March 12, 2024

To: Bonner County Commissioners

From: Joan Esnayra, Ph.D.

Re: Request for Reasonable Accommodation under the Americans with Disabilities Act (ADA) Title II

Dear Bonner County Commissioners,

I am a person with a disability as defined by the ADA.

My disability is termed Electrical Hypersensitivity Syndrome (EHS). See attached doctor's letter from Gunnar Heuser, M.D., Ph.D., for a description of my condition, its symptoms, functional impairments, and how it affects my neurological and cardiovascular systems.

My disability is recognized by the Social Security Administration (see attached disability benefits Notice of Award) and the Internal Revenue Service.

Bonner County is subject to Title II of the ADA because it is a local government entity.

As a disabled individual, I am requesting a reasonable accommodation (RA) to the policies and procedures related to a specific Planning Department activity, namely, the review, deliberation, and decision-making activities related to CUP0022-23.

In making this RA, what I seek is an equal opportunity to benefit from the cell phone services that may be provided by such a proposed cell tower.

What does the verb 'to benefit' mean in this context? In order explore this question, let's ask, "What is the opposite of the verb 'to benefit'?" Here are a few examples of antonyms for the verb 'to benefit':

> to suffer; to decline; to hurt; to worsen; to regress; to degenerate; to deteriorate; to languish; to crumble' to fade; to devolve; to become worse; to break down; to experience hardship; to fall apart; to get worse; to go backwards; to go downhill; to be at disadvantage; to be in decline; to fall to pieces.
>
> REF: https://www.wordhippo.com/what-is/the-opposite-of/benefit.html

Let's also consider the word 'Discrimination'. It means 'unequal treatment,' which, in its generic sense is not illegal. Disability discrimination, on the other hand, is unequal treatment based upon a person's disability, and that is illegal.

If a disabled person is not afforded an equal opportunity to benefit from cell tower services, the placement of which is dependent upon, and determined by, the County's legal authority, as mandated by the State of Idaho, the Bonner County Code, the Bonner County Comprehensive Plan, and the 1996 Telecommunications Act (TCA), then, we have a discrimination situation that must be rectified in order to be in compliance with applicable law(s).

In the attached letter, Dr. Martin Pall, who is a world expert in the science of EMF exposures, provides expert analysis of how the proposed cell tower will impact me, specifically. These impacts reflect the antonyms listed above. Nothing in Dr. Pall's expert testimony suggests that the proposed cell tower described in CUP0022-23 will afford me an *equal* opportunity to benefit from the cell phone services. As the saying goes, "Houston, we have a problem."

In addition to the frequent symptoms mentioned in Dr. Heuser's and Dr. Pall's letters, I have experienced three heart attacks and five minor strokes from exposure to electromagnetic radiation. I am alive today solely by the God's Grace, and I am grateful for His Mercy, as I struggle daily with this challenging condition that has rendered me unable to earn a living as a scientist, as I did for most of my life.

I do my best to take care of myself by living remotely, practicing avoidance from radio frequency exposure, and shielding myself from it, whenever feasible. Nonetheless, a 140-foot cell tower erected 853 yