs have been shown to be particularly active in producing effects on embryonic stem cells (Lee et al., 2014; Belyaev et al., 2009; Marková et al., 2010; Czyz et al., 2004; Xu et al., 2016; Bhargav et al., 2015; Odaci et al., 2008; Uchugonova et al., 2008; Wang et al., 2015; Teven et al., 2013). Because such stem cells occur at much higher cell densities in children, with stem cell densities the highest in the fetus and decreasing with increasing age (Belyaev et al., 2009; Marková et al., 2010), impacts on young children are likely to be much higher than in adults. The decreased DNA repair and increased DNA damage following EMF exposure strongly suggest that young children may be increasingly susceptible to cancer following such exposures (Belyaev et al., 2009; Marková et al., 2010; Czyz et al., 2004). EMF action on stem cells may also cause young children to be particularly susceptible to disruption of brain development (Xu et al., 2016; Bhargav et al., 2015), something that may be relevant to autism causation. These are all very problematic issues and we cannot rule out the possibility that there are other problematic issues as well. Redmayne and Johansson (2015) reviewed the literature showing that there are age-related effects, such that young people are more sensitive to EMF effects. It follows from these various findings that the placement of Wi-Fi into schools around the country may well be a high level threat to the health of our children as well being a threat to teachers and any very sensitive fetuses teachers may be carrying, as well.

5. How do EMF exposures lead to non-thermal health impacts?

The author found the answer to this question in the already published scientific literature (Pall, 2013). That study showed that in 24 different studies [there are now a total of 26 Pall (2015b)], effects of low-intensity EMFs, including microwave frequency and also extremely low frequency EMFs, static electrical fields and static magnetic fields could be blocked by calcium channel blockers, drugs that are specific for blocking voltage-gated calcium channels (VGCCs). There were 5 different types of calcium channel blockers used in these studies, each thought to be highly specific, each structurally distinct and each binding to a different site on the VGCCs. In studies where multiple effects were studied, all studied effects were blocked or greatly lowered by calcium channel blockers. These studies show that EMFs produce diverse non-thermal effects via VGCC activation Pall (2013, 2014, 2015a, 2015b, 2016a, 2016b)] in many human and animal cells. In plant cells, EMFs activate somewhat similar calcium channels and produce somewhat similar effects on oxidative stress, cellular DNA damage and calcium signaling (Pall, 2016a). Furthermore, many different effects shown to be produced in repeated studies by EMF exposures, including the effects discussed above, can be produced by downstream effects of VGCC activation, via increased [Ca2+]i, as discussed in detail below.

Before leaving this issue, it is important to discuss why the VGCCs are so sensitive to activation by these low-intensity EMFs. The VGCCs each have a voltage sensor which is made up of 4 alpha helixes in the plasma membrane, with each such helix having 5 positive charges on it, for a total of 20 positive charges (Pall, 2015b). These voltage sensor helixes are each called S4 helixes because each is the fourth helix in a distinct multi-helix domain. Each of these voltage sensor charges is within the lipid bilayer part of the plasma membrane. The electrical forces on the voltage sensor are very high for three distinct reasons (Pall, 2015b, 2015a, 2016a). 1. The 20 charges on the voltage sensor make the forces on voltage sensor 20 times higher than the forces on a single charge. 2. Because these charges are within the lipid bilayer section of the membrane where the dielectric constant is about 1/120th of the dielectric constant of the aqueous parts of the cell, the law of physics called Coulomb's law, predicts that the forces on those charges will be approximately 120 times higher than the forces on charges in the aqueous parts of the cell. 3. Because the plasma membrane has a high electrical resistance whereas the aqueous parts of the cell are highly conductive, the electrical gradient across the plasma membrane is estimated to be concentrated about 3000-fold. The combination of these effects means that comparing the forces on the voltage sensor with the forces on singly charged groups in the aqueous parts of the cell, the forces on the voltage sensor are approximately $20 \times 120 \times 3000 = 7.2$ million times higher (Pall, 2015b). The physics predicts, therefore, extraordinarily strong forces activating the VGCCs via the voltage sensor. It follows that the biology tells us that the VGCCs are the main target of the EMFs and the physics tells us why they are the main target. Thus the physics and biology are pointing in the same direction.

There are also additional findings pointing to the voltage sensor as the direct target of the EMFs. In addition to the VGCCs, there are also voltage-gated sodium, potassium and chloride channels, with each of these having a voltage sensor similar to those found in the VGCCs. Lu et al. (2015) reported that voltage gated sodium channels, in addition to the VGCCs were activated by EMFs. Tabor et al. (2014) found that Mauthner cells, specialized neurons with special roles in triggering rapid escape mechanisms in fish, were almost instantaneously activated by electrical pulses, which acted via voltage-gated sodium channel activation to subsequently produce large [Ca2+]i increases. Zhang et al. (2016) reported that in addition to the VGCCs, potassium and chloride channels were each activated by EMFs, although these other voltage-gated ion channels had relatively modest roles compared with the VGCCs in producing biological effects. Each of these three studies, the Lu et al. (2015) study, the Tabor et al. (2014) study and the Zhang et al. (2016) study used specific blockers for these other voltage-gated ion channels to determine their roles. The Tabor et al. (2014) study also used genetic probing to determine the role of the voltage-gated sodium channels. Lu et al. (2015) also used whole cell patch clamp measurements to measure the rapid influx of both sodium and calcium into the cell via the voltage-gated channels following EMF exposure. Sodium influx, particularly in electrically active cells, act in the normal physiology to depolarize the plasma membrane, leading to VGCC activation such that the voltage-gated sodium channels may act primarily via indirect activation of the VGCCs. In summary then, we have evidence that in animal including human cells, seven distinct classes of voltage-gated ion channels are each activated by EMF exposures: From the Pall (2013) review, four classes of voltage-gated ion channels were shown from calcium channel blocker studies, to be activated by EMFs, L-type, T-type, N-type and P/Q –type VGCCs. In this paragraph we have evidence that three other channels are also activated, voltage-gated sodium channels, voltage-gated potassium channels and voltage-gated chloride channels. Furthermore the plant studies strongly suggest that the so called TPC channels, which contain a similar voltage sensor, are activated in plants allowing calcium influx into plants to produce similar EMF-induced responses (Pall, 2016a). One can put those observations together with the powerful findings from the physics, that the electrical forces on the voltage-sensor are stunningly strong, something like 7.2 million times stronger than the forces on the singly charged groups in the aqueous phases of the cell. Now you have a stunningly powerful argument that the voltage sensor is the predominant direct target of the EMFs.

There is one additional finding that should be discussed here. In a study published by Pilla (2012), it was found that pulsed EMFs produced an "Instantaneous" increase in calcium/calmodulin-dependent nitric oxide synthesis in cells in culture. What Pilla (2012) showed was that following EMF exposure, the cells in culture, must have produced a large increase in [Ca2 +]i, this in turn produced a large increase in

nitric oxide synthesis, the nitric oxide diffused out of the cells and out of the aqueous medium above the cells into the gas phase, where the nitric oxide was detected by a nitric oxide electrode. This entire sequence occurred in less than 5 s. This eliminates almost any conceivable indirect effect, except possibly via plasma membrane depolarization. Therefore that the pulsed EMFs are acting directly on the voltage sensors of the VGCCs and possibly the voltage-gated sodium channels, to produce the [Ca2 +]i increase.

Why is it that the VGCCs, acting via calcium influx, seem to be much more important in producing EMF effects than are the other voltage-gated ion channels? Probably for three reasons: 1. Ca^{2+} ions under resting conditions in cells have about a 10,000-fold concentration gradient driving them into the cell, and over a million-fold electrochemical gradient also driving them into the cell. Because of this, one can have huge calcium influxes upon channel activation. 2. [Ca2 +]i produces many important regulatory effects, such that over activation of those effects can have very large pathophysiological consequences. 3. Sustained elevation of [Ca2+]i produces major cell damage.

6. How can the Wi-Fi effects be produced by EMF triggered VGCC activation?

Can the various effects produced by Wi-Fi and by other microwave frequency EMFs be produced by the downstream effects of VGCC activation? In order to determine that, one needs to consider the various downstream effects of VGCC activation, summarized in Fig. 1 and how these are likely to produce each of the effects of Wi-Fi and other microwave frequency EMFs. Let's consider Fig. 1.

As shown in the top left section of Fig. 1, microwave and lower frequency EMFs act via VGCC activation to produce increases in intracellular calcium [Ca2+]i. All of the downsteam effects of VGCC activation considered in Fig. 1 are produced by elevated (often excessive) [Ca2+]i.

Just to the right of [Ca2+]i in Fig. 1, you will see that elevated [Ca2+]i produced increases in nitric oxide (NO) synthesis. This is because two of the three types of enzymes producing NO are calcium-dependent. There is an NO signaling pathway that goes through increased cGMP and increased protein kinase G activity. Protein kinase G can act by raising the activity of the transcriptional regulatory factor, Nrf2, to produce the therapeutic effects produced by EMF exposures (Pilla, 2013; Pall, 2014; Pall and Levine, 2015).

High levels of NO can bind to heme groups on cytochromes (uppermost section, Fig. 1) inhibiting cytochrome oxidase, the terminal oxidase in the mitochondria, inhibiting ATP synthesis. NO can also inhibit cytochrome P450s involved in steroid hormone synthesis, lowering levels of estrogen, progesterone and testosterone (sex hormones).

The main pathophysiological effects of EMF exposures are produced via excessive calcium signaling (lower left) and the peroxynitrite pathway (lower right). Peroxynitrite levels are elevated because both NO and superoxide are elevated by increased [Ca2+]i with NO and superoxide reacting with each other to form peroxynitrite. Peroxynitrite and its CO_2 adduct, can break down to produce reactive free radicals, hydroxyl radical, carbonate radical and NO_2 radical which produce oxidative stress. These various oxidants act to produce greatly elevated NF-kappaB activity, leading to inflammation. All of this biochemistry and physiology is well-accepted and widely known with a single exception: The role of protein kinase G in raising Nrf2 has only recently been reviewed (Pall and Levine, 2015).

The ways in which these mechanisms can produce each of the seven effects produced by Wi-Fi, as well as other microwave frequency EMFs, are described in Table 3.

It can be seen from Table 3, that there are plausible mechanisms by which each of those seven effects can be produced by VGCC activation via known pathways. Given the complexities of biology, the mechanisms described in Table 3 may, in some cases, be over simplified.

There is one other finding, not related to the Wi-Fi findings, that is included in Table 3. A question that was raised in review of the paper was whether the heat shock stress elevation found following EMF exposure in many studies, could be produced by VGCC activation. As you can see from Table 3, it can be.

7. Other proposed biophysical mechanisms

One question that can be asked is how the VGCC activation mechanism compares with other biophysical models of non-thermal EMF effects. Belyaev (2015) has discussed a number of what he describes as biophysical models which are, therefore considered here. These models are basically theoretical models of how the weak electrical forces of the EMFs can interact with biologically plausible structures to produce non-thermal effects.

The first of these Belyaev considers is Fröhlich's theory. This is where there are "coherent longitudinal vibrations of electrically polar structures." The mechanism of Fröhlich's theory will not be considered here (the reader is referred to Belyaev, 2015). The author considers this to be a plausible mechanism for possible production of some non-thermal EMF effects. However, there are no specific testable predictions made by the theory that suggest how it could be tested, given the fact that there may be multiple possible targets of the EMFs according to

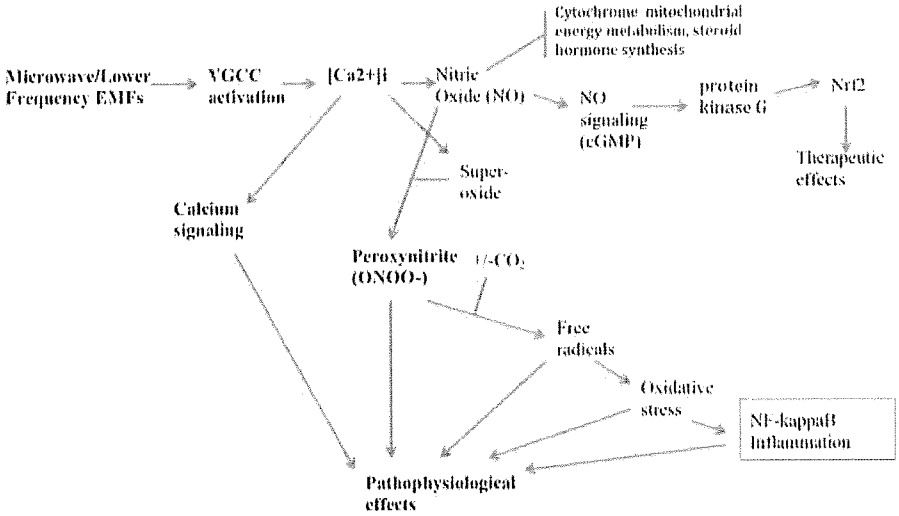

Fig. 1. Various pathways of action by which EMF VGCC activation can produce effects produced by EMF exposure (modified, with permission from Pall, 2015b).

Table 3
How Eight Established Effects of Wi-Fi and Other EMFs Can Be Produced by VGCC Activation.

EMF effect	Probable mechanism(s)
Oxidative stress	Produced by elevated levels of peroxynitrite and the free radical breakdown products of peroxynitrite and its CO_3 adduct. Four studies of EMF exposure, cited in Pall (2013) showed that oxidative stress following exposure was associated with major elevation of 3-nitrotyrosine, a marker of peroxynitrite, thus confirming this interpretation. Two other studies each found 3-nitrotyrosine elevation, both following 35 GHz exposures (Sypniewska et al (2010); Kalns et al., 2000).
Lowered male/female fertility, elevated spontaneous abortion, lowered libido	Both the lowered male fertility and lowered female fertility are associated with and presumably caused by the oxidative stress in the male and female reproductive organs. Spontaneous abortion is often caused by chromosomal mutations, so the germ line mutations may have a causal role. Lowered libido may be caused by lowered estrogen, progesterone and testosterone levels. It seems likely that these explanations may be greatly oversimplified. One mechanism that may be important in lowered fertility is that VGCC activation and consequent high [Ca2+]i levels is known to have a key role in avoiding polyspermy. Consquently, if this if triggered before any fertilization of an egg has occurred, it may prevent any sperm from fertilizing and egg.
Neurological/ neuropsychiatric effects	Of all cells in the body, the neurons have the highest densities of VGCCs, due in part to the VGCC role and [Ca2+]i role in the release of every neurotransmitter in the nervous system. Calcium signaling regulates synaptic structure and function in 5 different ways, each likely to be involved here. Oxidative stress and apoptosis are both thought to have important roles. Lowered sleep and increased fatigue are likely to involve lowered nocturnal melatonin and increased nocturnal norepinephrine.
Apoptosis	Apoptosis can be produced by excessive Ca2+ levels in the mitochondria and by double strand breaks in cellular DNA; it seems likely that both are involved following EMF exposure. A third mechanism for triggering apoptosis, endoplasmic reticulum stress (see bottom row in this Table), may also be involved.
Cellular DNA damage	Cellular DNA damage is produced by the free radical breakdown products of peroxynitrite directly attacking the DNA (see Pall (2018) for discussion).
Changes in non-steroid hormone levels	The release of non-steroid hormones is produced by VGCC activation and [Ca2+]i elevation. The immediate effects of EMF exposures is to increase hormone release and to raise, therefore, hormone levels. However many hormone systems become "exhausted" as a consequence of chronic EMF exposures. The mechanism of exhaustion is still uncertain, but it may involve oxidative stress and inflammation.
Lowered steroid hormone	Steroid hormones are synthesized through the action of cytochrome P450 enzymes; activity of these hormones is inhibited by binding of high levels of nitric oxide (NO) leading to lowered hormone synthesis.
Calcium overload	Produced by excessive activity of the VGCCs; secondary calcium overload is produced by oxidative stress activation of TRPV1, TRPM2 and possibly some other TRP receptors, opening the calcium channel of these receptors.
Heat shock protein induction	There is a large literature showing that excessive [Ca2+]i induces very large increases in heat shock proteins. This is thought to be produced by complex calcium signaling changes involving the endoplasmic reticulum, mitochondria and the cytosol and also involving excessive [Ca2+]i producing increasing protein misfolding (Gadhav, 2017; Park et al., 2014; Krebs et al., 2011). It should be noted that some calcium is essential for proper protein folding in the endoplasmic reticulum such that only excessive calcium leads to misfolding and consequent endoplasmic reticulum stress.

Fröhlich's theory.

A second possible mechanism involves the spin state of radical pairs. When radical pairs are generated from the breakdown of a non-radical molecule, these radical pairs often react back with each other to form another non-radical molecule, not necessarily identical to the original non-radical. What is postulated by this theory is that EMFs can interact with one or both radicals, changing their spin state and greatly lowering their ability to react back with each other, thus generating increased free radicals and therefore increased oxidative stress. The potential strong point of this theory is that it provides an explanation for the oxidative stress found following EMF exposure. However, as noted under oxidative stress in Table 3, there are 6 studies where oxidative stress following EMF exposure was associated with very high levels of 3-nitrotyrosine, a specific marker of peroxynitrite elevation. These studies argue, therefore, that oxidative stress following EMF exposure is produced by peroxynitrite elevation and is not primarily produced by this radical pair mechanism. It follows from this that the proposed radical pair mechanism cannot even explain the properties of oxidative stress production, let alone the various consequences of non-thermal EMF exposure that do not involve oxidative stress. Does that mean that the radical pair mechanism has no possible role in producing non-thermal EMF effects? No, but it does argue there is no evidence for any such role.

A third mechanism discussed in Belyaev (2015) is the electrosoliton theory proposed by Brizhik and colleagues, involving a "self reinforcing solitary wave packet." Brizhik and her colleagues discussed this in the context of reaching a threshold minimum energy state where both charged molecules and the EMF is in a coherent state, such that charge movement can ratchet from one state to another. This concept shows substantial similarity to what is thought to occur in the activation of the voltage sensor, that is discussed above. There we have four alpha helixes, each designated an S4 helix and with each S4 helix having 5 positive charges, with the 4 S4 helixes together making up the voltage sensor. Most of those positive charges are 3 amino acid residues apart from each other, such that the closest charged residues stick out from the helix pretty much on the same side of the helix. Three of those positive charges are electrostatically attracted to negative residues on other helixes thought to be in fixed positions. What is thought to happen in activation is that there a ratcheting of the S4 helixes toward the extracellular space, ratcheting such that the negative charges are now bound to a positive charge 3 residues away from the one that was previously bound. The ratcheting also produces some turning of the S4 helix. This needs to occur several times on each of the four S4 helixes to open the channel and allow calcium ions to flow. While I don't completely understand the Brizhik electrosoliton model, it may well be relevant to our understanding the VGCC activation, because the mechanism of the voltage sensor is similar to what Brizhik and her colleagues propose to occur in the electrosoliton model. Both the electrosoliton model and the voltage sensor activation mechanism involve both charge movements and ratcheting. In order to test these biophysical models one needs to have a specific mechanism where it may apply and where such tests can be done. In the case of the voltage sensor of the VGCCs, these tests have already been done.

These models are basically theoretical models of how the weak electrical forces of the EMFs can interact with biologically plausible structures to produce non-thermal effects. Their theoretical support is their strong point. They are weak, however, in providing any compelling evidence that they have causal roles in producing non-thermal

changes in cells in culture or in whole animal (or human) studies. They are also weak because they do not provide stated explanations for the range of EMF effects that have been documented.

Belyaev (2015) discusses microwave hearing in this context. He discusses the findings showing that people can hear microwave fields that are pulsed, including pulsed low intensity EMFs. While there is no doubt that these are very interesting observations on what are clearly non-thermal effects, they do not provide a biophysical model explaining how microwave hearing may occur. It is important, therefore to ask whether such microwave hearing could be caused by VGCC activation. It has been shown that hearing involves the activation of the VGCCs (Joiner and Lee, 2015). Furthermore, various otolaryngological conditions, including tinnitus, involve excessive VGCC activity, such that the calcium channel blocker, nimodipine is useful in their treatment (Monzani et al., 2015). These findings tells us that microwave hearing may be produced by VGCC activation. Consequently, microwave hearing may be interpreted as providing further support for the VGCC mechanism.

Following microwave hearing, Dr. Belyaev (2015) discusses plasma membrane and ion models. Here the VGCC mechanisms fit into the scheme, as do the other voltage-gated ion channels and the plant TPC channels, all discussed above as being activated by their voltage sensor following EMF exposures.

Finally, Dr. Belyaev (2015) discusses possible direct effects of EMFs on DNA, possibly leading to changes in chromatin structure and/or nuclear structure. There is a literature showing that aqueous solutions of DNA absorb microwave EMFs much more efficiently than do identical solutions not containing DNA. This clearly shows that DNA has a high absorbance of the EMFs, Furthermore, there are studies showing such dissolved DNA, when it absorbs such EMFs, undergoes structural changes as measured by biophysical techniques. All of this suggests that DNA is a plausible potential target for the EMFs. The problem is what are the predicted effects of such changes in DNA structure in living cells and organisms? Dr. Belyaev spends almost a page and a half in his paper discussing various possible models of interactions of DNA or of chromatin with EMFs. But again, how do we test any of these in living cells to demonstrate a role of such DNA or chromatin changes in producing any specific or general biological effects? Given the extraordinary complexity of living cells and organisms, there are only two powerful ways of demonstrating causal roles in such living cells and organisms. These are to use genetics or to use specific pharmacological agents. The extraordinary power of each of these approaches comes from the fact that these approaches allow researchers to vary one variable at a time out of the thousands of interacting variables in a living cell, allowing us to ask does that specific variable have a causal role in determining a specific response. But these two approaches can be used when specific proteins have specific roles, not when you are looking at the role of DNA structural changes, Fröhlich's theory, radical pair mechanisms or electrosoliton models. Fortunately the VGCC mechanism does allow this approach by studying various classes of calcium channel blockers, so here we do have hard data on widespread causal roles of VGCC activation in producing EMF effects.

8. Two other models for producing non-thermal effects

With the possible exception of the electrosoliton model, the author does not find any of the models discussed by Dr. Belyaev (2015) to have substantial evidence for roles in producing EMF effects. There are two other models which may be more compelling, each of which either produces increased [Ca2+]i.

Six studies have supported the view that calcium cyclotron resonance, has a role in producing biological effects produced by *certain specific frequencies* which can interact with Ca^{2+} ions to produce a cyclotron-like resonance (Foletti et al., 2010; Gaetani et al., 2009; De Carlo et al., 2012; Lisi et al., 2008; Pazur and Rassadina, 2009; Pazur et al., 2006). In each case, the effects involved a very specific frequency which produces the calcium cyclotron resonance and in three studies, these frequencies were shown to produce increases in [Ca2+]i levels. In the De Carlo et al. (2012) study, the calcium channel blocker nifedipine was shown to greatly lower the apparent calcium cyclotron resonance effect. This finding strongly suggests that the calcium cyclotron resonance can feed Ca^{2+} ions into the VGCCs, thus increasing the flow of Ca^{2+} ions through the VGCCs into the cell following EMF exposure. The frequencies studied here for cyclotron resonance, one was close to 7 Hz and the other was close to 50 Hz, are both in the extremely low frequency range and consequently are not relevant to microwave frequency effects. The finding that only very specific calcium cyclotron resonance frequencies produce these effects is the main evidence for this mechanism.

It is now well established that there is a magnetoreception mechanism found in many animals that can detect and respond to the very low intensity geomagnetic field. This has been most studied in bees and in birds, both of whom use it for navigation. This has been suggested to involve tiny particles of magnetite which occur in bacterial, animal and plant cells, including human cells. Kirschvink (1992) first proposed a model of how such a mechanism might act. He proposed that magnetite particles may be tethered through a microtubule and/or microfilament or perhaps other fibers to a mechanosensitive channel, such that tiny magnetic forces could open the mechanosensitive channels, allowing cation flow into the cells. It is still uncertain what mechanosensitive channel or channels might be involved, but most of the candidates are channels that allow both sodium and calcium to flow into cells. Hsu et al. (2007) suggested that such magnetite particles were linked in honeybees to an undefined calcium channel, such that magnetic field exposure produces increases in [Ca2+]i. The worm Caenorhabditis elegans had been shown to have a geomagnetic orientation system. Vidal-Gadea et al. (2015) found that certain specific neurons in C. elegans which may be geomagnetic sensory neurons, very low intensity geomagnetic fields could produce increases in [Ca2+]i in those specific neurons, even when they had no synaptic inputs, suggesting that these neurons themselves acted as geomagnetic sensors.

Cadiou and McNaughton (2010) reviewed the literature on a magnetite-based magnetoreception system in birds and its role in avian migration. They also reviewed findings on neurons found in the trigeminal nerve of birds, where magnetic fields as low as 200 nT can activate specific neurons. Trains of action potentials are produced by magnetic fields, plateauing in the region of 20–100 mT. Latency in a study presented by Cadiou and McNaughton (2010) was about 4 s, but other studies have reported latencies of about 2.5 s. Therefore these are rapid effects. Cadiou and McNaughton (2010) also discuss possible roles mechanosensitive channels, including a model similar to that proposed by Kirschvink (1992) and also three other models, each involving different ways of coupling forces on magnetite to opening of a channel. Magnetoreception has also been reported to occur in a mammal, the mole-rat (Wegner et al., 2006). There are also studies of magnetic compass orientation in salmonids, newts, sea turtles and other rodents. There is evidence in Drosophila, that a magnetic structure attached to cryptochrome is involved in magnetoreception, as opposed to magnetite.

The two mechanisms described in this section have minor roles, only acting, as far as we can tell, in very specific situations. The calcium cyclotron resonance mechanism only acts with a few specific frequencies in the extremely low frequency range. The magnetoreception mechanism only acts, as far as one can tell, on detecting the weak geomagnetic fields and only acts, as far as one can tell, in certain specific neurons. It is possible that this view may change with regard to the magnetoreception mechanism but what is clear is that the VGCC mechanism is vastly more important than either of these mechanisms, acting in diverse cell types and acting to provide responses to a very wide frequency range and even to static electrical fields and static magnetic fields. Because static magnetic fields only place forces on moving electric charges, this produced a puzzle on how they can

activate the VGCCs. Pall (2013) suggested that the solution to that puzzle is that the plasma membrane of animal cells is often moving, such that the charges in the voltage sensor are also moving and can, therefore, have forces placed on them by the static magnetic fields. These static magnetic fields, activating the VGCCs can be relative low intensity but probably must be much higher intensity than the extraordinarily weak geomagnetic fields. The reader is referred to Lu et al. (2015) for empirical information from an important static magnetic field study, where those static magnetic fields activate both VGCCs and voltage-gated sodium channels.

9. Foster and Moulder on Wi-Fi

The Foster and Moulder (2013) paper argues that there are no and cannot be any health effects of Wi- Fi. The first 7½ pages of the paper are, however, largely irrelevant to that issue. These pages discuss such issues as predicted peak power output, incident power density and the FCC and international safety guidelines. They also discuss specific absorption rate (SAR) values, a measure of heating. Because it is now established, as discussed above that thermal effects are not the relevant mechanism of non-thermal effects and that VGCC activation is the main mechanism of such effects, this whole section is irrelevant. Foster and Moulder (2013) discuss the issue of biological effects, praising 7 studies listed in table 4 of their paper as having "well-characterized exposure systems" of well defined SARS values, reporting that there were no effects in the rats or mice in those 7 studies. Those 7 studies are Laudisi et al. (2012), Sambucci et al. (2010), Aït-Aïssa et al. (2010, 2012, 2013) and Poulletier de Gannes et al. (2012, 2013). The first two studies come from one research group and the other five from another, albeit with some shared personnel.

Six or those seven studies (Sambucci et al., 2010; Aït-Aïssa et al., 2010, 2012, 2013; Poulletier de Gannes et al., 2012, 2013) used an exposure system described by Wu et al. (2009) that is important here and that was claimed to produce a near uniform exposure. Laudisi et al. (2012) used a somewhat similar exposure system of Ardoino et al. (2005), albeit another one that is also claimed to produce near uniform exposures. The important features here of the Wu et al. (2009) exposure system need to be examined in the light of the fact that, as discussed above, artificial EMFs are polarized with the polarization producing much larger biological effects than natural non-polarized EMFs (Belyaev, 2005, 2015; Panagopoulos et al., 2015a). The probable important feature of these polarized EMFs is that they put much larger forces on electrically charged groups (Panagopoulos et al., 2015a); since such forces are central to VGCC activation via the voltage sensor, as discussed above, they are likely to be central to the production of most biological effects. Let's examine Wu et al. (2009) with that issue in mind. It uses a large chamber surrounded by 1 mm aluminum mesh wire mesh to provide reflections of the EMFs. The chamber in which animals are exposed on a platform at its center, is also surrounded by antennae in all 6 directions (up, down, all four horizontal directions) such that each antenna is broadcasting with one polarization is opposed (at 180°) by another broadcasting with the 180° opposite polarization, as well as by four other antennae, broadcasting with 90° different polarization in each of the four possible directions. This produces a field that is more like a non-polarized EMF rather than the usual polarized artificial EMF. This move toward non-polarization is further exacerbated by the aluminum wire reverberation system whose reflections will generate vast numbers of reflections of different polarity, like a non-polarized EMF. The consequences of this is that the structure of this exposure system is clearly very different from that seen in Wi-Fi or any other artificially produced EMF that we may be exposed to, with biological effects produced via electrical forces being vastly less. Consequently this exposure system is not only inherently different from genuine Wi-Fi, it is predicted to be inherently less active than genuine Wi-Fi, regardless of what EMFs are being fed into the 6 antennae.

There is a second type of consequence of using such reverberation exposure systems. Because of the many reverberations occurring, the path lengths of different photons reaching a specific point in the exposed tissue, will often be quite different from each other, such that the phase of the EMFs produced will also be quite different from each other. This leads to the possibility of destructive interference and thus a second mechanism which is predicted to lead to substantial decreases in the intensity of the exposures. Because exposures are usually predicted by groups using such exposure chambers without considering such destructive interference, rather than being measured, the actual exposures may be substantially lower than are the predicted exposures. Both the polarization effect and the possible difference between predicted exposure and actual exposure were considered in an earlier study.

Xiao et al. (2006), using a different reverberation exposure chamber, discussed in Fig. 1 of that paper, how the various reverberations lead to the initial polarized EMF being converted to a non-polarized or at least, less polarized EMF. They also on p. 69 if that paper compared the predicted with the measured amplitude and found that the measured amplitude was only 78% of the predicted amplitude. These findings suggest that both of the lowered polarization and destructive interference discussed in the previous two paragraphs can have substantial roles in lowering biological responses produced when using such reverberation exposure chambers.

Laudisi et al. (2012) used a different exposure system, that of Ardoino et al. (2005) where the vast majority of the exposure is produced from reflections off a long cylindrical surface in a TEM cell, where the curvature of the cylinder will also produce a largely non-polarized EMF and different reverberation paths and consequent destructive interference, may both be expected to occur. Consequently the predicted low biological activity of EMFs produced by the Wu et al. (2009) system may be expected to also occur from this TEM exposure system Ardoino et al. (2005). It is not possible to study biological effects of EMFs from Wi-Fi, cell phones or any other important exposures using such exposure systems because of the polarization changes they produce from the original polarized EMFs and because of destructive interference.

Let's now shift to the issue of the important role of pulsations in producing biological effects and ask whether the EMFs fed into the antennae have pulsation patterns similar or different from genuine Wi-Fi. Poulletier de Gannes et al. (2012) used a non-pulsed (continuous wave) as did Wu et al. (2009), an EMF which will have, therefore, much lower biological effects that genuine Wi-Fi with its myriad of pulsations (Maret, 2015). The other 6 studies (Laudisi et al., 2012; Sambucci et al., 2010; Aït-Aïssa et al., 2010, 2012, 2013; Poulletier de Gannes et al., 2013) used computers with Wi-Fi cards. Such Wi-Fi cards are designed to communicate with genuine Wi-Fi antennae, but are used here to communicate with each other, using two such computers to generate "Wi-Fi". How the EMFs so generated compare with the pulsations of genuine Wi-Fi is a complete mystery and none of these papers provide any information to allow the reader to make such a comparison. It follows that these studies (Laudisi et al., 2012; Sambucci et al., 2010; Aït-Aïssa et al., 2010, 2012, 2013; Poulletier de Gannes et al., 2013) are not studying genuine Wi-Fi, even before the effects of the reverberation chamber and the reader is left with no evidence to compare these original EMFs with genuine Wi-Fi. In summary, then none of the EMFs used in these studies are genuine Wi-Fi, with them differing from genuine Wi-Fi in three different ways: the antenna locations produce a substantial difference from genuine Wi-Fi regarding EMF polarization and this is further exacerbated by the effects of the aluminum mesh reverberation producing further lowering of any polarization; differences in path lengths of different photons produce substantial destructive interference; the initial EMF fed into the antennae differs substantially from genuine Wi-Fi, with the main concern here being due to the issue of pulsation patterns and biological effects.

Let's shift now to the claim made by Foster and Moulder (2013) that there were no effects found in any of these 7 studies. Rothman et al.,

Modern Epidemiology, 3rd Edition is a highly respected source of information, cited over 18,500 times according to the Google Scholar database. It states (p. 151, bottom) that: "A common misinterpretation of significance tests is that there no difference between two observed groups because the null test is not statistically significant, in that P is greater that the cutoff for declaring statistical significance (again, usually .05). This interpretation confuses a descriptive issue (whether two observed groups differ) with an inference about the superpopulation. The significance test refers only to the superpopulation, not the observed groups. To say that the difference is not statistically significant means only that one cannot reject the null hypothesis that the superpopulation groups are the same; it does not imply that the two groups are the same." It follows that the claim of "no effect" that Foster and Moulder (2013) make about each of these 7 studies in Table 4 of their paper is false because one can never legitimately make such a claim; one can at most claim that there were no statistically significant differences.

However there are other reasons to reject those claims that need to be considered for each of these 7 studies. Each of these 7 studies fails to provide raw numerical data, the lack of which is problematic, given the other flaws that follow. **1).** Laudisi et al. (2012) finds in Table 2, that two T cell populations are statistically significantly different in pre-natally exposed mice vs sham controls: DP and CD4SP cells are significantly affected by exposure in mice at 26 weeks after birth; CD4SP cells are affected in female mice at 5 weeks after birth ($P < .02$ in each case). Furthermore in each of the measurements in Laudisi et al. (2012), only 11 or 12 mice were studied, tiny numbers. It follows that claims in Foster and Moulder (2013) that there were no effects are false or misleading for 3 distinct reasons: You can never make such claims even in large studies; there were 3 comparisons each of which showed statistically significant effects; this study was done with tiny numbers of animals being compared and thus had extremely low statistical power. **2).** Sambucci et al. (2010) also had a tiny numbers, with 11 or 12 per group studied in Table 2, from 6 to 35 studied in Table 3 and 6 to 12 studied in Table 4. The claims of no statistically significant effects in Figs. 2, 3, 4 and 5 are based on the tiny numbers in Table 3, are therefore, based on studies with very low statistical power. **3).** The first part of the Ait-Aissa et al. (2010) paper focused on GFAP values, a measure of gliosis, which is a risk factor for glioma formation. The groups studied in Fig. 4 of Ait-Aissa et al. (2010) range from 3 to 10, so again we have tiny numbers and the authors report that none of the exposures, SAR= .08, = .4, or = 4 W/Kg produced statistically significant changes according to their statistical calculations. As in the other studies, no raw data are provided but Fig. 4 provides bar graph information which includes median values for each of the 10 different regions of the brain in these rats, control rats and also rats exposed either pre-natally or both pre-natally and post-natally. For 5 of those brain regions, M4, CA1, CA2, CA3 and DG, the median values are high enough that one can see which are higher and which are lower from the graph. It appears to this author that the median values go up from the sham exposures to the lowest intensity (= .08), that they drop going to the next intensity (= .4) and that they go up going to the highest intensity studies (= 4). You may recall (see above) that there are certain windows of exposure that give the highest biological response but with both lower and higher intensities giving lower responses. It follows that the complex apparent dose-response curve of Ait-Aissa et al. (2010), can be explained by these window effects. The question is whether any such apparent changes are statistically significant? I did, therefore a Chi-square analysis of these data, to determine statistical significance, using both the only prenatal and both prenatal and postnatal exposures (see Fig. 4 in Ait-Aissa et al., 2010). Those data show that in 10 out of 10 cases, the median value increased going from sham to .08 ($P < .002$.). Similarly, in 10 out of 10 cases, the median value drops going from .08 to .4 ($P < .002$). However in 8 out of 10 cases, the median value increases going from .4 to 4 ($P < .07$), falling just short of statistical significance. The median values increased with exposure, comparing the sham values with the values at 4 ($P < .02$). It follows from this, that three of the comparisons show statistically significant changes, and the fourth falls just short of statistical significance. Does this mean that that we should conclude that Wi-Fi can cause gliosis and thus possibly gliomas? No, but only because they did not study Wi-Fi. It should be noted, however that the long-term effects on the brain from pre-natal exposures may be relevant to autism causation.

4). Pouletier de Gannes et al. (2012) also suffered from tiny numbers in their study, with 12 to 15 rats studied in each group in Fig. 1, only 5 females in each group in Table 1, 12 to 15 rats in each group in both Table 2 and Table 3. **5).** Ait-Aissa et al. (2012) also suffers from tiny numbers of rats in the various studies. It used from 9 to 12 pregnant female rats in each group to attempt to assess EMFs impact of reproduction; it used 9 to 12 juvenile rats to determine if EMFs act to change antibody production; it used 9 to 12 young rats to determine whether EMFs impact growth over time. These tiny numbers mean that failure to find statistical significant changes has very low power to support any inferences. **6).** Ait Aissa et al. (2013) had similar problems with tiny numbers, 6 to 12 in Fig. 5, 5 to 11 in Fig. 8 and 6 to 12 in Fig. 9. **7).** Pouletier de Gannes et al. (2013) also suffers from tiny numbers. Fig. 1 groups each had 12 males or females and there were also groups of 12 studied in Table 1, Fig. 2 and Table 2. Regarding, the authors give no information regarding statistical significance or lack thereof; rather they only state that the values of these groups were "similar", without providing a definition of "similar". However in comparing the values of testis weight and epididymis weight at 4 W/Kg exposure vs sham control, they provided values for the mean and standard error of the mean (SEM). It is usually the case that when the mean values differ by more than 2.4 times the SEM, the difference is statistically significant. Here the testis weight, comparing sham with 4 W/Kg, values differed by 3.18 times the SEM and the epididymis weight differed by 3.40 times the SEM, each arguing strongly for statistical significance. This raises the question of why the authors failed to provide their P values?

An additional flaw of these 7 supposed Wi-Fi studies is that they each studied exposures of 2 h per day, 5 days per week except for one that only studied one hour per week, 5 days per day. Given that many people are exposed to Wi-Fi fields for 5, 6, 8 or more hours per day, this is another factor which argues that these studies may have been set up to minimize any effects seen.

To sum up the other flaws:

1. The 6 antennae of the reverberation chamber used in 6 out of 7 studies, minimized probable effects produced through the arrangement of the antennae in such a way as to greatly lower the polarization of the EMFs.
2. The use of 1 mm aluminum wires to produce the reverberation reflections, further decreases such polarization, again lowering probable effects. These structures are clearly very different from those found in genuine Wi-Fi, emphasizing the point that these are not genuine Wi-Fi studies, because of 1 and 2 here.
3. Differences in path lengths for different photons, produced by reverberation produce substantial destructive interference.
4. Furthermore the EMFs fed into the antennae are not genuine Wi-Fi either. It follows from this that claims that these are studies of genuine Wi-Fi made by both the authors of these individual studies and by Foster and Moulder (2013) are false.
5. The claims made by Foster and Moulder (2013) that there are no effects produced are also false; the most that may be legitimately concluded is that there is no statistically significant evidence of effects.
6. Each of the 7 studies used only tiny numbers of animals in each group studied, such that lack of statistical significance, because of the low power of these studies, drastically limits the drawing of inferences.
7. Finally, 3 out of 7 had evidence of statistically significant effects,

with each of these being ignored by Foster and Moulder.

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EXHIBIT I

Epidemiologic Evidence Relevant to Radar (Microwave) Effects

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Public and occupational exposures to microwave (RF) radiation are of two main types. The first type of exposures are those connected with military and industrial uses and, to some extent, broadcast exposures. It is this type that most of the data cited in this study draw upon. The second type, cellular telephones and their associated broadcast requirements, have raised concerns about current exposures because of their increasingly widespread use. Four types of effects were originally reported in multiple studies: increased spontaneous abortion, shifts in red and white blood cell counts, increased somatic mutation rates in lymphocytes, and increased childhood, testicular, and other cancers. In addition, there is evidence of generalized increased disability rates from a variety of causes in one study and symptoms of sensitivity reactions and lenticular opacity in at least one other. These findings suggest that RF exposures are potentially carcinogenic and have other health effects. Therefore, prudent avoidance of unneeded exposures is recommended as a precautionary measure. Epidemiologic studies of occupational groups such as military users and air traffic controllers should have high priority because their exposures can be reasonably well characterized and the effects reported are suitable for epidemiologic monitoring. Additional community studies are needed. — *Environ Health Perspect* 105(Suppl 6):1579–1587 (1997)

Key words: leukemia epidemiology, brain cancer epidemiology, nonionizing radiation epidemiology, cellular telephones and health, TV and radio broadcast towers, military electronic equipment effects

Introduction

It is widely recognized that radiation exposures such as X-rays, gamma radiation, and ingestion of radioisotopes can produce increases in the incidence of cancer in man and animals, although there is disagreement about dose–response relationships. These types of radiation, because their energy is sufficient to cause ionization, are called ionizing radiation. This is distinguished from nonionizing radiation, which includes ultraviolet (UV), visible light, 50 to 60 cycle (also called extremely low frequency radiation [ELF]), and radiofrequency or microwave (RF) radiation (*1–4*). Conventionally, it was thought that nonionizing radiation was not carcinogenic, even though there has been evidence of skin carcinogenicity from UV radiation for some time (*5*). In the last decade, there has been extensive study and evaluation of ELF following evidence that childhood leukemia increased among children who lived in homes in Denver, Colorado, near power lines and distribution facilities (*6*).

ELF studies have included extensive evaluations of occupational and residential exposures, but there has been considerable difficulty in establishing dose–response relationships or mechanisms (*7*).

Evaluation of RF exposures was conducted primarily by military- and security-oriented government agencies, and earlier studies recently have been reevaluated. Because of the rapid development and use of cellular telephone systems, which involve widespread public exposures, reevaluation of exposure risk becomes urgent. The radiation emanates mainly from handheld devices and from the many broadcast facilities needed to maintain such systems. It is generally agreed that the physical attributes of UV, ELF, and RF exposures are sufficiently different so that separate evaluations of the possible risks of each are justified. Nevertheless, some potential mechanisms of biologic reaction and dose–response relationships among different types of subjects and exposures are common to all three exposures. I discuss the types of epidemiologic evidence with possible relevance to evaluation of RF exposures.

Neoplastic responses, if they do occur, may have a long latency period. If one waits for cancer to occur, exposed populations have increased risk for the duration of the latency period. Even if cancer risks cannot yet be unequivocally demonstrated, some measure of protection should be taken as early as possible because it may take some time to determine the definitive relationship between RF and cancer.

A second reason for urgency with respect to taking protective measures is that, because of rapid increases in the numbers of persons exposed to increased RF exposures in connection with cellular telephone use, some biologic basis is needed as a guide for prudent protective behavior. It is possible that a system of biologic indicators can be found that would allow identification of increased cancer risk. This possibility seems worth exploring.

Originally, heating of tissues by RF was considered the basic mechanism through which radiation affected exposed individuals. Therefore, existing protective principles and practices are built around avoidance of the thermal effects of such exposures. There has been increasing concern that this approach may not be adequate and with this in mind, the International Conference on Non-Thermal Effects of Microwave Radiation was convened in November 1996. The proceedings of this conference are being prepared for publication.

With regard to epidemiologic evidence of radiation, a report published in January 1995 (*8*) focused primarily on military, industrial, and broadcast exposures. This study was supplemented by the review of Rothman et al. (*9*) in May 1996, Grayson's

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Abbreviations used: AE, aviation electrician's mate(s); ALL acute lymphatic leukemia; ANLL, acute nonlymphatic leukemia; AT, aviation electronics technician(s); ELF, extremely low frequency radiation (50–60 cycles); FM, frequency modulation; FSHSS, Foreign Service Health Status Study (Lilienfeld Report); FT, fire control technician(s); O/E, observed to expected; OR, odds ratio(s); RD, radarmen; RF, radiofrequency or microwave; RM, radiomen; RR, relative risk; SIR, standardized incidence ratios; TV, television; U.S. EPA, U.S. Environmental Protection Agency; UV, ultraviolet.

report on brain cancer in U.S. Air Force personnel (*10*), and by three published community studies of cancers possibly associated with proximity to broadcast facilities (*11–13*).

This review is intended only to update previous assessments of cancer risk from RF radiation. Like the earlier report (*8*), it is not intended to be a comprehensive review or a balanced report of all possibly relevant findings. Also, there is no attempt to critically evaluate these findings.

Finally, Swedish government agency assessments of ELF effects led to prudent avoidance policies (*14,15*), which may be applicable to presently available knowledge about RF. This appears to be an attractive alternative to waiting until the data are convincing enough to achieve agreement on new and enforceable standards.

Possible Effects of Radiofrequency Radiation

Evidence up to 1994 as reviewed in Goldsmith (*8*) indicates the likelihood of the following effects from exposure to RF radiation in certain populations: reproductive effects such as increased spontaneous abortion, changes in blood counts, increased somatic mutation, and increased incidence of childhood and other cancers. Other findings have suggested effects such as cataract, nonspecific disabilities, and symptoms in sensitive persons (headache, ocular problems, fatigue, dizziness, memory impairment, and sleep difficulties).

The Evidence

Reproductive Outcomes

Study of Physiotherapists. Ouellet-Hellstrom and Stewart (*16*) reported on a study of female physiotherapists who used either RF or short-wave apparatus and were queried about the outcome of pregnancies. The frequency generated by short-wave equipment was 27.12 MHz and by microwave equipment was 915 MHz and 2450 MHz.

The survey was conducted among female members of the American Physiotherapy Association in the United States. Of 11,598 respondents who reported having at least one pregnancy, 6684 (57.6%) reported using short-wave or microwave diathermy. These 6684 women reported 14,989 pregnancies, of which 1791 ended in miscarriages, called case pregnancies. Of the remaining pregnancies with sufficient data, 12,949 were classified as control pregnancies. Exposure was defined as occurring if the woman had been using one of the physiotherapy modes during the first trimester of pregnancy and during the preceding 6 months. Cases were matched to controls by mother's age at time of conception and by the number of years elapsed between the pregnancy and the date of filling out the questionnaire. A number of confounders were included, among which was prior fetal loss.

Of the case and control mothers, 11.9 and 9.5%, respectively, were using microwaves during the pregnancy; the odds ratio (OR) for spontaneous abortion increased as the number of exposures increased from 5 or less to 20 or more per month. The trend was significant whether or not prior fetal loss had occurred. For women exposed to short-wave radiation, 22.3% lost their baby prior to the 7th week of pregnancy, whereas the figure for unexposed women was 24.4%. Of the microwave-exposed women, 47.7% had miscarriages prior to the 7th week of pregnancy compared to 14.5% of nonexposed women.

Measured values of stray emissions near waist level ranged from 0.04 to 16.58 mW/cm^2 for electric fields with short-wave units, and these units produced magnetic fields of 0.09 to 8.32 mW/cm. For microwave diathermy the electric field leakage was from 0.08 to 1.20 mW. Leakage measured 15 cm from the source was as high as 15 mW/cm^2. Duration of the therapist exposures was usually only a few minutes per treatment.

Moscow Staff Study. The exposures of U.S. embassy personnel in Moscow are described in Goldsmith (*8*), based on Lilienfeld at al. (*17*) (Table 1). Studies were done among Moscow embassy employees, staff dependents, and other personnel and compared with similar groups in other Eastern European embassies.

The study known as the Foreign Service Health Status Study (FSHSS) or Lilienfeld Report (*17*) was designed to compare the experience of employees in the Moscow embassy with those of similar employees in other Eastern European embassies on the assumption that the latter were not exposed to RF radiation. There was some evidence that these employees were exposed as well, but the contract officer dismissed the possibility as being based on hearsay. In a meeting with the State Department Contract Officer Dr. Pollack about the submitted draft of the Lilienfeld Report, G. Jacobson noted that the reference to a potential infertility effect in the sutdy might be inappropriate because the experimental work was done at very high doses and there are no controlled human studies (*18*). According to the minutes of the meeting, "this clause will be modified to reflect the very speculative nature of the reports, but the FSHSS data will be presented as is" (*17*).

The final report makes no reference to any possible impact on infertility, but it does present some data (Table 1) that show more frequent complications among Moscow workers compared to those from other embassies.

Thus, we are left with higher rates of complications of pregnancy at the Moscow embassy for a problem that originally was thought to affect fertility. It seems most likely related to or actually to be spontaneous abortion.

Systematic Alterations in Red or White Blood Cell Counts

When radar was first identified as a health risk, Daily (*19*) reported a statistically significant increase in immature red blood cells among workers exposed to radar. These studies were summarized by Follis et al. (*20*). Early studies at Lockheed Aircraft (Burbank, CA) by Barron et al. (*21*) were later dismissed on the grounds "that there was variation in the interpretations by a laboratory technician" (*22*). Bach found that rats exposed at 13 mW/cm had changes in blood cell counts (*23*).

Goldoni (*24,25*) compared the hematological findings in 25 male air traffic control technicians exposed to radar with those for 10 electronic technicians whose work was distant from a microwave

Table 1. Complications of pregnancy, childbirth, and puerperium (*ICD-8*, Codes 630–678) among women employees in the Foreign Service Health Status Study (*17*).

	Ever[a,b]		After index[a,c]		
	Moscow	Comparison	Moscow	Comparison	*p*
	19 (6%)	19 (3%)	11 (3.5%)	9 (1.3%)	0.04
SMBR	1.7	0.67			

Abbreviations: *ICD, International Classification of Diseases, 8th Revision*; SMBR, standardized morbidity ratio. [a]Refers to the initial tours of duty during which exposures occurred. [b]Whether the condition occurred at any time; [c]Whether the condition occurred after the initial tour of duty.

source. The radar was in the range of 1250 to 1350 MHz, with a strength varying from 10 to 20 μW. Radar-exposed workers had significantly lower levels of leukocytes and red cells than workers distant from the microwave source. In a follow-up study of 49 radar-exposed technicians, thrombocyte and leukocyte counts decreased significantly but stayed within normal limits (*25*).

A hematologic study of Moscow foreign service workers was submitted to the U.S. government on 7 October 1976 by Tonascia and Tonascia (*26*). They found, on comparing the data for Moscow-based employees with that from foreign service exams conducted in the United States, that

> The differences between the two groups with respect to every parameter except monocytes (% and counts) are highly statistically significant ($p < 0.001$) after appropriate transformation. Specifically the Moscow group had a higher mean hematocrit, the Moscow group had a lower neutrophil percentage, but higher percentages for the other three cell types (lymphocytes, eosinophils, and monocytes). The white cell counts are strikingly higher in the Moscow group.

Several statistically significant changes occurred over time in the Moscow group; specifically, mean hematocrit increased and a 3-fold increase in monocyte count occurred. Neutrophil percentages fell and then rose; the reverse pattern was observed for the lymphocytes (*26*).

Vukelic et al. (*27*) studied the effects of RF radiation on 72 physiotherapists and physiatrists in Croatia. They found a significantly positive correlation between length of service and white cell count, and an association of years of exposure with low red cell count.

Tornqvist et al. (*28*) studied 706 power station workers at 3-year intervals and found that the white blood cell counts were decreased slightly because of exposures to magnetic fields.

Evidence of Mutational Activity in Human Incubated White Blood Cells

The initial examination of Moscow embassy workers, conducted when it became known they were being irradiated by Soviet transmitters, was done to study the possible effects of radiation on chromosomes in blood samples (*26*). Beginning in February 1966, 3 to 4 years after the microwave irradiation was first detected, samples were taken for chromosomal analysis. Twenty spreads were scored per sample; results are shown in Table 2 (*18*).

Overexposed Air Traffic Controllers. Garaj-Vrhac et al. (*29*) examined six men accidentally exposed while repairing microwave devices used for air traffic control in Zagreb. These subjects usually worked alternate days in a microwave field of 1250 to 1350 MHz with power density of 10 μg/W to 20 mW/cm^2. The accidental exposure was greater than these figures but by how much is not known. The results of chromosome aberration analysis during 1984 to 1990 showed no increase in chromosomal abnormalities compared to the control. Table 3 shows results for the accidentally exposed subjects.

Two things are clear from this experience: Microwave irradiation can produce genotoxic effects, and recovery can occur with a half-time of about 15 weeks when about one-third of the spreads show aberrations. Both chromosomal and chromatid reactions occur.

It is conventional wisdom to assume that nonionizing radiation cannot produce such changes, but there is evidence that this view is incorrect. For example, cattle in the field exposed *in vivo* near a large military RF emitter in Skrunda, Latvia (*30*), showed more positive micronuclei test results than unexposed cattle. Bovine lymphocytes *in vitro* respond to microwave exposure using the same test (*31*). Genotoxic changes are found in Chinese hamster cells *in vitro* (thymidine incorporation and chromosomal and chromatid changes) (*32*) and in human lymphocytes *in vitro* (*33*) using micronuclei tests.

A series of studies from Croatia and Italy have also demonstrated that radar exposures are mutagenic both *in vivo* and *in vitro* (*29,32–35*).

In a paper about the effect of RF radiation on the cell genome (*32*), the investigators used cultured Chinese hamster cells exposed to 7.7 GHz at power densities of 30 mW/cm^2 for 15, 30, and 60 min. Using tritiated thymidine and autoradiography, the incorporation of thymidine into DNA after a 4-hr incubation decreased in a stepwise manner according to the length of exposure and almost completely recovered

Table 2. Results of tests for chromosomal changes in metaphase spreads of lymphocytes cultured *in vitro* among selected Moscow embassy employees.

Mutagenic level[a]	Designator	Subjects, no
5	Extreme	0
4	Severe	6
3.5	Intermediate	5
3	Moderate	7
2.5	Intermediate	5
2	Questionable	5
1	Normal	6
Growth failure		2

[a]Grading of mutagenic processes and clinical interpretations of these findings were provided by Dr. G. Jacobson (George Washington University Medical School, Washington, DC), who wrote: "Patients who repeat at level 3 or higher should not reproduce until 6 months after somatic levels have returned to 2 or 1. Patients at level 4 should be withdrawn from mutagenic exposure and monitored each month until less than 3 is obtained on two consecutive samples" (*18*). Dr. Jacobson also wrote, "I feel impelled, as in past reports, to emphasize the necessity to study serial samples on the same individual and when possible to study the subject prior to exposure" (*18*). Apparently, no such follow-up or serial studies were done.

Table 3. Type and percentage of chromosomal aberrations after accidental exposure to high-power density pulsed RF radiation.

Subject no	Date	Chromatid breaks	Chromosome breaks	Acentrics	Dicentrics	Rings	Total aberrations, %
1	16/11/1990	2	1	2	1	–	3.0
	5/12/1990	–	1	1	1	–	1.5
2	6/12/1990	–	4	8	4	1	8.5
	25/02/1991	4	1	3	1	–	4.5
3	6/12/1990		3	10	3	–	8.5
	26/02/1991	4	3	3	2	–	6.0
4	20/12/1990	–	1	2		1	2.0
	16/01/1991	–	3	5	1	1	5.0
5	11/12/1990	–	6	40	9	3	33.0
	14/02/1991	1	4	31	6	2	22.0
	13/03/1991	4	7	18	6	1	18.0
	17/04/1991	6	6	6	[a]	[a]	9.5
	22/05/1991	3	4	6	2	–	7.5
	2/06/1991	1	–	5	1	–	3.5
6	20/12/1990	–	4	2	1	–	3.5
	30/01/1991	–	2	1	–	1	2.0

[a]Chromatid interchange 1.

in 24 hr. In addition, chromosomal aberrations increased stepwise according to the duration of exposure. The background percent abnormal metaphase was 1.7%; with a 15-min exposure it increased to 4.8%, with 30 min, 6.3%, and with 60 min, 8.9%. Garaj-Vhrovac et al. (*33*) report on the relationship between colony-forming ability, chromosome aberrations, and the incidence of micronuclei in V79 Chinese hamster cells exposed to RF radiation. These authors were able to demonstrate damage to cell genomes and changes in chromosome structure based on observations of structural chromosomal aberrations and micronuclei tests. The exposures used were 7.7 GHz and 30 mW for 15, 30, and 60 min. The structural changes replicated the changes observed in their initial paper. The micronuclei/1000 cells were background 0.016, and with a 15-min exposure, 0.043; 30 min, 0.050, and 60 min, 0.073. The authors believe that these results cannot be explained on the basis of cell heating.

In a third paper, Garaj-Vhrovac et al. (*34*) used human lymphocytes instead of Chinese hamster cells, and a correlation was shown between micronuclei percentages and specific chromosomal aberrations (acentric fragments and dicentric chromosomes). Temperature was held constant, and an additional level of power density of 0.5 mW/cm^2 was added. Its use led to a 2.7% aberration and 1.4% micronuclei compared to control levels of 1.5 and 0.9%.

In another paper the authors also traced the occurrence and repair of chromosomal aberrations in personnel repairing aircraft traffic control radar (*29*). The signal was ordinarily in the range of 1250 to 1350 MHz with a field strength varying from 10 μW to 20 mW/cm^2. Under ordinary exposure circumstances no long-term trend in chromosomal abnormalities was found. The six overexposed personnel were accidentally exposed to much higher levels in connection with work on equipment repair.

d'Ambrosio et al. (*35*) also found genotoxic effects of amplitude modified microwaves on human lymphocytes in culture. The signal was 9 GHz, modulated at 50 Hz with a specific absorption ratio of 90 mW/g and the exposure was for 10 min.

These findings are epidemiologically important because of the need for biological indicators of exposure and also because of the theory that somatic cell mutations lead to increased risk for cancer. The usefulness of such tests as a biological monitor seems clear from the data and the findings of excess numbers of mutations among chromosomes in the blood of the group exposed at the Moscow embassy (*17*).

In a prospective study of persons with stable mutations, Hagmar et al. (*36*) found an increase in lymphoreticular cancer, but no such effect was seen in persons with transient changes or changes of a chromatid type. A recent review by Akiyama et al. (*37*) summarizes the present understanding of the prognostic importance of somatic cell mutation.

Table 4. Age-adjusted cancer and leukemia annual incidence rates for males and females in census tracts with broadcasting towers compared to those without such towers (Honolulu, Hawaii, 1979–1983) and compared to statewide rates per 100,000 (1978–1981).

Area	Males		Females	
	Incidence	SIR[a]	Incidence	SIR[a]
All site cancer				
Tracts with towers	439.6 (488)[b]	1.45*	368.6 (417)	1.27*
Tracts without towers	318.0 (135)	1.05	246.8 (103)	0.85
Statewide	341.2 (5468)	–	272.4 (4658)	–
Leukemia				
Tracts with towers	15.2 (16)	1.58	7.6 (8)	1.45
Tracts without towers	2.4 (1)	0.27	5.0 (2)	0.97
Statewide	9.4 (163)	–	5.3 (90)	–

*$p<0.01$. [a]Standardized for age. [b]Numbers of cases in parentheses. Statewide data are based on the Surveillance, Epidemiology, and End Results Program Report 1973–1981 (*39*). Original incidence data given by Goldoni (*21*) for 5 years.

Cancer in Children and Others

Study of Broadcast Facilities and Adjacent Populations in Hawaii. A unique opportunity to study the cancer incidence in the vicinity of radio broadcasting towers occurred in Honolulu, Hawaii. This situation existed in part because the hills surrounding Honolulu are a nature preserve, so the radio towers are located in many of the populated census tracts of the city. The study includes cancer incidence data for 1978 to 1981.

Two State Health Department officals used the State Cancer Registry (*38*) to compare the cancer incidence of nine census tracts that included broadcast towers with two demographically similar tracts with no towers (*39*). The U.S. Environmental Protection Agency (U.S. EPA) measured RF radiation at 21 locations and reported that public exposures at 12 of the locations exceeded currently recommended limits. At two outdoor sites, exposures were greater than 1000 μW/cm^2, but at distances greater than 100 to 150 feet from the towers, the exposure levels generally were below 100 μW/cm^2. U.S. EPA officials stated that RF radiation in Honolulu did not pose an immediate risk to the public but officials did not comment on long-term risk. They suggested that further studies be done by the Federal Communications Commission.

The data for all cancer incidence rates and for leukemia overall for males and females, adjusted for age, are shown in Table 4. If the data are adjusted by race rather than by age, the standardized incidence ratio (SIR) for total cancer, both sexes, in tracts without towers is 1.07 compared with 1.88 in tracts with towers, the latter being significantly elevated. For leukemia, the SIR is 0.59 for tracts without broadcasting towers and 2.08 for tracts with broadcasting towers.

The Childhood Leukemia Cluster on the Waianae Coast, Hawaii. In 1985, a pediatric oncologist informed the Hawaii Department of Health that he had seen an unusual number of children with leukemia in the small communities of the Waianae coast. This situation was confirmed by the Hawaii Cancer Registry in 1986 (*40*). In 1990 the department conducted a more detailed investigation and a case–control study. A case was defined as a child under 15 years of age diagnosed with acute leukemia between 1977 and 1990 who had spent at least 25% of his or her lifetime in the area before diagnosis. Fourteen cases met this definition, of which twelve were permanent residents and two had spent 2 to 3 days a week in the area. Based on the state's cancer registry, the number of cases to be expected was about one every 2 years or about seven in 14 years. Seven of the cases occurred during the 3 years 1982 to 1984. After 1985, case incidence returned to expected levels—one case every 2 years (*40*).

Among the seven cases identified from 1982 to 1984, five were acute nonlymphocytic leukemia (ANLL), whereas statewide, three of four cases were acute lymphocytic leukemia (ALL). Six of seven cases were girls; childhood leukemia appears to be

somewhat more common in boys. Four of the girls were between 9 and 12 years of age, whereas the peak onset for childhood leukemia is around 3 years of age.

In the case–control study of 14 cases and 56 matched controls of the same sex born within 6 months of the cases studied, no statistically significant risk factors were defined. There were, however, elevated OR for other cases of cancer in the family (OR = 3.4 with 95% CI of 0.70–16.41) and for having ever resided within 2.2 miles of the Lualualei Naval Broadcast Facility and its two low frequency radio towers (OR 2.2; 95% CI of 0.65–7.56).

The authors suggest that improper storage of oil may have been associated with risk of benzene exposure, a known adult leukemogen. No adequate environmental measurements were available for radiation or benzene exposure. Some measurements of electric or magnetic fields were made by the U.S. EPA in 1990, but the measurements were made primarily along roads and not in areas where children lived and played. Nine of the fourteen cases were of Hawaiian or part-Hawaiian ethnic origin, and there is some evidence that Hawaiians and Maoris of New Zealand have lower rates of ALL and higher rates of ANLL than other ethnic groups.

The authors concluded that "...closeness to the low frequency radio towers at Lualualei Naval Station may have a weak association with leukemia, even though it is not statistically significant. This cannot be considered proof that anything emanating from the station actually caused the leukemia" (*40*).

North Sydney Study. Hocking et al. (*11*) reported on cancer incidence and mortality in the proximity of television (TV) towers; cancer incidence and mortality for the 1972 to 1990 period for nine municipalities in North Sydney, Australia, were collected. Three municipalities were closer to the TV broadcasting facilities than the other six, and hence, exposed to more RF radiation. The calculated power density in the more exposed areas ranged from 8 to 0.2 $\mu W/cm^2$ at a 4-km radius. At a distance of 12 km, power density was 0.02 $\mu W/cm^2$. They found that for all ages, there was little difference in incidence of brain cancer. For leukemia, however, the incidence rate ratio for adults was 1.24 (95% CI 1.09–1.40), whereas for children it was 1.58 (95% CI 1.07–2.34), with a mortality rate ratio of 2.32 (95% CI 1.35–4.01). The authors were unsuccessful in identifying confounders to explain these results.

The signals emitted by the TV towers were 100 kW video amplitude modulated and 10 kW audio frequency modulated on carrier frequencies from 63 to 215 MHz. The authors had no prior knowledge of a possible cluster of leukemia cases near the towers.

United Kingdom Studies. Dolk et al. (*12*) reported on leukemia incidence near the Sutton Coldfield radio and TV transmitters for the years 1974 to 1986. In addition, they studied adult leukemia incidence near 20 high power TV/frequency modulation (FM) transmitters in Great Britain (*13*). The Sutton Coldfield study examined data within a 10-km radius in 10 bands of increasing distance. The innermost area was within 2 km of the transmitter; adult leukemia relative risk (RR) was 1.83 (95% CI 1.22–2.74). Actually, one case lived within 0.5 km when 0.11 km could have been expected on the basis of cancer registry experience and the numbers of person-years of observation. While this results in an RR of 9, emphasizing location of a single case is likely to represent a poorly defined range of risk (Figure 1). There was a significant decline in risk with increased distance ($p < 0.001$) from the transmitters. Expected numbers of leukemias in the 10-km zones near transmitters were calculated on the basis of national rates stratified by 5-year age groups, socioeconomic deprivation quintile, and region.

In a second Dolk et al. (*13*) study the same procedures were used to evaluate risks surrounding 20 other broadcast facilities in the UK for the same period; 3305 cases were identified, with an overall observed-to-expected (O/E) ratio of 1.03 (95% CI 1.0–1.07). Decline in risk with distance was significant for all sites combined. Results in this study were similar to those of the Sutton Coldfield study (*12*). There was no significant excess risk for persons living within 2 km of the transmitters and excess risk was not greater than 15% in any distance band up to 10 km. However, the decline in risk for adult leukemia with distance from the transmitters was significant ($p < 0.05$). Eight of the transmitters broadcast FM and three TV at power equivalent to transmission in the Sutton Coldfield study (*12*). One of the transmitters, Crystal Palace, was located in an unusually densely populated area and appeared to be associated with almost half the cases of leukemia. In the band between 2 and 3 km from the transmitter the adult leukemia O/E ratio was 1.33. Figure 1 shows some of these gradients for the Sutton Coldfield transmitters and for two other groups of stations, one with greater power than the other.

Rothman et al. (*9*) tabulated studies that might relate leukemia to occupational or recreational exposures to RF radiation and studies that related such exposures to brain malignancies. The risk ratios for leukemia were > 1.0 for 19 studies and ≤ 1 for 7. For brain tumors the RR was > 1 for 9 studies, and ≤ 1 for 4.

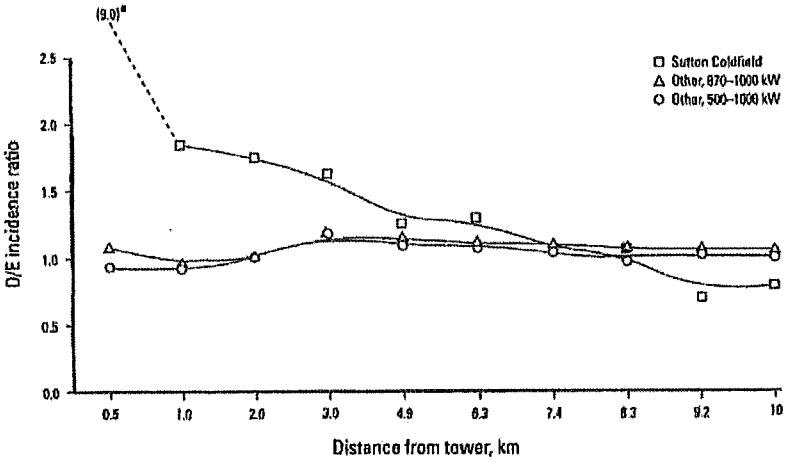

Figure 1. The O/E leukemia incidence ratio by distance from TV and FM broadcast towers. The trends are shown for Sutton Coldfield and for two subsets of other such facilities in the United Kingdom for adults more than 15 years of age for the years 1974 to 1986. One subset is for facilities broadcasting TV in the range of 870 to 1000 kW and the second includes those with power from 500 to 1000 kW. *The O/E incidence ratio of 9.0 is based on a single case. Based on Dolk et al. (*12,13*).

Grayson (*10*) reported on brain cancer among U.S. Air Force personnel, and found that rank (socioeconomic factor) was the most important predictor. When this was taken into account, nonionizing radiation exposure was more important than ionizing radiation and microwave exposures more significant that low frequency exposures. The positive association for military rank had an OR of 3.30 (95% CI 1.99–5.45) for senior officers. For ionizing radiation, the association is negative. The military-rank-adjusted OR is significantly elevated for RF: 1.38 (95% CI 1.01–1.90), but not for ELF: 1.28 (CI 0.95–1.74).

Another study of military personnel and radiation exposures was that by Szmigielski (*41*), who examined cancer by site among Polish military personnel during the period 1970 to 1989. He found a relationship between exposures to high frequency (RF radiation) and cancer morbidity. About 3700 of the approximately 128,000 personnel were classified as exposed and data were tabulated for 12 types of cancer and four age groups. The overall cancer morbidity for exposed personnel was 119.1/100,000 per year compared on an age-adjusted basis to 57.6 in the nonexposed group. The greatest O/E ratios were found for chronic myelocytic leukemia, 13.9; myeloblastic leukemia, 8.62; and non-Hodgkin's lymphoma, 5.82.

A cluster of six cases of testicular cancer among traffic policemen using microwave generators suggests that microwave exposures can cause cancer of the testicle (*42*). Other epidemiologic studies of exposed military personnel point in the same direction (*43,44*).

Lin et al. (*45*) collected data on brain cancer deaths among white males for the state of Maryland, and examined occupations stated on the death certificates Included were 951 brain tumors, of which 370 were glioblastomas, 149 astrocytomas, and 432 had unspecified histology.

Fifty glioma and astrocytoma deaths among workers in occupations with a high probability of electrical exposures were matched by age with a sample of the population by age from the 1979 census. The expected number of such occupations in the general population was about one-third of that observed (18/50) for cases. A case reference study showed that the occupational category of electric or electronic engineer and technician had three times the number of cancer cases as the referent population (18 vs 6; $p < 0.05$). When the specified occupations were ranked by definite, probable, or possible exposures to electromagnetic fields, the OR for astrocytoma and glioma were 2.15, 1.95, and 1.44, respectively.

Garland et al. (*46*) studied leukemia among occupational groups with potential electromagnetic field exposure in the U.S. Navy. Because they studied personnel who were hospitalized while on active duty, the study cannot include personnel with leukemia of substantial latency or those who were not hospitalized. In fact, one occupational group, electrician's mate(s), showed consistent excess of risk for leukemia.

Follow-up Study of 40,000 Korean War Naval Personnel. In the Robinette et al. (*47*) study, naval personnel were divided into occupational groups with low and high exposures by the occupational designator for the personnel. Within these two catagories were three occupational classes, shown in Table 5.

Table 5 shows the occupational groups and numbers of cases. Table 6 gives rates for all deaths (per 1000) during the follow-up period of 1950 to 1974: rates for deaths attributable to disease, malignant disease, and malignancy of the lymphatic and hematopoietic systems. Death rates for the group with the highest exposure, aviation electronics technician(s) (AT), are significantly higher than those for the remaining men for all deaths, disease-related deaths, deaths from malignancy, and deaths from malignancy of the lymphatic and hematopoietic systems. Although it was true that this group had a higher mean age at onset of the follow-up study (23.4 years) than the average of the whole group (21.3) this mean age was younger than the average for aviation electrician's mate(s) AE (24.7), a category that showed no increase in deaths from any malignancy or from other diseases. The authors adjusted for age, but in doing so combined the AT group with the fire control technician(s) (FT) group, which had a low malignancy rate. These two groups, which were about the same size, had 10 and 1 cases of lymphopoeitic or hematological malignancies, respectively. For this population, compensated disability by body system is shown in Table 7 for the two high-exposure groups compared to the remainder of the population Both numbers and crude rates are given as well as the expected number of cases for the more exposed group based on the data for the remainder.

Additional Studies of Cancer in Children and Others. Among the many tabulations from the Lilienfeld report (*18*), those for data about leukemia are shown in Table 8, based on data excerpted from the Lilienfeld report by Goldsmith (*8*). Although the numbers are small, there is significant excess for child dependents in both Moscow and other embassies, as well as an excess for employees and dependents in both locations. Estimated exposures at the Moscow embassy were from 5 to 18 $\mu W/cm^2$.

Table 5. U.S. Naval personnel by occupational category during the Korean War and deaths by cause group, 1950 to 1974.

	Low exposure			High exposure		
	RM	RD	AE	ET	FT	AT
Number of persons	9253	10,116	1412	13,078	3298	3733
Total deaths	296	308	61	441	144	198
From disease	161	165	22	199	81	77
From malignant disease	39	47	8	65	16	27
From malignancy of the lymphatic and hematopoietic systems	6	14	0	18	1	10

Abbreviations: ET, electronics technician(s); RD, radioman; RM, radarman. Data based on Robinette et al. (*47*).

Table 6. U.S. Naval personnel by occupational category during the Korean War and crude death rates per 1000 by cause group, 1950 to 1974.

	Low exposure			High exposure		
	RM	RD	AE	ET	FT	AT
Number of persons	9253	10,116	1412	13,078	3298	3733
Total death rates	32.0	30.4	43.2	33.7	43.7	53.0
From disease	17.4	16.3	15.6	15.2	24.6*	20.62*
From malignant disease	4.21	4.65	5.66	4.97	4.85	7.23*
From malignancy of the lymphatic and hematopoietic systems	0.65	1.38	0.00	1.38	0.3	2.68*

*Significantly increased, $p < 0.05$ compared to less-exposed groups. Data based on Robinette et al. (*47*). For occupational class definitions see Table 5 and text.

Table 7. Number of U.S. Naval personnel receiving Veterans Administration compensation in 1976, by diagnostic group, for two high-exposure groups (FT and AT) relative to the low-exposure groups exposed during the Korean War.

Diagnostic group	FT and AT		All others		FT and AT, expected no
	No	Rate/1000	No	Rate/1000	
Musculoskeletal	119*	16.9	403	11.90	83.7
Special sense organs	42	6.0	152	4.49	31.6
Systematic conditions	5*	0.7	7	0.20	1.45
Respiratory	51*	7.3	171	5.05	35.5
Cardiovascular	47*	0.7	142	4.19	29.5
Digestive	55	7.8	229	6.76	47.6
Genitourinary	19	2.7	99	2.92	20.6
Hemic, lymphatic	3	0.4	10	0.30	2.08
Skin	58	8.2	227	6.70	47.1
Endocrine	11	1.6	46	1.36	9.55
Neurologic	16	2.3	54	1.60	11.2
Nerves	3	0.4	41	1.21	8.5
Epilepsies	0	–	16	0.47	3.32
Mental conditions	46	6.5	198	5.85	41.1
Other	2	0.3	19	0.56	3.95
Total diagnoses	477**	67.84	–	53.61	376.94
Total populations	7031		33,059		

*Significantly increased, $p<0.05$; **significant, $p<0.01$. Data based on Robinette et al. (*44*).

Table 8. Leukemia among U.S. embassy employees and child dependents in Moscow and other Eastern European embassies.

Population	Moscow embassy		Other embassies		Total O/E
	Observed	Expected	Observed	Expected	
Employees	2	0.8	3	1.7	5/2.5
Child dependents	2	0.5*	3	0.7*	5/1.2*
Total	4	1.3*	6	2.4*	10/3.7*

*Significantly elevated O/E ratio, $p<0.05$. Based on table in Goldsmith (*8*).

Evidence of Other Health Effects

Lenticular Changes. Toncheva et al. (*48*) studied 87 persons working with radar and 150 eye-matched controls. The radar workers were divided into five risk groups according to frequencies of microwave exposure (200 KHz to 26 GHz) and power density (8 μW to 300 mW/cm^2).

They found three specific radiation cataracts in persons working with extremely high microwave exposure. Lens changes were associated with level of exposure in different risk groups. Changes such as opacities and posterior polar defects are criteria for microwave exposure.

Nonspecific Disability. In their study of Korean War Veterans, Robinette et al. (*47*) obtained data for disability by body system in 1976. As noted in a previous analysis (*8*), the AT workers, those presumed to have received the most radiation exposures, were combined with the lesser-exposed FT to make what was designated the high-exposure group.

In the ten categories in Table 7 (categories with five or fewer cancer cases are not included) the high (FT+AT) group is higher than the remaining groups, with lower exposures in nine of ten body systems (significant by sign test at $p<0.05$).

The overall disability rate of 67.8/1000 is significantly greater than 43.1 by Poisson criteria. As is apparent from the combination of the two highest exposure job categories and the nature of the job classification procedure as described by the investigators, this analysis probably underestimates the effects of exposure.

Nonspecific Neurological and Sensitivity Reactions. Silverman (*49*) noted some nonspecific reactions to RF radiation, and a more recent review (*50*) brings these findings up to date. More research is needed to better define these reactions.

Interpretations

Available data suggest that RF radiation be considered a carcinogenic risk, a position already taken in an internal U.S. EPA document (*51*) in 1990 when there was much less evidence of the potential harmfulness of RF radiation.

Except for the Moscow staff, which includes both workers and dependents, most of the exposures studied are relevant to occupation. The most relevant to cases of community exposure risks today are those involving populations living near broadcast facilities. Cellular telephone users have not been exposed in definable numbers for a long enough time period for an adequate study to be made of cancer incidence.

However, interpretations must take into account the report of the Repacholi et al. study (*52*) of lymphoma-prone mice, who showed a doubling of the incidence of lymphoma over an 18-month period when exposed to modulated radiation similar to far-field cellular telephone exposures. This initial finding of experimental evidence of cancer from cellular-telephonelike exposures emphasizes the importance of examining epidemiologic evidence of such effects. Possibly the most suitable source for such data would be the more detailed study of exposures of military personnel or air traffic controllers who received definable exposures and have undergone a sufficient period of follow-up. Evaluation of such nonspecific symtoms as headache, sleep disturbances, and unfavorable reproductive outcomes of populations living near broadcast facilities should have priority for community studies.

The evidence may or may not justify more restrictive regulation of occupational exposure; for community exposures, however, the evidence justifies prudent avoidance (*14,15*). The concept has been presented by a group of Swedish government agencies in response to the evidence concerning ELF exposures. The plan is basically voluntary and stresses education about risks and economic analysis of uncertain risks and the possible costs of their avoidance.

Included among the actions to take under the rubric of prudent avoidance is epidemiologic monitoring (*53*), a system of standardized health status measurements of presumably reversible effects, which can, if unfavorable trends are discerned, become the basis for higher levels of population protection. The availability of a number of potentially reversible biologic responses makes this an unusually attractive possibility.

A second type of action is to provide realistic procedures to minimize the exposures. Shielding the head and face from exposures to the antennae of hand-held cellular telephones, and guidelines for keeping an adequate distance between broadcasting sources and civilian populations, are clearly indicated.

Further work is needed on the possibility of carcinogenicity in experimental systems of RF exposures. These systems should be separate from evaluations of

ELF, which does not appear to have the same set of effects.

This review casts some doubt on efforts to distinguish ionizing from nonionizing radiation with respect to their health effects. It also raises doubt about the protective role of regulations based solely on the thermal effects of RF radiation, which is the basis for current standards.

There seems to be some evidence from the Moscow study and community studies in the vicinity of large FM and TV broadcasting facilities that exposures as low as 2 $\mu W/cm^2$ may have long-term health effects.

A comprehensive and critical review of the epidemiologic data available on health risks from RF exposure should be carried out and the reasonable measures for avoidance of the identified risks should be described and evaluated.

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EXHIBIT H-4

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Electrohypersensitivity: a functional impairment due to an inaccessible environment

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Abstract: In Sweden, electrohypersensitivity is recognized as a functional impairment which implies only the environment as the culprit. The Swedish view provides persons with this impairment a maximal legal protection, it gives them the right to get accessibility measures for free, as well as governmental subsidies and municipality economic support, and to provide them with special Ombudsmen (at the municipality, the EU, and the UN level, respectively), the right and economic means to form disability organizations and allow these to be part of national and international counterparts, all with the simple and single aim to allow persons with the functional impairment electrohypersensitivity to live an equal life in a society based on equality. They are not seen as patients, the do not have an overriding medical diagnosis, but the 'patient' is only the inferior and potentially toxic environment. This does not mean that a subjective symptom of a functionally impaired can not be treated by a physician, as well as get sick-leave from their workplace as well as economic compensation, and already in the year 2000 such symptoms were identified in the Internal Code of Diagnoses, version 10 (ICD-10; R68.8/now W90), and have been since. But the underlying cause still remains only the environment.

Keywords: electrohypersensitivity; functional impairment; immunohistochemistry; skin; UN Convention.

Background

A functional impairment is defined as difficulties that substantially interfere with or limit functioning in one or more major life activities including the following:

- Basic daily living skills (e.g. eating, bathing, dressing);
- Instrumental living skills (e.g. maintaining a household, managing money, getting around the community, taking prescribed medication); and
- Functioning in social, family, and vocational/educational contexts.

In health, any loss or abnormality of physiological, mental, or anatomical structure or function, whether permanent or temporary, is regarded as a functional impairment. The existence of a medical condition does not necessarily restrict functional capacity, but may form part of the underlying cause for a functional impairment. However, much more common are obstacles in our surrounding environment resulting in everyday functional impairments that all are part of being a human being in a society. Humans, and particularly children, often run into situations where the environment provides hurdles and difficulties. Such can be language barriers, educational hinders, physical, chemical or physiological blocks or toxicities, and problems of understanding. Neither one of these causes makes anyone a patient; we are all still normal healthy persons as well as citizens, and with correct avoidance or adaptive reactions to an inferior environment.

The Swedish view

Let me now conclude the following: The Swedish approach to electrohypersensitivity is to view it as a functional impairment, thus focusing on the environment as the culprit (which, as of the above, is the general definition, including the UN one, of functional impairments). This provides persons with this impairment a maximal legal protection, it gives them the right to get accessibility measures for free, as well as governmental subsidies and municipality economic support, and to provide them with special Ombudsmen (at the municipality, the EU, and the UN level, respectively), the right and economic means to form disability organizations and allow these to be part of national and international counterparts, all with the simple and single aim to allow persons with the functional impairment electrohypersensitivity to live an equal life in

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a society based on equality. They are not seen as patients, the do not have an overriding medical diagnosis, but the 'patient' is only the environment – inferior and potentially toxic. This does not mean that a subjective symptom of a functionally impaired can not be treated by a physician, as well as get sick-leave from their workplace as well as economic compensation, and already in the year 2000 such symptoms were identified in the Internal Code of Diagnoses, version 10 (ICD-10; R68.8/now W90), and have been since. But the underlying cause still remains only the environment.

Thus, 'functional impairment' and 'medical condition' are not mutually exclusive. They are different things. In the everyday world, having electrohypersensitivity (EHS) is definitely a huge disability and it impairs the ability of a person to have an optimal quality of life. If it was not an impairment, only those interested in esoterica would care about it. So, the pragmatic aspect that makes it important is the simple fact that it is an impairment that disables people's lives. (And interventions for people who have the condition include medical treatments of their symptoms, but not of their environmental causes – there we need technicians, physicists, electricians, and others to make the latter accessible.) About this, the current whole society needs to be educated. Furthermore, prevention is yet another aspect that definitely should be in the discussion; when will we see my dream of tomorrow's green, human-friendly technology come into play?

As stated, in Sweden, electrohypersensitivity (EHS) is an officially fully recognized functional impairment [i.e. it is not regarded as a disease; N.B. this is not special for Sweden, the terms "functional impairment" and "disease" are defined according to various international documents (see below)]. Survey studies (1) show that somewhere between 230,000–290,000 Swedish men and women – out of a population of 9,000,000 – report a variety of symptoms, including typical cutaneous ones such as stinging, burning and itching sensations primarily in the face, upper chest and back, hands and arms, when being in contact with electromagnetic field (EMF) sources. The symptoms of EHS are classified as an occupationally-related symptom-based diagnosis (code ICD-10; R68.8/now W90) by the Nordic Council of Ministers since 2000 (2). Swedish electrohypersensitive people have their own handicap organization, The Swedish Association for the Electrohypersensitive, which has its own websites in both Swedish and English (3). This organization is included in the Swedish Disability Federation (Handikappförbundens SamarbetsOrgan; HSO; as a consequence of this, The Swedish Association for the Electrohypersensitive receives an annual governmental subsidy). HSO is the united voice of the Swedish disability associations towards the government, the parliament, and national authorities, and is a cooperative body that today consists of 43 national disability organizations (with The Swedish Association for the Electrohypersensitive being 1 of these 43 organizations) comprised of about 500,000 individual members. It has its own website in Swedish, parts of which are also in English (4).

Swedish municipalities, of course, have to follow the UN 22 "Standard Rules on the equalisation of opportunities for people with disabilities" ("Standardregler för att tillförsäkra människor med funktionsnedsättning delaktighet och jämlikhet" (5). Since 2007 they have been upgraded into the UN "Convention on Human Rights for Persons with Functional Impairments") (5). As a result of this, all people with disabilities shall be given the assistance and service they have the right to according to the Swedish Act concerning Support and Service for Persons with Certain Functional Impairments ("LSS-lagen") and the Swedish Social Services Act ("Socialtjänstlagen"). The municipalities are responsible for making sure that everyone gets enough support. Everyone is required to remember that such men and women may need individual and different kinds of support. What works for one, may not at all work for another.

In Sweden, impairments are viewed from the point of the environment. No human being is in itself impaired, there are instead shortcomings in the environment that cause the impairment (as with the lack of ramps for the person in a wheelchair, or rooms requiring electrosanitisation for the person with EHS). This environment-related impairment view, furthermore, means that even though one does not have a complete scientifically based explanation for the impairment EHS, and in contrast to what many individuals involved in EMF discourse at present, the person with EHS shall always be met in a respectful way and with all necessary support with the goal to eliminate the impairment. This implies that the person with EHS shall have the opportunity to live and work in an electrosanitised environment.

This view can fully be motivated in relation to the present national and international disability laws and regulations, including the UN 22 Standard Rules/UN Convention, the Human Rights Act of the EU, and the Swedish Action Plan for Persons with Impairments ("Den nationella handlingplanen för handikappolitiken – Från patient till medborgare") (6). The last part of this national Action Plan carriers a very important sentence: "Från patient till medborgare" = "From patient to citizen" which stresses the modern view of the functionally impaired: they are not any longer seen as patients but citizens

reacting to an inferior environment with a completely relevant avoidance behavior (cf. below). For such avoidance reactions there are no demands for any official "recognition", no demands for diagnoses, and – of course not – no medical/psychiatric treatments.

The first step for a person in Sweden with a functional impairment is to contact the municipality's special civil servant for disability issues, as well as the various handicap organizations and authorities, to achieve accessibility measures of various types with the sole aim to have an equal life in a society based on equality (as being in accordance to the The UN 22 Standard Rules on the Equalization of Opportunities for People with Disabilities/The UN Convention on Human Rights for Persons with Functional Impairments).

An impairment is – by definition – not defined by someone else or proven by certain tests, etc. The impairment is always personal (private) and develops when in contact with an inferior environment. [N.B. Remember that functional impairments are only based upon each individual's impaired accessibility to – and contact with – an inferior environment (cf. the UN), thus, there is actually no need for any "recognition" in local laws (cf. the UN). In Sweden, the former Minister of Health and Social Affairs, Lars Engqvist – as a member of one of the previous governments – anyhow gave his "approval" in a letter dated May, 2000 [Regeringskansliet 2000-04-06, Dnr S2000/2158/ST]. He also made it clear in his response that for EHS persons there are no restrictions or exceptions in the handicap laws and regulations. Thus, these laws and regulations are to be fully applied also for EHS persons.]

Remember we all must adhere to and follow all the handicap laws and regulations. To do the opposite is a serious violation and should immediately be reported/filed as an official legal complaint to your local authorities, parliament, government, the EU and the UN. This is of particular importance since Katri Linna, the former Swedish Diskrimineringsombudsman (=the Equality Ombudsman), clearly states in the newspaper Sydsvenskan (7) that "electrohypersensitivity is – according to the law – a functional impairment and I recommend EHS persons that are discriminated to file a complaint"

The history of electrohypersensitivity

Historically, there have been – since the introduction of electricity as a general power source – anecdotal stories about persons whom we today interpret as being actually electrohypersensitive. The very first case may have been Nikola Tesla (10 July 1856–7 January 1943) a Serbian-American inventor, electrical engineer, mechanical engineer, physicist, and futurist, best known for his contributions to the design of the modern alternating current (AC) electricity supply system. Descriptions of his health status closely resembles what we today would have named electrohypersensitivity. A surge of similar case reports were also seen during the amateur radio (DX) years.

In more recent times, as early as in the 1970s, a report from the former Soviet Union described a "microwave syndrome". The Soviet military recognized early on the possible side-effects from radar and radio radiation. This microwave syndrome was seen in up to a quarter of the military personnel working with radio and radar equipment. They showed symptoms such as fatigue, dizziness, headaches, problems with concentration and memory, sleep disturbances, and being hot tempered. The treatment suggested was a change of assignments and to keep away from exposure. Rest, physical exercise, and nutritious food were also offered (8).

Also in the 1970s the newspaper industry was one of the first to supply it's employees with personal computers using visual display terminals. Complaints of headaches and visual problems, as well as clusters of miscarriages and birth defects in children born to female editors and other newspaper employees, generated some publicity. In addition, many people who worked in the electronics industry in Sweden, including an estimated 12% of the electrical engineers in that industry, became electrically sensitive, and helped form the current Swedish disability organization initially called Föreningen för el-och bildskärmsskadade (Association for the Electrosensitive), or FEB. This was later renamed Elöverkänsligas Riksförbund (The Swedish Association for the Electrohypersensitive).

In the United States, then-Representative Al Gore held Congressional hearings in 1981 on the health effects of computer screens. In Sweden, the hard work of many brilliant trade union officials, members and their affiliates, such as Bruno Hagi, Per-Erik Boivie, Martin Andersson, Jan Åberg, Torbjörn Klittervall, Åke Bergman, and of excellent journalists, especially Gunni Nordström and Carl von Schéele, brought the problem to the attention of the general public during the 1980s and 1990s. Nordström's and Schéele's books "Sjuk av bildskärm" (9) and "Fälslaget om de elöverkänsliga" (10) are regarded as classics.

During the 1990s the Swedish trade union movement, notably the Swedish Confederation of Professional Employees (TCO) and Union of Clerical and Technical Employees in Industry (Sif), led the world in actively tackling, and working on solutions for the growing problem of

both chemical and electromagnetic pollution in the workplace – and spreading the word internationally. Among very many ventures, Sif's "No-Risk Project", enlisting the involvement of a team of researchers, including Martin Andersson from LIBEREL AB, and myself from the Karolinska Institute, as well as the "Healthy Office Project" in partnership with the Luleå University of Technology (LTU), are both highly important milestones. At that time, Sif was the largest trade union for white-collar workers in Sweden until it merged in January 2008 with another trade union (HTF) to form a new organization, Unionen. Unionen is currently Sweden's largest trade union on the private labour market and one of the largest white-collar unions in the world.

The TCO label is known internationally for its precautionary environmental standards for computer monitors that place limits on both electromagnetic radiation (EMR) and chemical emissions (TCO'92, TCO'95, TCO'99, TCO'03, and soon TCO'15). Although not eliminating EMR and chemical emissions from computer monitors, it was ground-breaking in that it was the first case of a union organization, representing both members and consumers, successfully influencing manufacturers to improve the design of their products to reduce potentially harmful emissions. Their corresponding standards for e.g. smartphones, tablets, and headsets, are currently gaining more and more momentum.

Today the most famous electrohypersensitive person is Gro Harlem Brundtland (20 April 1939), the former Prime Minister of Norway and the former Director General of the UN World Health Organization (WHO).

Cutaneous analyses

One of the very first to analyze cutaneous biopsies of electrically sensitive individuals, was the late assistant professor and histopathologist Björn Lagerholm at the Karolinska Hospital, Department of Dermatology, in Stockholm, who did these ground-breaking observations already in the middle of the 1980s. He found an increase in the mast cell number, but, unfortunately, he could never publish it in a peer review-based scientific journal.

His interest very much started with a female bank employee that had received a work injury compensation for skin changes after sitting in front of a visual display monitor. Björn Lagerholm described in great detail her skin changes, which turned out to be very similar to the kind of cutaneous alterations you may encounter in connection with ultraviolet light, X-ray or radioactivity damage. Björn Lagerholm's reputation as a histopathologist was undisputed. In addition to the female bank employee, he also examined nearly 100 further similar cases, all having the same skin changes. The outcome of this analysis was, however, not supported by two other histopathologists, and not by his clinical colleagues, Dr. Mats Berg and professor Sture Lidén.

Björn Lagerholm was never able to pursue his ground-breaking and very elegant studies. They would be burried for several years, until I and my collaborators re-initiated them in the late 1980s and early 1990s.

For me it was immediately clear that persons claiming skin reactions after having been exposed to computer screens very well could be reacting in a highly specific way and with a completely correct avoidance behavior reaction to a toxic environment/world, especially if the provocative agent was radiation and/or chemical emissions, such as plastic components or flame retardants – something later focussed upon by professor Denis L. Henshaw and his collaborators at Bristol University [this is covered in Gunni Nordström's book "Mörkläggning – Elektronikens rättslösa offer" (11)] — just as you would do if you had been exposed to e.g. sun rays, X-rays, radioactivity or chemical odours.

I began to study the skin of electrohypersensitive persons, and provided evidence that they may have a real skin condition that is provoked by sitting in front of a computer screen, using a mobile phone, a tablet, or being close to smart meters, routers, telecom towers and power installations. The damage was similar to that caused by ultraviolet radiation from the sun. I also showed that the radiation from computers causes measurable changes even in the skin of "normal people," and also in the skin of laboratory animals.

I named the new syndrome "screen dermatitis" in Experimental Dermatology, 1994 (12), and "electrosupersensitivity" at the Workshop on Electromagnetic Hypersensitivity, EU/EC, in Graz, 1995 (13). However, since such individuals also usually complained of other symptoms, such as chest pain, memory loss, fatigue, insomnia, dizziness, nausea, and headache, the more general term "electromagnetic hypersensitivity" later came into use, and it is nowadays shortened to "electrohypersensitivity".

In Sweden the prevalence of electrohypersensitivity was first estimated at 1.5% (14) with a newer estimate of 2.6%–3.2% (15). In Austria the prevalence was estimated to be <2% in 1994 but it had increased to 3.5% in 2001 (16). In Switzerland 5% of the population has been estimated to suffer from EHS (17). In California the prevalence of self-reported sensitivity to electromagnetic fields was 3.2% and with 24.4% of those surveyed reporting sensitivity to

chemicals as well (18). The Canadian Human Rights Commission reported that approximately 3% of Canadians have been diagnosed with environmental sensitivities, including chemicals and EMFs in their environment (19). In the report the author especially recommended improving the environmental quality at work places. Finally, a prevalence as high as 13.3% has been reported in Taiwan (20).

One aim of my and my collaborators studies has been to investigate the presence of intraepidermal nerve fibers in normal human skin from healthy volunteers using the new marker PGP 9.5 (21–23). The intraepidermal nerve fibers are found as close as 20–40 μm from the surface, which makes it highly possible that surrounding electromagnetic fields may affect them.

In facial skin samples of electrohypersensitive persons, the most common finding is a profound increase of mast cells. We have not only used histamine, but also other mast cell markers such as chymase and tryptase, but the pattern is still the same as reported previously for other electrohypersensitive persons (13). From these studies, it is clear that the number of mast cells in the upper dermis is increased in the EHS group. A different pattern of mast cell distribution also occurred in the EHS group, namely, the normally empty zone between the dermo-epidermal junction and mid-to-upper dermis disappeared in the EHS group and, instead, this zone had a high density of mast cell infiltration. These cells also seemed to have a tendency to migrate towards the epidermis (=epidermiotrophism) and many of them emptied their granular content (=degranulation) in the dermal papillary layer. Furthermore, more degranulated mast cells could be seen in the dermal reticular layer in the EHS group, especially in those cases which had the mast cell epidermiotrophism phenomenon described above. Finally, in the EHS group, the cytoplasmic granules were more densely distributed and more strongly stained than in the control group, and, generally, the size of the infiltrating mast cells was found to be larger in the EHS group as well. It should be noted that increases of similar nature later on were demonstrated in an experimental situation employing normal healthy volunteers in front of cathode ray tube (CRT) monitors, including ordinary household television sets and personal computer screens (24).

In one of the early papers (12), we made a sensational finding when we exposed two electrohypersensitive individuals to a TV monitor situated at a distance of 40–50 cm away from them. When we looked at their skin under a microscope, we found something that surprised us. In this article, we used an open-field provocation, in front of an ordinary TV set, of persons regarding themselves as suffering from skin problems due to work at video display terminals. Employing immunohistochemistry, in combination with a wide range of antisera directed towards cellular and neurochemical markers, we were able to show a high-to-very high number of somatostatin-immunoreactive dendritic cells as well as histamine-positive mast cells in skin biopsies from the anterior neck taken before the start of the provocation. At the end of the provocation the number of mast cells was unchanged, however, the somatostatin-positive cells had seemingly disappeared. The reason for this latter finding was discussed in terms of loss of immunoreactivity, increase of breakdown, etc., but it may simply indicate a common and well-known reaction to radiation, namely the migration of the classical allergen-recognizing dendritic cells from the skin to more deeply located immune-competent organs such as lymph nodes and the spleen. The high number of mast cells present may explain the clinical symptoms of itch, pain, edema, and erythema, and such a high number is again a classical sign of a radiation damage.

We have compared facial skin from electrohypersensitive individuals with corresponding material from normal healthy volunteers (25). The aim of that particular study was to evaluate possible markers to be used for future double-blind or blind provocation investigations. Differences were found for the biological markers calcitonin gene-related peptide (CGRP), somatostatin (SOM), vasoactive intestinal polypeptide (VIP), peptide histidine isoleucine amide (PHI), neuropeptide tyrosine (NPY), protein S-100 (S-100), neuron-specific enolase (NSE), protein gene product (PGP) 9.5, and phenylethanolamine N-methyltransferase (PNMT). The overall impression in the blind-coded material was such that it turned out easy to blindly separate the two groups from each other. However, no single marker was 100% able to pin-point the difference, although some were quite powerful in doing so (CGRP, SOM, S-100). In parallel investigations, we also found alterations of the Merkel cell number in the facial skin of electrohypersensitive persons (unpublished research). However, it has to be pointed out that we can not, based upon those results, draw any definitive conclusions about the cause of the changes observed. Blind or double-blind provocations in a controlled environment (26) are necessary to elucidate the underlying causes for the changes reported in this particular investigation.

I and my collaborator, Dr. Shabnam Gangi, in two papers of theoretical nature (27, 28), have put forward a model for how mast cells and substances secreted from them (e.g. histamine, heparin, and serotonin) could explain sensitivity to electromagnetic fields. The model bounces off from known facts in the fields of UV- and

ionizing irradiation-related damages, and used all the new papers dealing with alterations seen after, e.g. power-frequent or microwave electromagnetic fields, to propose a simple summarizing model for how we can understand the phenomenon of the functional impairment electrohypersensitivity.

In the first paper (27), we describe the fact that an increasing number of persons say that they get cutaneous problems as well as symptoms from certain internal organs, such as the central nervous system and the heart, when being close to electric equipment. A major group of these persons are the users of video display terminals (computers, tablets, mobile phones, etc.), who claim to have subjective and objective skin- and mucosa-related symptoms, such as pain, itch, heat sensation, erythema, papules, and pustules. The central nervous system-derived symptoms are, e.g. dizziness, tiredness, and headache. Erythema, itch, heat sensation, edema, and pain are also common symptoms of sunburn (UV dermatitis). Alterations have been observed in cell populations of the skin of electrohypersensitive persons similar to those observed in the skin damaged due to ultraviolet light or ionizing radiation. In electrohypersensitive persons a much higher number of mast cells has been observed. It is known that UVB irradiation induces mast cell degranulation and release of TNF-α. The high number of mast cells present in the electrohypersensitivity group and the possible release of specific substances, such as histamine, may explain their clinical symptoms of itch, pain, edema, and erythema. The most remarkable change among cutaneous cells, after exposure with the above-mentioned irradiation sources (ultraviolet light, X-rays, ionizing radiation), is the disappearance of the Langerhans' cells (cf. above). This change has also been observed in electrohypersensitive persons, again pointing to a common cellular and molecular basis.

In the second publication (28), the relationship between exposure to electromagnetic fields and human health is even more in focus as are the cutaneous mast cells. Mast cells, when activated, release a spectrum of mediators, among them histamine, which is involved in a variety of biological effects with clinical relevance, e.g. allergic hypersensitivity, itch, edema, local erythema, and many types of dermatoses. From the results of our and others studies, it is clear that electromagnetic fields affect the mast cell, and also the dendritic cell, population, and may degranulate these cells. The release of inflammatory substances, such as histamine, from mast cells in the skin results in a local erythema, edema, and sensation of itch and pain, and the release of somatostatin from the dendritic cells may give rise to subjective sensations of ongoing inflammation and sensitivity to ordinary light.

Against this background, it is very interesting to see our early findings from the 1980s and 1990s strongly supported by the work of Drs. Belpomme and Irigaray. According to them, both EHS and multiple chemical sensitivity (MCS) appear to paint a common picture of inflammation-related hyper-histaminemia, oxidative stress, autoimmune response and BBB opening, and a deficit in melatonin excretion. According to Belpomme and Irigaray (2015; this volume) EHS and MCS share a common pathological mechanism mainly involving the central nervous system. (Another interpretation is, of course, that the mechanism is not of a pathological central nervous system/brain nature at all but just a correct cellular reaction throughout the body to a toxic environment, moving the focus from the persons affected right to the surrounding environment nowadays filled with anthropogenic electromagnetic fields and chemicals at levels surpassing our most vivid imagination.)

Perspectives of risk – analysis and management

It is important that people understand that there are real risks associated with exposure to the radiation emitted by cell phones, their base stations, routers, tablets, wireless smart meters, computers, powerlines, and similar gadgets. We know that exposure to this radiation may impact and damage DNA, leading to possible cancer risks, neurological diseases, alter memory and concentration capacity, affect learning, it can alter our immune defence, and also affects male and female germ cells and fertility, it has effects on neurological functions such as cognition, behavior, performance, mood status, learning, memory, concentration, problem solving, morality, disruption of sleep and changing the sleep pattern, and may cause headaches and dizziness. The radiation is also harming animals and nature, and is therefore as much an environmental as human health issue. Important new research has already been presented on EMF impacts on DNA, heart function and the role of electrification in the diseases of civilization, such as heart disease, diabetes, cancer and suicide.

Beyond government regulations, it is imperative that each person educates themself and employs safer methods when using their modern devices. The complete lack of biologically-based exposure standards or hygienic safety levels by our health protection agencies, and the lack of preventive actions, can turn out to be a huge mistake. To not act today would be deemed ethically and morally completely corrupt in the future.

Many times since the early 1980s I have pointed to that the public's usage of cell phones has become the largest full-scale biological and medical experiment ever with mankind, and I was also the first person to firmly point out that this involuntary exposure violates the Nuremberg Code's principles for human experimentation, which clearly states that voluntary consent of human subjects is absolutely essential. Among many effects seen, the very serious one is the deterioration of the genome. Such an effect – if seen in a food item under development or in a potential pharmaceutical drug – immediately would completely ban it from further marketing and sale – genotoxic effects are not to be allowed or spread. Furthermore, when men place cell phones in their front pocket, it should be noted that experimental studies have demonstrated that after similar exposures there is a decrease in sperm count as well as in the quality of sperm, which is a phenomenon that could affect society's overall ability to procreate in the future. Experiments in mice point to that it may be true already in five generations time. More recently, my colleague Örjan Hallberg and myself have conducted important epidemiological studies (29–34) showing that wireless communication networks may be causing significant illness throughout society. We have also shown that increased rates of asthma as well as certain types of cancer are strongly correlated with exposure to radio broadcasting during the twentieth century.

Strong concern has been voiced by the public, and by scientists as well as public health and environmental policy experts, that the deployment of technologies that constantly expose billions of people worldwide to new sources of electromagnetic fields may pose a pervasive risk to public health (35–38). Such exposures did not exist before the age of industry and information. Prolonged exposure appears to disrupt biological processes that are fundamental to bacterial, plant, animal and human growth and health. Life on earth did not evolve with biological protections or adaptive biological responses to these electromagnetic field exposures. Exceptionally low levels of electromagnetic fields – apart from the sun rays and the geomagnetic field – existed during the time that all life evolved on earth in the order of less than a billionth to one ten-billionth of a Watt per meter squared. A rapidly accumulating body of scientific evidence of harm to health and well-being constitute warnings that adverse health effects can occur with prolonged exposures to artificial electromagnetic fields at biologically active frequencies or frequency combinations.

In November, 2009, the Seletun Scientific Panel adopted a Consensus Agreement (39) that recommends preventive and precautionary actions that are warranted now, given the existing evidence for potential global health risks. We recognize the duty of governments and their health agencies to educate and warn the public, to implement measures balanced in favor of the Precautionary Principle (40), to monitor compliance with directives promoting alternatives to wireless, and to fund research and policy development geared toward prevention of exposures and development of new public safety measures.

The Panel also strongly recommends that persons with electrohypersensitivity symptoms (EHS) be classified as functionally impaired in all countries rather than with "idiopathic environmental disease" or similar indistinct categories. This terminology will encourage governments to make adjustments in the living environment to better address social and well-being needs of this subpopulation of highly sensitive members of society, and – as a consequence – protect everyone now as well as in the coming generations from toxic environmental exposures. It is important to note that numeric limits recommended by the Seletun Scientific Panel, as well as by other bodies of society, do not yet take into account sensitive populations (EHS, immune-compromised, the fetus, developing children, the elderly, people on medications, etc). Another safety margin is, thus, likely justified further below the numeric limits for EMF exposure recommended by the Panel.

Legal, moral and ethical as well as practical consequences

As previously mentioned Sweden has officially recognized EHS as a disability. However, a lot of work still has to be carried out by the electrohypersensitive persons, as well as for them, and their disability organization, The Swedish Association for the Electrohypersensitive to achieve complete equality. Accessibility and adaptation are key issues for allowing EHS, and others with recognized functional impairments, to gain/regain their rightful independence. As is well known and well documented, such support can also benefit the entire society.

Society must recognize in practical applications the right of the electrohypersensitive to be different, to their distinguishing feature. Society must recognize the right of the electrohypersensitive to have an equal life in a society based on equality. Treating members of the community equally is not something that should be done as a favor; nor is it something that any parliament or government should politely request other inhabitants to provide

others with. Equality is not something to be done "out of the goodness of one's heart". It is something one does because it is expected of every citizen, because inaccessibility and discrimination are prohibited by law. Thus, it is not legal to deliberately make the situation worse for persons with functional impairments.

Some medical doctors and dentists have described at an early stage the electrohypersensitive persons as "old crones in the throes of the menopause", "the poorly educated", "hypochondriacs", "radiation ladies", or victims of union-driven fears, mass media-based psychoses, imagination phenomena, Pavlovian conditioning, techno-stress alterations, etc. These prejudiced care-providers used these terms despite often never having met an electrohypersensitive person or carrying out research in the field. Unfortunately, some medical doctors and a few scientists still instead want EHS to be a medical/psychiatric diagnosis, i.e. with patients with an undefined disease syndrome, the latter instead being the focus of medical/psychiatric treatments (thus, no automatic accessibility measures, including shielding of the environment).

In January 2015, we could witness in Sweden a huge massmedia-based attack launched against persons with the functional impairment electrohypersensitivity. They were accused of not having a proper medical diagnosis, no proofs to back their claims of ill health when exposed to the moderns society's artifical electrosmog, and so it was meant that they obviously should not be entitled to any economic support from the Swedish state.

Medical doctors (including the head of the Swedish Medical Association, Dr. Heidi Stensmyren), journalists, reporters, news anchors, and newspaper editors, all participated in this witch-hunt. This could be read in major newspapers, and heard and viewed in radio and TV channels. One Swedish medical doctor, Lena Hillert – on prime time public TV news – even introduced Santa Claus as a way of trying to ridicule persons with the functional impairment electrohypersensitivity.

The odd thing was that The Swedish Association for the Electrohypersensitive had done nothing wrong. In Sweden all disability organizations can apply for economic support, and – based on the number of members – a fixed governmental subsidy per person is granted via The National Board of Health and Welfare (Sw. "Socialstyrelsen").

There is much that could be done to increase accessibility: educating architects, planners, scientists, technologists and the general public more effectively about EHS, its causes and how it can be minimized; undertaking properly funded independent multidisciplinary research into EHS and showing that such work can make a difference. Creating work, home and general environments that are more user-friendly for EHS sufferers so that they feel included and not excluded in the rich tapestry of life (cf. ref. 41).

In a recent paper by Hagström et al. (42), it is concluded that "the official treatment options, psychotherapy and medication, did not have any significant effect. Instead, according to 76% of 157 respondents the reduction or avoidance of electromagnetic fields helped in their full or partial recovery. The best additional support for EHS were given as: "dietary change" (69.4%), "nutritional supplements" (67.8%) and "increased physical exercise" (61.6%)". Their results rhyme very well with previous studies (43–46).

Also remember that forcing people with such functional impairments out into various sanctuaries or zones is completely in opposition to the UN disability laws, the Standard Rules, and it's Convention, and could be the beginning of a terrible trend. No, instead make it easy for you – make sure to connect to all the UN texts, realize that it's the whole environment that must be accessibility-adapted, and do not forget that such accessibility measures actually are 100% positive for everyone to share. People with functional impairments should have full access to the entire society, not just a small part of it.

It is proposed that, as with those with other recognized disabilities, the electrohypersensitive persons must therefore, in every situation and by all available means, demand respect, representation and power. They shall very clearly reject all approaches which reflect a mentality of "feeling pity for them" or "caring for them" by introducing flimsy medical diagnostic criteria and 'treatments' based on cognitive behavioral therapy, antidepressants, vitamines, minerals, and massage (!). Inaccessibility is not a personal problem. It is a problem for society. Inaccessibility is not about attitudes. It is about discrimination. And discriminatory actions and conduct shall not be dealt with by well-meaning talk about treatment. Discrimination is already illegal!

In addition, to all such well-meaning medical/psychological treatments there is a potentially very dark backside: The possibly induced long-term health effect of any given treatment. I discussed it for the first time already back in the mid-80s when it was suggested that cognitive therapy should be used to 'teach' EHS persons that computers and computer screens were completely "safe". I then asked the clinical dermatologists and psychologists (who were in charge of that particular project) who would take the personal responsibility for future long-term health effects, such as cancer, in these employees. But no one stepped forward.

Complete accessibility

The former Swedish Prime Minister Göran Persson has declared that 2010 should be the final target year for the "Swedish Disability Action Plan" – "From patient to citizen" – adopted in 2000, according to which the whole of Sweden is to be completely adapted to those with disabilities (7). In addition, there is the EU "Human Rights Act" and the UN "Standard Rules on Equalization of Opportunities for People with Disabilities", nowadays updated to the UN "Convention on Human Rights for Persons with Functional Impairments" (6).

One of the most important ideas in these documents is the "principle of accessibility" stating that people with disabilities are to have full access to public services. This is the basis for the fast introduction in Sweden and other countries of kneeling buses, wheelchair ramps, hearing loops, automatic door-openers, beveled pavement edges, etc. However, it has to be asked where are the measures for the electrohypersensitive persons?

How are they to be able to be a normal part of the community with complete access to council offices, post offices, means of transport, cinemas, restaurants, hospital care and other facilities when electromagnetic pollution can affect them detrimentally? Providing every electrohypersensitive person with individually designed assistance, good care and stimulation to create participation in the community are very responsible tasks that require a high level of skill.

Considerably greater demands must be made on education, training and work supervision than has been the case to date. Sharing experiences between different activities must take place much more smoothly and in a way that is completely free of prestige.

People with the disability EHS have an exciting future ahead of them. Work has already started to produce results on a number of these issues, including building planning and construction (44). However, a lot more can and must be done. There are still many years of hard, constructive and consistent work waiting. I hope that everyone with the functional impairment EHS all over the world can find a common platform for this work, and help move the work forward through community and solidarity in the interdisciplinary work that is required.

It is now so important for everyone to decide on the continued direction of their activities as well as their focus and for people to work together with integrity as a team. A continued energetic action plan for the next few months and years together with a broad collaboration with other disability associations are of the utmost importance here. As clearly stated by the UN (6), there must be an end to nonchalance, lack of consideration, indifference and lack of respect on the part of society for those with disabilities for all our sakes. As moral human beings we should never accept negative discriminatory treatment or an insulting special treatment of those with impairments.

An equal life in a society based upon equality

When it comes to functional impairments, it is always only action that speaks, nothing else. To ensure that everyone acts within the UN Human Rights Convention is of paramount importance, and that persons with EHS is promptly given complete accessibility is the only acceptable goal, as is proper symptom identification and treatment when possible, but only when asked for by the disabled person Himself/Herself. However, the latter should never be used instead of the first.

Finally, to water the existing legislation down would make matters far worse for all the persons with EHS and/or multiple chemical sensitivity (MCS), as well as for their relatives. Therefore one must question attempts to move EHS from the functional impairment paradigm to the patient/disease one. It took me, and others, decades to get the EHS persons the protection of The UN 22 Standard Rules on the Equalization of Opportunities for People with Disabilities/The UN Convention on Human Rights for Persons with Functional Impairments. It will take less than a minute to destroy this. Mark my words.

The world is watching what we do, and we have the opportunity to do the right thing. Biomedical research is of immense importance to form a potential base for further symptom relief as well as for understanding the mechanisms behind, but let it not stand in the way of the human right of persons with EHS, MCS, and other functional impairments, to live an equal life in a society based upon equality.

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EXHIBIT N

Opt-Out and Opt-in Programs Across the U.S.

An increasing number of states and/or utilities are offering opt-outs of smart meters. While smart meters should be banned—along with all other wireless devices and digital electrical meters—until they are made safe for humans and other organisms, at least opt-outs provide some relief from the harm of EMFs. Below is a table of smart meter opt-outs and opt-ins. Eugene, Oregon, offers an *opt in* to smart meters.

Analog meters are definitively offered as the opt-out meter in four states: Arizona, California, Maine, and Texas. The Nevada public service commission urges the utilites to provide the meter that will have the "greatest customer acceptance," which, clearly, is the analog.

Three states offer the "existing meter" as the opt-out meter. An existing meter might or might not be analog. Georgia, Hawaii, and Consumers Energy in Michigan.

Three states let the utility decide: Florida, Maryland, and Nevada. The Nevada public service commission urges the utilites to provide the meter that will have the "greatest customer acceptance," which, clearly, is the analog.

Four states or utilities definitively forbid analog opt-outs: Fountain in Colorado, DTE in Michigan, Central Hudson Valley in New York (AMR), and Port Angeles in Washington (touted as the first place to ban smart meters—not much help you are required to have a digital meter on your home!).

Two states/districts require smart meters: Pennsylvania and Washington, DC.

The remaining states with opt-outs have policies that are unclear or that do not provide for analog opt-outs.

Opt-outs that do not provide for analog meters are the least useful, because of the harm from dirty electricity. Opt-outs like that currently in Michigan and other states that simply turn off the radio that transmits to the utility but leave in the transmitter, antennas, and the ZigBee wireless radio are the absolute worst.

Note that the AMR meters offered by utilities like Tucson Electric Power (TEP) are as dangerous as the regular smart meter. They are *not* a drive-by AMR meter that wakes up only when someone with handheld device drives by. They communicate with the utility every 30 seconds according to the information on TEP's own website, often via collection towers. In some instances, their signal is picked up by drive-by meter readers. Regardless, *any* AMR meter and *any* digital meter generates dirty electricity. The only meter that does not generate dirty electricity or emit RF is an analog meter.

Smart Meter Bans or Opt-Ins

Smart Meter Bans in California
Over 50 local governments in California have voted to ban smart meters. See StopSmartMeters.org for a list.

Unincorporated areas of Marin County vote to ban smart meters for another year. The Board of Supervisors, declaring "a current and immediate threat to public health, safety and welfare," outlawed installation of the meters but left enforcement issues up in the air. The state Public Utilities Commission, and not the county board, has jurisdiction over the Pacific Gas & Electric Co. meter program.

County supervisors also agreed to post information about Smart Meters on the county website, and consider appointment of a "point person" to help residents with questions about meter matters.

PG&E's Brittany McKannay said 191,931 Smart Meters have been installed in Marin, and 3,495 customers have opted out. *February 4, 2014.*

Fairfax Town Council in Marin County, California unanimously votes to impose three-year ban on the installation of Pacific Gas and Electric's smart meters. Fairfax Mayor David Weinsoff said that when the Fairfax Town Council conducted public hearings on smart meters in 2010, the community's opposition was overwhelming. "When a community speaks so loudly and so wisely, really there was no question that the council should continue to impose this moratorium," Weinsoff said.

Just like DTE, the California utility says that local entities cannot impose a moratorium. Several California governmental entities, including the Fairfax Town Council and the Marin County Board of Supervisors, have filed an administrative challenge to California's opt-out ruling.

Fairfax Councilman Larry Bragman says that the meters are a poor investment of ratepayers' money and that "there are potential health effects that have not been fully studied. The impact to privacy has not been dealt with effectively by the California Public Utilities Commission (CPUC). That is becoming more of an issue, now that the awareness of privacy issues has become so much more a matter of public concern." *February 10, 2014.*

Opt-In in Eugene, Oregon. Eugene, Oregon's municipal utility, EWEB, requires people to opt in to smart meters. People can keep their existing meter, which may or may not be an analog. City of Eugene Resolution No. 1322, October 1, 2013.

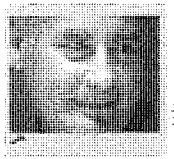
https://www.smartmetereducationnetwork.com/optout-status-other-states.php

EXHIBIT J

Source : www.next-up.org

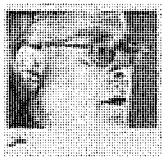
The Microwave Syndrome

(Public version)



Claude Monnet

Ph.D. in Radiology
Next-up représentant
Vice president.



Pierre le Ruz

Ph.D. in Physiology
President of the Scientific
Commission of Criirem.

▶ 1 - Electromagnetic fields (EMF) from microwaves have an impact on any living matter, (300 MHz to 300 GHz = hyper frequencies = microwaves).

▶ 2 - In accordance with the universal laws of physics, all scientific studies carried out in the world show that further to radiation's from hyper frequency or microwaves either thermal effects or non-thermal specific effects can be observed. The specific effects and the microwave syndrome (or hyper frequency syndrome) were first described as early as 1960 by Russian scientists. Later on in 1998, this will be confirmed in an American peer reviewed paper (NCBI) that will explicit the link between this syndrome and exposure to pulsed hyper frequencies.
(Refer to world studies on EMF : 1, 1', 2, 3, 4).

- Thousands of doctors launch warnings (Doctor's calls), the subject makes the headlines in newspapers on and TV; unfortunately in some cases only the sensational is covered: stress, depression, or even suicide.
- A large and recent survey was recently published by the Swiss Federal Office for Public Health on a group of 342 general practitioners (GPs) consultations from which it appears that 5 % of the Swiss population attribute symptoms to electromagnetic fields (EMF).
- In November 2006, CRIIREM and NEXT-UP took part to a European opinion consultation organised by the European Commission on evidence relative to this syndrome on populations in Europe.
- ONG's have taken an active part to the European Commission recent seminar in Brussels.

▶ 3 – Analysis of all the world scientific studies on effects of hyper frequencies on humans :

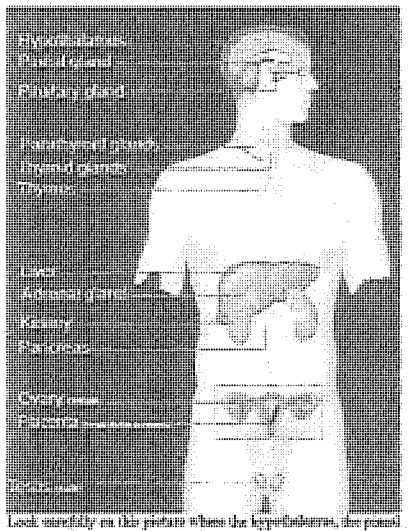
A – Contrary to the messages commonly conveyed by some people, no one study in the world has been able to prove the harmlessness of electromagnetic radiation from microwaves on human beings.

B – Most scientific studies to date that are not entirely or partially financed by telecoms (including the wireless industry and mobile phone operators) not only show biologic effects, but also the so called health effects on living beings, and therefore on humans.

C – The most important scientific study world-wide done today is the one from WTR (Wireless Technology Research - USA) 100 % financed by telecoms to a level of 28 millions dollars. It shows so called biologic effects but also gives evidence of so called health effects in particular on DNA that were later confirmed by the large REFLEX study made by the European Community.

- Alert letter from George Carlo, Ph.D., general co-ordinator of the WTR US Study.
- Video interview with Dr George Carlo, Ph.D., (extract).
- Alert letter from Pr. Franz Adlkofer, general co-ordinator European Union Study REFLEX.
- Video interview with Pr Franz Adlkofer (extract).

▶ 4- The interaction of EMF from hyper frequencies with the human bio-electromagnetic metabolism generates the so-called microwave syndrome or hyper frequency syndrome.



Look carefully on this picture where the hypothalamus, the pineal gland and the hypophyse are located !...

Mechanisms
Mechanisms and effects in alarm and resistance phase characterise the microwave syndrome :

▶ First case, alarm phase :
The brain subject to a short stimulation of artificial EMF radiation of microwave type triggers specific reactions from the organism involving neuronal, neuro-endocrinal, metabolic and behavioural responses.

A- Those responses fall in the general scheme of an individual stress adaptation, this in a more or less adapted manner.

B - The take over of the stress element (radiation) is done by :
- The Central Nervous System (CNS),
- The Peripheral Nervous System (PNS)
- The Endocrinal System (ES).

Those responses are divided in 3 stages :

1.Stress reception by sensorial organs and their relating nerves.

2.Programming of the stress reaction at the cortex and the limbic system (LS) levels (tonsils, olfactory bulb, hippocampus, septum, mamillary body ...). The Cortex/LS couple is a comparative analysis system using a data bank of "memory" based on experiences. Therefore the brain compares the new situation with past experiences in order to create an adapted response.

3.Activation of the response from the organism via the tonsils and the hippocampus that act on the hypothalamus and on the cerebral trunk reticulated formation in order to activate the Vegetative Nervous System (VNS) and the Adrenal Gland (AG). The alarm amplitude is regulated by the Limbic System (LS).

▶ Second case, resistance phase :

After the alarm phase, if the stress element persists even at low dosage (ex : radiation from phone masts) or becomes chronic (e.g. : radiation of an addicted mobile phone user who does not respect the body auto regulation in relation to the SAR rating of his mobile), the result is that the hypothalamus, etc ... are going to assess these constant stress and activate the secretion of various hormones, . . .
Unfortunately as a rule, a human being exposed to artificial radiation's does not have a pre-established strategy in his cognitive repertoire to efficiently defend himself against this type of attack. Therefore the hypothalamus stimulation produces a stereotyped non adapted response to this type of aggression ... which often increases the negative impacts (e.g. the case of the college of Chabeuil in France).

- As far as the metabolism of some people is concerned, everything will go smoothly temporarily during a period from a few days to several decades, however their health capital is eroding really rapidly. There is a risk that pathologies such as Alzheimer's disease that "usually" appear when approaching the third age risk could appear earlier

- As far as the metabolism of all the people in a state of weakness (sick people, elderly), foetus, baby's, etc... there is a rapid exhaustion and state of disorder of all nervous and endocrine systems, and consequently of the immune system.

This will promote and initiate the emergence of a number of known pathologies.
The end result of this mechanism is what is called the MICROWAVE SYNDROME.

France 3
Pierre Le Ruz,
Ph.D. in Physiology

The most usual pathologies resulting from the microwave syndrome are (non exhaustive list) :

- **Dystonic cardiovascular syndrome** : bradycardia, tachycardia, hyper/hypo blood pressure, and atherosclerosis...
- **Chronic diencephalic syndrome** ; dizziness, sleep troubles, concentration disturbance, sensory troubles, and loss of concentration, chronic fatigue syndrome.
- **Chronic asthenia syndrome** : fatigue, irritability, nausea, headaches, anorexia, and depression.
- **Cancerous pathology** : leukaemia, glutathione and melanoma, breast cancer, ... (the file InVS)
- **Dermatologic pathology** : dermatitis, dermatosis, eczema, psoriasis, and skin allergy.
- **Dopaminenergic pathology** : Parkinson, legs without rest, loss of sensibility in 4 limbs, tighten arms at wake-up, cramps in limbs, ...
- **Immune pathology** : blood formula (high rate of lymphocytes), ...
- **Hyper Sensibility pathology** : attributed beforehand to psychic disturbances !
- **Pre and post-native pathology** : great premature (often before or around gestation age), toxic foetopathy, miscarriage, retarded growth, biometrics, genotype modification, then puberty modification (of which associated to BBB opening).
- **Procreative pathology** : drastic decrease of semen (infertility) ...
- **Hypogonadism pathology** : drastic decrease of libido (stimulating follicle hormone) *(video/PASSword)*.
- **Brain pathology** : tumours, opening of the **BBB** (Blood - Brain Barrier), electroencephalogram disturbances, ...
- **Standard pathology** : visual and hearing perturbations, nose bleedings, injured corner lips, jaw bleedings, fibromyalgy, allergy, asthma, tooth neuralgia, etc...
- **Psychical pathology** : lack of concern, introversion, passiveness, submissiveness, depression and mental anorexia, suicide ... and cerebral activity (behaviour control).
- **Disturbances of socio-professional behaviour** : irritability, discomfort, and ... increased risk of accident. Stress, depression, suicide.

EXHIBIT K

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Electromagnetic field exposure and health: Microscopic, radiological and stereological studies

Süleyman Kaplan, PhD.

Concerns about the effects of electromagnetic radiation on the human body have increased due to advances in and ever-greater use of communication devices. The biological effects of exposure to electromagnetic fields (EMF) are currently a popular subject for researchers. EMF exposure is reported to cause various effects on the human body. However, it is unclear whether these effects cause any adverse physical outcomes. How organisms are affected by this growing worldwide mobile phone use is an important question, and several scientific studies have been performed in order to investigate this.

Numerous devices are known to emit EMF, such as communication equipment, substations, mobile phone base stations, radio and television transmitters, electrical equipment at home and work, and high voltage lines, in addition to the many electrical systems in the environment. However, while life is made considerably easier by these devices, they may also have deleterious effects on health. Mobile phones are now starting to be used at elementary school age, meaning that lifetime exposure to EMF is increasing. Studies have shown that symptoms such as stress, headache, fatigue, anxiety, decreased learning potential, impairment in cognitive functions and poor concentration may occur due to exposure to microwave radiation emitted by mobile phones. Several metabolic processes and mechanisms in the organism are also affected by EMF exposure. The body's chemical integrity may be damaged by EMF exposure due to the fact that high electromagnetic absorption is capable of altering the electrical current in the body. Different parameters have recently been used to assess the biological effects of *in vivo* and *in vitro* exposure to non-ionizing radiation in animals, humans and cells. Unbiased evaluation of the beneficial or hazardous effects of EMF exposure from various different viewpoints using ultrastructural and morphological approaches is now required. The main goal of this special issue is to shed light on this subject.

Kıvrak et al.'s review reports the effects of oxidative stress on antioxidant systems and suggests that EMF may cause oxidative stress in several tissues. The authors report that various antioxidants can be used to prevent the potential damage resulting from EMF exposure.

Warille et al.'s review discusses the enzymatic and hormonal changes occurring during EMF exposure and the underlying cellular mechanisms. The mechanism of these effects may differ, depending on the intensity of magnetism. The authors suggest that more studies involving experimental and epidemiological characteristics and focusing on the deleterious effects of EMF exposure on enzymatic and hormonal mechanisms are now required.

Kocaman et al. focus on the protective effects of omega-3 on the rat adrenal gland exposed to 900-MHz EMF. The authors report that omega-3 treatment reduces the side-effects of EMF exposure in the rat kidney.

Deniz et al. investigate the effects of mobile phones, not only on the morphology of the human brain but also on cognitive performance using stereological and spectroscopic methods and neurocognitive tests. Their results indicate that subjects who talk on mobile phones for prolonged periods during the day display low concentration and attention.

Another study by Deniz et al. reports that the oxidative damage exerted by electromagnetic radiation emitted by cell phones in the rat kidney is ameliorated by folic acid. That study used stereological techniques to evaluate glomeruli numbers as well as kidney volume.

Etet et al. report that high illuminance visible electromagnetic radiation leads to various changes in the functions of the brain and general health in mice, which are partially mediated by damage to the neocortex-entorhinal cortex-hippocampus axis. These findings suggest a need for caution in the use of high illuminance artificial light for extended periods.

Kıvrak et al.'s paper reveals the effects of 900-MHz EMF emitted by mobile phones on Ammon's horn and the dentate gyrus in the hippocampus and cerebellum of male *Wistar albino* rats. The authors also investigate the neuroprotective effects of antioxidants such as *Boswellia sacra* and folic acid against the side-effects of EMF exposure. The stereological and histological results of this study indicate that EMF exposure causes deleterious effects on the cells of the hippocampus and cerebellum. *Boswellia sacra* and folic acid were observed to exhibit protective effects in the cells of the hippocampus and cerebellum against exposure to 900-MHz EMF.

Chandel et al. focus on the role of cell phone electromagnetic field radiations (EMF-r) in inciting oxidative damage in onion (*Allium cepa*) roots at a frequency of 2100 MHz. Their study suggests that onion roots undergo abiotic stress caused by EMF-r and that this induces oxidative damage in the plants.

Altun et al. report the effects of pulsed digital electromagnetic radiation emitted by mobile phones on the central nervous system of the adult *Wistar albino* rats. That study found neuroprotective effects of melatonin (Mel) and omega-3 (ω3) based on the antioxidant defence system.

We would like to express our particular gratitude to all the authors for their excellent submissions to this special issue. We also express our sincere thanks to the invaluable reviewers from all around the globe who gave their precious time to review the submitted papers. We are especially grateful for their rapid responses throughout the reviewing process. Finally, our profound thanks to Editor-in-Chief Prof. Abdulmoneam Al-Hayani and his team for their vital editorial support in the production of this special issue.

EXHIBIT L

Ranking Electricity Meters for Risk to Health, Privacy, and Cyber Security

Table 1 summarizes the risk rankings of electricity meters, based on the detailed analysis that follows. "5" is the highest risk. Blank is the lowest risk. Capital letters group meters with similar, but not necessarily identical, risk rankings. The priorities among the three types of risk addressed are these:

- **Health:** The meters are arranged in descending order by Risk to Health, which the author believes to be the single most important risk factor.
- **Privacy:** The meters with the same Risk to Health are arranged in descending order by Risk to Privacy.
- **Cyber Security:** The meters with the same Risk to Health and the same Risk to Privacy are arranged in descending order by Risk to Cyber Security.

The Wireless Smart Meter poses the highest risk in all three categories of risk. In contrast, the Traditional Analog Meter poses the lowest risk in all three categories of risk. This analog meter can also be correctly called:

- Traditional Analog Mechanical Meter
- Traditional Analog Electromechanical Meter
- Traditional Analog Mechanical Meter with No Wireless Communications Capability
- Traditional Analog Mechanical Meter with No Electronic Circuitry.

Table 1: Summary of Risk Rankings

	Meter Category	Type of Communication	Wireless	Wired	Overall Risk (5 is highest. Blank is lowest.) Risk to Health	Risk to Privacy	Risk to Cyber Security
G	SMART METER	WAN/ HAN	✓		5	5	5
F	AMR Meter	Bubble Up	✓		4	4	
	Analog Meter (plus wireless digital electronics)	Bubble Up	✓		4	4	
E	AMR Meter	Wake Up	✓		3	2	
	Analog Meter (plus wireless digital electronics)	Wake Up	✓		3	2	
D	SMART METER	Internet cable/fiber		✓	2	4	4
	AMR Meter	Internet cable/fiber		✓	2	4	
C	SMART METER	Telephone landline		✓	2	3	3
	AMR Meter	Telephone landline		✓	2	1	
B	Basic Digital Electronic Meter	None			2		
A	Traditional Analog Meter	None					

Introduction[1]

The manufacturers of electricity meters offer a wide variety of models. And many of these models are available with a dozen or so options, leading to many possible combinations. These meters have capabilities beyond what is required to measure the electricity consumed for the purpose of issuing a monthly bill. Unfortunately, the new capabilities present a host of risks to health, to privacy, and to cyber security, as has been widely discussed elsewhere. But, briefly stated --

- The risks to health arise primarily from the fact that many electricity meters communicate wirelessly with the electric power companies. They transmit radiofrequency radiation, at microwave frequencies, day and night, every day of the year, forever. That radiation travels through homes and businesses readily, and penetrates the unborn, the children, and the adults alike, disrupting health. Every transmitting meter in a community irradiates everyone in that community. So does every community-based transmitter/receiver that the electric power companies have erected to communicate wirelessly with those meters.
- The risks to privacy arise from the fact that many of the meters capture and transmit very highly time-resolved information about electricity consumption. That detailed information can reveal much about the activities taking place inside the homes and the businesses, sufficient, for example, to reveal when no one is there.
- The risks to cyber security arise, in part, from the fact that some types of meters can accept incoming wireless commands that may come from nefarious sources. Many of those meters can respond to wireless commands to shut off the electrical power to a home or a business entirely, or to accept new software programming. That new programming can alter the functions of the meters and can do so invisibly to the owners of the homes and the businesses.

The variety of meters now in service, and the many risks that the meters can pose, mean that it has become impossible for the customer of an electric power company to know what capabilities his meter has and what risks those capabilities imply for him, his family, his business, and his community. In short, the electric power companies have moved --

- *from* the accepted practice of measuring electricity consumption once a month for the purpose of issuing a proper monthly bill
- *to* the questionable practice of monitoring the daily activities inside individual homes and businesses, as reflected in their detailed patterns of electricity consumption

[1] The author, Ronald M. Powell, is a retired career U.S. Government scientist (Ph.D. in Applied Physics from Harvard University, 1975). During his Government career, he worked for the Executive Office of the President, the National Science Foundation, and the National Institute of Standards and Technology. Contact: (301) 926-7568, ronpowell@veriton.net.

- *then* broadcasting those details over the air using wireless technology that pollutes entire communities with hundreds of millions of bursts of microwave radiation every day, forever.

Purpose of this Document

The purpose of this document is to provide some perspective about the types of meters available and the relative risks that they pose. Because there are so many types of meters available, not all of them could be addressed here. The meters selected for this document are those that I have seen in service most often in my state of Maryland. Many, if not most, of these meters are also employed in other states.

Principal Conclusions

Only one type of meter, the Traditional Analog Mechanical Meter with No Wireless Communications Capability, represented by the last row in Table 2 on page 6, offers ***all*** three of the following positive characteristics:

- no risk to health from RF radiation exposure, because it generates no RF radiation
- no risk to privacy, because it cannot be remotely read
- no risk to cyber security, because it cannot be remotely accessed so it cannot be hacked.

In sum, the meter that poses the lowest risk to health, to privacy, and to cyber security is the Traditional Analog Mechanical Meter with No Wireless Communications Capability. Tragically, this is the meter that many electric power companies in many states, including my state of Maryland, have chosen to remove.

Only one type of meter examined here, the Wireless Smart Meter, represented by the first row in Table 2 on page 6, has ***all*** three of the following negative characteristics:

- the highest risk to health because it has the highest Peak Radiofrequency (RF) Power Output, and because it has either the first or the second highest number of bursts of RF radiation per day, depending on its operating mode

- the highest risk to privacy because its data are potentially accessible to the second largest number of people, but with the greatest ease of access to the data stream (because it is wireless), and because it provides data with the greatest timeliness, the greatest granularity (finest time-resolution), and the greatest variety (most types of data), all of which make that data highly intrusive

- the highest risk to cyber security because it is potentially accessible to the second largest number of people, but again with the

greatest ease of access (because it is wireless), and because it is the most vulnerable to being harmed itself, and because it is the most able to cause harm

  - because it contains a shutdown switch capable of shutting off all power to the customer when triggered to do so by a wireless remote signal

  - because it is software reprogrammable to perform new functions, whether beneficial or harmful, entirely invisibly to the customer.

In sum, the meter that poses the highest risk to health, to privacy, and to cyber security is the Wireless Smart Meter. Tragically, this is the meter that many electric power companies in many states, including my state of Maryland, have chosen to install.

The other meters discussed in this document fall somewhere in between the above two meters.

- All of these other meters pose a risk to health from RF radiation exposure, although to widely differing degrees.
- All, but one of those other meters, pose a risk to privacy.
- Two of those other meters pose a risk to cyber security.

Organization of the Rest of this Document

Ranking Electricity Meters for Risk to Health

Table 2 on page 6 ranks electricity meters for their risk to health, based on the characteristics of the "Sources of Radiofrequency (RF) Radiation" that the meters contain. All of the terms used in the table are described in the text that immediately follows the table.

The rows in the table name the "Meter Category" into which each meter falls and the "Type of Communication", whether wireless or wired, that each meter employs. In the central part of the table, the column headings and subheadings describe the "Sources of Radiofrequency (RF) Radiation" in each meter and the characteristics that those sources have that are of relevance to their risk to health. The presence of a red cell in a given row and column means that the meter described in the heading of that row employs the RF radiation source described in the heading of that column and poses a risk to health.

The first of the column subheadings indicates the "Peak RF Power Output" of each RF source. Note that the sources are arranged, from left to right, from the lowest to the highest levels of Peak RF Power Output. Peak RF Power Output is one of the most important factors affecting the risk to health. The higher the Peak RF Power Output, if other factors are equal, the more RF radiation is being delivered and the higher the associated risk to health.

The second of the column subheadings indicates the number of "Bursts of RF (Radiation) per Day" or per month, if known, issued by each Source of RF Radiation. The higher the number of Bursts of RF Radiation per Day, the more RF radiation is being delivered and the higher is the associated risk to health.

The last column on the right side of Table 2 labelled "Overall Risk", shows a numeric ranking for the "Risk to Health", for each row, that is for a given meter with a given type of communication. The higher that number, the higher is the associated risk to health. All of the numbers in the red cells under "Risk to Health" indicate a *rank order*. That is a Rank 5 meter presents more risk to health than a Rank 4 meter. Rank 5 represents the highest risk, and a blank cell represents the lowest risk. Note that the rankings are not quantitative beyond the order that they indicate. That is, the rankings do not indicate *how much a given meter differs from another*. The quantitative difference is better judged by examining the differences in Peak RF Power Output and in Bursts of RF per Day, where those numbers have been provided. In some cases, I did not have good numbers to offer; but I still made an educated, but only qualitative, guess, such as "very low" or "high". The purpose was to indicate the range into which I would expect the actual numbers, if known, to fall. I flagged each such entry as "unknown".

Note that no meter in Table 2 has Risk 1. That is largely because every electricity meter in the table, except the Traditional Analog Meter, has at least *two* sources of RF Radiation: digital electronics and a switching power supply. Both of these sources generate RF radiation even if at a very low Peak RF Power Output. Note that I assume here that all meters with digital electronics have switching power supplies, even though I have not been able to confirm this. Only one meter, the Traditional Analog Meter, has no sources of RF radiation and thus merits a blank for Risk to Health, indicating that it is clearly the safest meter for health of them all.

Table 2: Ranking Electricity Meters for Risk to Health

(RED means "generates radiofrequency radiation at a level potentially risky to health".)

Meter Category ↓	Type of Communication ↓			Sources of Radiofrequency (RF) Radiation in Meter						Overall Risk 5 is highest. Blank is lowest.
				Unintentional Radiators		Intentional Radiators (transmitters)				
				Digital Electronics	Switching Power Supply	Not Networked Transmitter/Receiver		Home Area Network (HAN) Transmitter/Receiver	Wide Area Network (WAN) Transmitter/Receiver	
	Peak RF Power Output →			*very low (unknown)*	*very low (unknown)*	*low (1 mW)*	*low to medium (1-100 mW)*	*medium (100 mW)*	*high (1000 mW)*	Risk to Health
	Bursts of RF per Day →			*high (unknown)*	*high (unknown)*	*low (8 per month)*	*high (50,000 per day)*	*high (unknown)*	*high (10,000 (avg) to 190,000 (max) per day)*	
		wireless	*wired*							
SMART METER Digital Advanced Metering Infrastructure (AMI)	WAN/ HAN	✓								5
	Internet cable/fiber		✓							2
	Telephone landline		✓							2
Digital Automated Meter Reading (AMR)	Bubble Up	✓								4
	Wake Up	✓								3
	Internet cable/fiber		✓							2
	Telephone landline		✓							2
Basic Digital Electronic Meter	None									2
Analog Meter (plus wireless digital electronics)	Bubble Up	✓								4
	Wake Up	✓								3
Traditional Analog Meter	None									

Description of Electricity Meters

Meter Categories

A **Smart Meter** is the key component in what is called the **Advanced Metering Infrastructure (AMI)**. As a result, Smart Meters are often referred to as AMI Meters. The Smart Meter is a digital electronic device that monitors the flow of electricity into, and out of, a customer's residence or business and provides two-way communication between the customer's meter and the electric power company. The information sent to the electric power company describes many characteristics of that flow of electricity, and on a very timely and highly time-resolved ("granular") basis. That communication is usually accomplished with a **wireless** transmitter/receiver, called a Wide Area Network (WAN) because it has a high enough Peak RF Power Output to travel great distances. It is this WAN which gives rise to most of the health concerns about Smart Meters, while simultaneously heightening both the privacy and the cyber-security concerns, both because of the wide area covered by the signals and because of the accessibility of any signal sent through the air.

Each Smart Meter can be, and usually is, equipped with a second **wireless** transmitter/receiver, called a Home Area Network (HAN), to communicate wirelessly with individual so-called Smart Appliances located inside a home or a business, further heightening health, privacy, and cyber-security concerns. The HAN is intended to return to the electric power company information on the identity and the use of each Smart Appliance in a home or business. Whether the HAN can also enable the electric power company to control those Smart Appliances is unknown to me.

Communication between Smart Meters and electric power companies can also be accomplished with **wired** technologies, such as telephone landlines and Internet connections (cable or fiber), which significantly reduce the radiation exposure produced by the Smart Meters, but do not eliminate it entirely. Wired communications for Smart Meters are not employed in my state of Maryland, as far as I know. As an example of a **wired** installation, fiber optic cable is employed in Chattanooga, Tennessee to communicate with that town's Smart Meters.[7]

The digital **Automated Meter Reading (AMR)** meter is another category of digital electronic device that monitors the flow of electricity into, and out of, a customer's residence, but not usually at as high a level of detail as a Smart Meter. The AMR meter can provide **wireless** communication but not usually directly back to the electric power company. Rather, the AMR meters communicate wirelessly with a passing utility vehicle (drive-by reader) or with a passing meter reader on foot (walk-by reader), each equipped with

[7] Your Gig is Here. Right here, in Chattanooga (http://www.chattanoogagig.com/). How Chattanooga beat Google Fiber by half a decade, The Washington Post, September 17, 2013. (http://www.washingtonpost.com/blogs/the-switch/wp/2013/09/17/how-chattanooga-beat-google-fiber-by-half-a-decade/).

the electronic equipment needed to communicate with the AMR meter. Because the distance over which the AMR meter must communicate is shorter than that of a Smart Meter, the Peak RF Power Output of an AMR meter is usually less than that of a Smart Meter. Two different types of wireless communications are offered: Bubble Up and Wake Up. They are described on page 9. If AMR meters are equipped with the proper options, they can also communicate through wired Internet connections (either cable or fiber optic) or through telephone landlines. Either of these latter two approaches enables communication all the way back to the electric power company, so no drive-by or walk-by meter reader is required.

A **Basic Digital Electronic Meter** is the simplest of all digital electronic meters. It does not employ any wireless or wired communications technology. Such meters are read by a walk-by meter reader who must enter the customer's property. These meters have just two sources of RF radiation -- digital electronics and switching power supply -- so they pose a lower risk to health than some of the other meters. These meters may be able to store a very large amount of data collected between readings by the walk-by meter reader, depending on the options (such as additional electronic memory) that they contain, which may raise some privacy concerns. And that data may be readable very quickly through an electronic or optical interface that the walk-by meter reader connects to the meter. But because such included capability is optional and because these meters are read infrequently (once a month typically), I have ranked these meters as having no risk to privacy. These meters pose no risk to cyber security, either, since they cannot be remotely accessed and therefore cannot be hacked.

An **Analog Meter (plus wireless digital electronics)** is a Traditional Analog Mechanical Meter or, equivalently, a Traditional Analog *Electromechanical* Meter, to which has been added digital electronic circuits, powered by a switching power supply, to give the meter wireless Wake-Up or wireless Bubble-Up communications capability. These capabilities are discussed further on page 9. They enable the meter to be read remotely from a short distance by a drive-by or a walk-by meter reader so that the meter reader does not have to enter the customer's property.

The Traditional Analog Mechanical Meter with no Wireless Communications Capability or, equivalently, the Traditional Analog *Electromechanical* Meter with No Wireless Communications Capability, is what is usually meant when the shorter descriptions -- Analog Meter, or Analog Mechanical Meter, or Analog Electromechanical Meter -- are used. This meter contains no digital electronic circuits and thus needs no switching power supply to power those circuits. This type of meter contains no sources of RF radiation, whether intentional or unintentional, and thus does not give rise to a risk to health from exposure to RF radiation. This type of meter also poses no risk to privacy because it cannot be remotely read, and it poses no risk to cyber-security because it cannot be remotely accessed and therefore cannot be hacked. This result is reflected in the blank cells across the bottom row of Table 2 on page 6 and the bottom row of Table 3 on page 12, reflecting the fact that this meter is the safest one available with regard to risk to health, privacy, and cyber security.

Sources of Radiofrequency (RF) Radiation

All of the sources of RF radiation in electricity meters can radiate directly into the air. Those sources can also deliver RF electrical current directly into the house wiring, or they can induce RF electrical current in the house wiring, which then can radiate into the air. The sources of radiation are described below in order of increasing Peak RF Power Output. While the sources are of widely different levels, even the least powerful of them, the "Unintentional Radiators", can disrupt highly sensitive individuals and thus cannot be ignored.

Unintentional Radiators are sources of radiofrequency radiation that radiate because of their inherent nature, not because the radiation is needed to perform a communications function.

> **Digital Electronics** operate by turning the flow of electrical current on and off sharply and rapidly. These transitions in current flow produce unintentional radiofrequency radiation, whether or not the same electronics also produce intentional radiofrequency radiation for wireless communication. This unintentional radiation is likely lower in Peak RF Power Output, and higher in frequency, than the unintentional RF radiation from Switching Power Supplies.
>
> **Switching Power Supplies** convert the high incoming line voltage from the electrical power system to the lower voltages required to power digital electronic circuits. In this conversion process, these supplies turn the flow of electrical current on and off sharply and rapidly. These transitions in current flow produce unintentional radiofrequency radiation. This unintentional radiation is likely higher in Peak RF Power Output, and lower in frequency, than the unintentional RF radiation from Digital Electronics. (Switching power supplies are also called switched-mode power supplies or switching-mode power supplies.)

Intended Radiators are sources of radiofrequency radiation that must radiate to perform their intentional function, which in this case is the transmission of information in a wireless communication system.

> The **Not Networked Transmitter/Receiver** category includes both the Bubble-Up and the Wake-Up types of communication for electricity meters. Together these two types of communication are sometimes called "encoder, receiver, transmitter" or "ERT" communications. The meters are not linked together in a network.
>
> > **Bubble-Up** meters send their meter readings as wireless signals every second or so, all day and all night long, every day of the year. The purpose of such frequent transmissions is to assure that a signal is available whenever a drive-by or a walk-by employee of the electric company passes with electronic equipment that can pick up and store the information carried by that signal. Bubble-Up meters are offered with Low and Medium Peak RF Power Outputs. I have not yet

> > learned whether some Bubble-Up meters have a receiver for a different purpose, such as to accept changes in their internal programming.
> >
> > **Wake-Up** meters contain receivers that listen for a wireless signal (a "Wake-Up" signal) transmitted by a drive-by or a walk-by meter reader and then respond with a series of transmissions that contain the meter's information. Eight such transmissions, one immediately after the other, appear common. The Wake-Up meters do not transmit at all in between such wake-up signals. As far as I can determine, Wake-Up meters operate only at Low Peak RF Power Output. Because of their infrequent transmissions and their Low Peak RF Power Output, Wake-Up meters produce much less radiofrequency radiation than Bubble-Up meters.
>
> A **Home Area Network (HAN) Transmitter/Receiver** is one of two wireless methods of two-way communication that is usually, but not always, included in Wireless Smart Meters. The HAN is sometimes called the Zigbee Network, after the technology on which it is based. The HAN is designed to communicate with emerging so-called Smart Appliances inside each home or business. The purpose is to monitor those appliances and to transfer information about their identity and their use back to the electric power company. It is possible, but not yet clear to me, that the HAN will enable the electric power companies to exert a degree of remote control over Smart Appliances. The HAN transmits RF radiation (at microwave frequencies) with a Medium Peak RF Power Output. I have not yet found data on how often the HAN transmits its bursts of RF radiation, but I suspect that the burst rate will be high, like that of other types of local area networks (LANs). Depending on that answer, the radiation produced by the HAN may rival or exceed the radiation produced by the WAN.
>
> A **Wide Area Network (WAN)** is a wireless method of two-way communication for Smart Meters and usually takes the form of a so-called Mesh Network. The WAN transmits RF radiation (at microwave frequencies) at a High Peak RF Power Output and thus has considerable range, hence the "wide" in Wide Area Network. In such a network the Smart Meters communicate with each other constantly. Each Smart Meter sends information about the use of electricity in the home or the business that it primarily serves. Each Smart Meter also relays information from the Smart Meters of neighboring homes and businesses. This relay action helps to assure that the information ultimately reaches the community-based transmitters/receivers, erected by the electric power company, which then transmit the information back to the company by any of a variety of methods. The intense level of communication employed by this Mesh Network results in 10,000 bursts of RF radiation per day from each meter (on average) and up to 190,000 bursts of RF radiation per day (at a maximum) from each meter.[3] Because of this intense level of communication, the number of bursts of RF radiation blanketing an entire community equipped with Smart Meters can reach tens of millions to hundreds of millions of bursts per day. That does not count the bursts of radiation sent throughout the

[3] Pacific Gas and Electric Company's Response to Administrative Law Judge's October 18, 2011 Ruling Directing it to File Clarifying Radio Frequency Information, page 5. (http://emfsafetynetwork.org/wp-content/uploads/2011/11/PGERFDataQuestionsAlternatives_11-11-11rev.pdf)

community by the community-based transmitters/receivers erected by the electric power company to communicate with the Smart Meters. I have not yet found any data on the Peak RF Power Output, or the Bursts of RF Radiation per Day, of such transmitters/receivers.

Levels of Peak RF Power Output

Four levels of Peak RF Power Output are referenced in this document:

- "Very Low" power means well below 1 milliwatt (mW) of Peak RF Power Output.
- "Low" power means 1 milliwatt (mW) of Peak RF Power Output.
- "Medium" power means 100 milliwatts (mW) of Peak RF Power Output.
- "High" power means 1000 milliwatts (mW), which is equivalent to 1 watt (W), of Peak RF Power Output.

Wired Methods of Communication

Some Smart Meters, and some AMR Meters, can be equipped to return information to the electric power company through wired communications systems. These systems employ no intentional radiators and thus do not raise health concerns associated with intentional RF radiation itself. They still employ digital electronics and switching power supplies, however; so they do still add to the level of unintentional RF radiation in the environment. Two types of wired communications systems are referenced in this document:

- The "Internet" can be used via wired technologies, such as coaxial cable and fiber-optic cable.
- "Telephone Landlines" can be employed that are based on copper wiring, coaxial cable, and fiber-optic cable.

Ranking Electricity Meters for Risk to Privacy and Cyber Security

Table 3 on page 12 addresses the same Meter Categories and the same Types of Communication that are addressed in Table 2 with regard to Risk to Health. But Table 3 addresses the Risk to Privacy and the Risk to Cyber Security. However, for purposes of easy comparison, the overall findings for Risk to Health from Table 2 are carried forward and placed in the Overall Risk column on the right side of on Table 3.

Table 3: Ranking Electricity Meters for Risk to Privacy and Cyber Security

(RED means risk to health. BLUE means risk to privacy. GREEN means risk to cyber security.)

Meter Category	Type of Communication			Risk to Privacy					Risk to Cyber Security			Overall Risk (5 is highest. Blank is lowest.)		
				Remote Access to Data Stream		Nature of Data			Remote Access to Meter					
	Criteria for Ranking →	wireless	wired	Number of People with Potential Remote Access	Ease of Gaining Remote Access	Timeliness	Granularity	Variety	Number of People with Potential Remote Access	Ease of Gaining Remote Access	Vulnerability of Meter to Being Harmed or to Doing Harm	Risk to Health	Risk to Privacy	Risk to Cyber Security
SMART METER Digital Advanced Metering Infrastructure (AMI)	WAN/ HAN	✓		4	5	5	5	5	4	5	5	5	5	5
	Internet cable/fiber		✓	5	2	5	5	5	5	2	5	2	4	4
	Telephone landline		✓	1	1	1	5	5	1	1	5	2	3	3
Digital Automated Meter Reading (AMR)	Bubble Up	✓		3	5	5	5	2	3	5		4	4	
	Wake Up	✓		2	5	1	1	2	2	5		3	2	
	Internet cable/fiber		✓	5	2	5	5	2	5	2		2	4	
	Telephone landline		✓	1	1	1	1	2	1	1		2	1	
Basic Digital Electronic Meter	None					1	1	1				2		
Analog Meter (plus wireless digital electronics)	Bubble Up	✓		3	5	5	5	2	3	5		4	4	
	Wake Up	✓		2	5	1	1	2	2	5		3	2	
Traditional Analog Meter	None													

Criteria for All Rankings

There are many characteristics of each electricity meter that affect the risk that it poses to health, to privacy, and to cyber security. Unfortunately, information is not publically available for many of those characteristics. The characteristics for which some information is available, and which I have selected for use here, are shown in the column headings in Table 3.

Legitimate disagreement about the risk rankings is entirely understandable if only because there are many different configurations possible for even a single model of an electricity meter, and because individual judgment has to be exercised to produce any risk rankings. Even so, my hope is that the overall rankings presented here will be useful to identify at least:

- the type of meter that presents the highest risk
- the type of meter that presents the lowest risk
- the types of meters that fall in between, even if there is disagreement about the relative rankings of those in between.

Here are the criteria that I have used for ranking risk in each of the three risk categories:

Risk to Health

Risk to Health is higher when the RF radiation exposure produced is higher. And the RF radiation exposure is higher when the Peak RF Power Output is higher, and when the number of bursts of RF radiation per day is higher, other factors being equal.

Peak RF Power Output

Risk ↑	High Peak RF Power Output (1 watt)
	Medium Peak RF Power Output (100 milliwatts)
	Low Peak RF Power Output (1 milliwatts)
	no RF Power Output

When the Peak RF Power Output was unknown, as it was for Digital Electronics and Switching Power Supply in Table 2 on page 6, I made an educated, but qualitative, guess, specifically "very low" and marked that entry "unknown".

Number of Bursts of Radiation per Day

Risk ↑	High number of bursts of RF radiation per day (more than 10,000 per day)
	Medium number of bursts of RF radiation per day (There were no meters in this group, so I did not define a specific level.)
	Low number of bursts of RF radiation per day (8 per month)
	No burst of radiation, ever

When the number of bursts of radiation per day was unknown, as it was for the Home Area Network (HAN) in Table 2 on page 6, I made an educated, but qualitative, guess of "high" and marked the entry "unknown".

Number of People Exposed to Radiation

The Risk to Health is also increased, in a societal sense, when more people are exposed to the radiation. The number of people that are exposed to the radiation is greater when the Peak RF Output Power is higher, because the size of the region exposed to the radiation increases, encompassing more people. So the Peak RF Output Power plays at least two roles in risk, increasing both the risk to each individual, and the number of individuals at risk. Thus, the heading "Number of People Exposed to Radiation" does not appear explicitly in Table 2 on page 6.

Risk ↑	Largest number of people is reached with High Peak RF Power Output (1000 milliwatts).
	Medium number of people is reached with Medium Peak RF Power Output (100 milliwatts).
	Smallest number of people is reached with Low Peak RF Power Output (1 milliwatt).
	No people are reached with zero Peak RF Power Output.

Risk to Privacy

The criteria that affect the risk to privacy are different for different "audiences" for the data. By the *audience* I mean the people who are obtaining and exploring the data and thus invading someone's privacy. Here, I consider two different audiences:

- people *outside* of the electric power company
- the electric power company itself.

For people outside of the electric power company, the risk to privacy can be viewed as dependent on two categories of criteria:

- remote access to the data stream
- nature of the data.

But for the electric power company, which has full access to all of the data produced by its electricity meters, access is not an issue. Therefore, only the nature of the data is relevant here.

I considered what the electric power company might do with the data that it gathers, such as provide it to others outside the electric power company, which is an acknowledged public concern. That is an unknown that I could not see how to score here. But if this should occur, it would clearly be a monumental concern.

Table 3 on page 12 can be used to assess the risk to privacy from both audiences because the criteria have been separated into the two major categories just described. However, the overall "Risk to Privacy", as reported on the right side of that table, is reflective of both categories and thus assumes that the audience is the people outside of the electric power company. Inspecting the risk rankings under "Nature of the Data" alone, the overall "Risk to Privacy" from the electric power company can be seen to be equally as high, or higher, than that from the people outside the electric power company.

Remote Access to the Data Stream

The risk to privacy increases with the number of people who can potentially gain access, and with the ease with which those people can actually gain access to that data stream. By *access* to the data stream, I mean access through the system of which the meter is a part, not access obtained by physically cutting into a wire, a cable, or a fiber.

Number of People with Potential Remote Access to the Data Stream

Risk ↑	Wired Internet data stream (both cable and fiber optic) provides the largest number of people with potential for remote access to data stream.
	Wireless data stream provides an intermediate number of people with potential for remote access to data stream.
	Wired telephone landline provides the smallest number of people with potential for remote access to data stream.
	none

When "none" applies there is no risk to privacy, no matter how the other criteria related to privacy are ranked. This case occurs for the Traditional Analog Mechanical Meter with No Wireless Communications Capability, and for the Basic Digital Electronic Meter.

Ease of Gaining Remote Access to the Data Stream

The risk to privacy also increases with the ease with which the people with potential remote access can actually gain access to the data stream.

Risk ↑	Wireless data stream is the easiest to which to gain remote access because it travels through the air.
	Wired Internet data stream (cable or fiber optic) is less easy to which to gain remote access.
	Wired telephone landline is the least easy to which to gain remote access.
	no remote data stream to access

When there is no remote data stream to access, there is no risk to privacy, no matter how the other criteria related to privacy are ranked. This case applies only for the Traditional Analog Mechanical Meter with No Wireless Communications Capability, and for the Basic Digital Electronic Meter, both of which have no wireless or wired communications capability.

Nature of the Data

The characteristics of the data affect the degree to which it is useful for invading privacy.

Timeliness

Data that are more timely are more likely to be useful for invading privacy.

Risk ↑	Timely data are more useful for invasion of privacy.
	Old data are less useful for invasion of privacy.

Granularity

Data that are more highly time resolved (more granular) are more likely to be useful for invading privacy.

Risk ↑	Highly granular (highly time-resolved) data are more useful for invasion of privacy.
	Less granular (less time-resolved) data are less useful for invasion of privacy.

Variety

Higher numbers of types of data are more likely to be useful for invading privacy.

Risk ↑	More data types are more useful for invasion of privacy.
	Fewer data types are less useful for invasion of privacy.

Risk to Cyber Security

Note that I use the phrase "cyber security" in a very limited sense in this document. The phrase refers to the security of the meter from external signals

- that can disrupt the meter itself (such as, by changing its readings or by changing its internal programming)
- that can cause the meter to do damage outside of itself (such as, by shutting down all power to the customer).

I have not considered cyber security in the sense that the meter might serve as a gateway to the network of which the meter is a part, and do damage through that network. Whether that is possible is beyond my knowledge. Certainly, if that is possible, that would be a monumental concern.

Nor have I considered cyber security in the sense that the community-based transmitters/receivers of the electric power company might serve as gateways to the network. Again, that is beyond my knowledge. If possible, that would also be a monumental concern.

Number of People with Potential Remote Access to the Meter

The risk to cyber security increases with the number of people who have potential remote access to the meter.

Risk ↑	Wired Internet data stream (both cable and fiber optic) provides the largest number of people with potential remote access to the meter.
	Wireless data stream provides an intermediate number of people with potential for remote access to the meter.
	Wired telephone landline provides the smallest number of people with potential for remote access to the meter.
	none

When "none" applies there is no risk to cyber security, no matter how the other criteria related to cyber security are ranked. This case occurs only for the Traditional Analog Mechanical Meter with No Wireless Communications Capability, and for the Basic Digital Electronic Meter.

Ease of Gaining Remote Access to the Meter

The risk to cyber security increases with the ease of gaining access to the meter remotely.

Risk ↑	Meter with wireless communications is the easiest to access remotely because all signals travel through the air.
	Meter with wired Internet communications (cable or fiber optic) is less easy to access remotely.
	Meter with wired telephone landline communications is the least easy to access remotely.
	Meter with no means of remote access.

When there is "no means" of remote access, there is no risk to cyber security, no matter how the other criteria related to cyber security are ranked. This case applies only for the Traditional Analog Mechanical Meter with No Wireless Communications Capability, and for the Basic Digital Electronic Meter, both of which have no wireless or wired communications capability.

Vulnerability of Meter to Being Harmed or to Doing Harm

The risk to cyber security increases with the vulnerability of the meter to being harmed or to doing harm.

Risk ↑	Meter has an internal shutdown switch. (That switch is always remotely controllable whenever present.)
	Meter has no internal shutdown switch.
	Meter can be accessed but has no ability to act on incoming signals.

Risk ↑	Meter has internal software that can be remotely reprogrammed.
	Meter has no internal software, or has internal software that cannot be remotely reprogrammed.
	Meter can be accessed but has no ability to act on incoming signals.

If the meter cannot act on incoming signals, it is not vulnerable in the sense meant here. Several of the meters addressed in this document appear to be invulnerable, in this sense, as shown in Table 3 on page 12.

Limitations to this Analysis

There are many limitations to this analysis:

- The variety of electricity meters available is so great that not all of them could be addressed here.
    - That variety is increased by the availability of hardware options and software programmability for some of the meters.
    - Some of the hardware options can be implemented not only by the manufacturers of the meters, but also by the electric power companies, both before installation and after installation.
    - Some of the software programmable capabilities can be exercised not only before installation but also after installation, and even *remotely* after installation and thus invisibly to the customer.

- The risk presented by a given meter is highly dependent on the options included in it. So the generalizations made here for a given category of meters may apply to a greater or a lesser degree depending on those options.
- New meters are being created all the time.
- It is impossible to obtain all of the information pertinent to assessing the risk of a given meter to health, privacy, and cyber security, for several reasons:
    - Some of the information needed is regarded as confidential or proprietary
        - by the manufacturers of the meters
        - by the testing laboratories that prove the compliance of the meters with the regulations of the Federal Communications Commission
        - by the electric power companies that buy and install the meters for their customers.
    - Some manufacturers and electric power companies decline to provide information about the meters, even if that information is not formally considered confidential or proprietary. No reason is usually given.
    - Some of the information needed may never have been generated.
    - Even for information that exists, there may be no legal requirement for that information to be revealed to the public, short of a court order. Such a court order has been employed in at least one instance, and provided vital information about Wireless Smart Meters that it had not been possible to locate previously.[4]

Closing

In spite of the limitations to this analysis, I hope that this document gets close enough to capturing the principal distinctions among the many types of meters addressed here to provide some useful perspective on their relative risk. Perhaps this document will motivate those who know more about the meters than I do to develop their own ranking.

[4] Pacific Gas and Electric Company's Response to Administrative Law Judge's October 18, 2011 Ruling Directing It to File Clarifying Radio Frequency Information, page 5. (http://emfsafetynetwork.org/wp-content/uploads/2011/11/PG&EDataQuestionsAlternatives_11-11-11sm.pdf)

EXHIBIT M-1

ADA Disability Definition

How is Disability Defined?

Disability is defined as a physical or mental impairment that substantially limits one or more major life activities. A physical impairment is a physiological condition, cosmetic disfigurement, or anatomical loss that affects one or more of the following body systems: neurological, musculoskeletal, special sense organs, respiratory, speech organs, cardiovascular, reproductive, digestive, genito-urinary, hemic and lymphatic, skin, and endocrine.

A mental impairment is a mental or psychological disorder such as mental retardation, organic brain syndrome, emotional or mental illness, and specific learning disabilities.

The ADA does not list diseases or conditions that are considered disabilities, however it does list those which are NOT included. Not covered under the ADA are homosexuality, bisexuality, transvestism, transsexualism, compulsive gambling, kleptomania, pyromania, pedophilia, exhibitionism, voyeurism, gender identity disorders not resulting from physical impairments, other sexual behavior disorders and psychoactive substance use disorders resulting from the current illegal use of drugs. The ADA does not cover individuals who are currently engaging in the illegal use of drugs.

A short-term condition such as a broken limb generally is not a disability. The test is whether the impairment substantially limits one or more major life activities and should be determined by examining the extent, duration and impact of the impairment.

The EEO considers the following as major life activities: walking, seeing, speaking, hearing, breathing, learning, performing manual tasks, caring for oneself, working, sitting, standing, lifting, reaching, thinking, concentrating, interacting with others, and sleeping.

EXHIBIT M-2

Recognition of the Electromagnetic Sensitivity as a Disability Under the ADA

Recognition of the Electromagnetic Sensitivity as a Disability Under the ADA
The Architectural and Transportation Barriers Compliance Board (Access Board) is the Federal agency devoted to the accessibility for people with disabilities. The Access Board is responsible for developing and maintaining accessibility guidelines to ensure that newly constructed and altered buildings and facilities covered by the Americans with Disabilities Act and the Architectural Barriers Act are accessible to and usable by people with disabilities. In November 1999, the Access Board issued a proposed rule to revise and update its accessibility guidelines. During the public comment period on the proposed rule, the
Access Board received approximately 600 comments from individuals with multiple chemical sensitivities (MCS) and electromagnetic
sensitivities (EMS).

The Board has taken the commentary very seriously and acted upon it. As stated in the Background for its Final Rule Americans with
Disabilities Act (ADA) Accessibility Guidelines for Buildings and
Facilities; Recreation Facilities that was published in September 2002:

"The Board recognizes that multiple chemical sensitivities and
electromagnetic sensitivities may be considered disabilities under the ADA if they so severely impair the neurological, respiratory or other functions of an individual that it substantially limits one or more of the individual's major life activities. The Board plans to closely examine the needs of this population, and undertake activities that address accessibility issues for these individuals".

Following its recognition of electro sensitivity and its declaration of commitment to attend to the needs of the electromagnetic sensitive, the Access Board contracted the National Institute of Building Sciences (NIBS) to examine how to accommodate the needs of the electro sensitive in federally funded buildings. In 2005 the NIBS issued a report.

The link for the report:
http://web.archive.org/web/20060[illegible]/ica.nibs.org/docs_project.pdf
From Report (page 11):

Electromagnetic Fields
For people who are electromagnetically sensitive, the presence of cell phones and towers, portable telephones, computers, fluorescent lighting, unshielded transformers and wiring, battery re-chargers, wireless devices, security and scanning equipment, microwave ovens, electric ranges and numerous other electrical appliances can make a building inaccessible.

The National Institute for Occupational Safety and Health (NIOSH) notes that scientific studies have raised questions about the possible health effects of EMF's. NIOSH recommends the following measures for those wanting to reduce EMF exposure – informing workers and employers about possible hazards of magnetic fields, increasing workers' distance from EMF sources, using low-EMF designs wherever possible (e.g., for layout of office power supplies), and reducing EMF exposure times (11).

Dafna Tachover

http://www.electrosmogprevention.org/smart-meter-resources-links/ada-accommodations-info/recognition-of-the-electromagnetic-sensitivity-as-a-disability-under-the-ada/

EXHIBIT M-3

Smart Meter Harm

Overbilling, fires, health problems, inaccuracy, hacking & cybersecurity, interference, privacy loss, and more....

Federal agency advocates for the EMF-disabled and reducing electromagnetic fields in buildings — the 2005 U.S. National Institute of Building Sciences Indoor Environmental Quality report

Smartmeterharm.org

Posted on January 26, 2017

Introduction

The Architectural and Transportation Barriers Compliance Board (Access Board) is an independent federal agency devoted to accessibility for people with disabilities. The Access Board is responsible for developing and maintaining accessibility guidelines to ensure that newly constructed and altered buildings and facilities covered by the Americans with Disabilities Act and the Architectural Barriers Act are accessible to and usable by people with disabilities. In November 1999, the Access Board issued a proposed rule to revise and update its accessibility guidelines. During the public comment period on the proposed rule, the Access Board received approximately 600 comments from individuals with multiple chemical sensitivities (MCS) and electromagnetic sensitivities (EMS). They reported that chemicals released from products and materials used in construction, renovation, and maintenance of buildings, electromagnetic fields, and inadequate ventilation are barriers that deny them access to most buildings.

...

There are a significant number of people who are sensitive to chemicals and electromagnetic fields. Surveys conducted by the California and New Mexico Departments of Health and by medical researchers in North Carolina found 16 to 33 percent of the people interviewed reported that they are unusually sensitive to chemicals, and in the California and New Mexico health departments' surveys 2 percent to 6 percent reported that they have been diagnosed as having multiple chemical sensitivities. C. Miller and N. Ashford, "Multiple Chemical Intolerance and Indoor Air Quality," in Indoor Air Quality Handbook Chapter 27.8 (McGraw-Hill 2001). Another California Department of Health Services survey has found that 3 percent of the people interviewed reported that they are unusually sensitive to electric appliances or power lines. P. LeVallois, et al., "Prevalence and Risk Factors of Self-Reported Hypersensitivity to Electromagnetic Fields in California," in California EMF Program, "An Evaluation of the Possible Risks From Electric and Magnetic Fields (EMFs From Power Lines, Internal Wiring, Electrical Occupations and Appliances, Draft 3 for Public Comment, April 2001" Appendix 3 (http://www.dhs.ca.gov/ehib/emf/RiskEvaluation/riskeval.html).

Individuals with multiple chemical sensitivities and electromagnetic sensitivities, who submitted written comments and/or attended the public information meetings on the draft final rule, requested that the Access Board include provisions in the final rule to make buildings and facilities accessible for them.

The Board has not included such provisions in their rules, but they have taken the commentary very seriously and acted upon it. As stated in the Background *[this is in the General Issues section]* for its Final Rule Americans with Disabilities Act (ADA) Accessibility Guidelines for Buildings and Facilities; Recreation Facilities: http://www.access-board.gov/recreation/final.htm [no longer working link – see below – 1]

"The Board recognizes that multiple chemical sensitivities and electromagnetic sensitivities may be considered disabilities under the ADA if they so severely impair the neurological, respiratory or other functions of an individual that it substantially limits one or more of the individual's major life activities. The Board plans to closely examine the needs of this population, and undertake activities that address accessibility issues for these individuals.

The Board plans to develop technical assistance materials on best practices for accommodating individuals with multiple chemical sensitivities and electromagnetic sensitivities. The Board also plans to sponsor a project on indoor environmental quality. In this project, the Board will bring together building owners, architects, building product manufacturers, model code and standard-setting organizations, individuals with multiple chemical sensitivities and electromagnetic sensitivities, and other individuals. This group will examine building design and construction issues that affect the indoor environment, and develop an action plan that can be used to reduce the level of chemicals and electromagnetic fields in the built environment."

This report and the recommendations included within are a direct outgrowth from that public comment process. The Access Board contracted with the National Institute of Building Sciences

Recommendations for Accommodations

People with chemical and/or electromagnetic sensitivities can experience debilitating reactions from exposure to extremely low levels of common chemicals such as pesticides, cleaning products, fragrances, and remodeling activities, and from electromagnetic fields emitted by computers, cell phones, and other electrical equipment.

The severity of sensitivities varies among people with chemical and/or electromagnetic sensitivities. Some people can enter certain buildings with minor accommodations while others may be so severely impacted that they are unable to enter these same spaces without debilitating reactions. Furthermore tolerances to specific exposures can vary greatly from one individual to the next.

...According to the Americans with Disabilities Act (ADA) and other disability laws, public and commercial buildings are required to provide reasonable accommodations for those disabled by chemical and/or electromagnetic sensitivities.

...

The Committee acknowledges that while the scientific evidence may be inconclusive about whether ambient electromagnetic fields pose a substantial health risk to the general population, the presence of EMF is an access barrier for people who are electromagnetically sensitive. Therefore, the Committee recommends that measures be taken to reduce EMF whenever possible in order to increase access for these individuals as well as taking a precautionary approach to protecting the health of all.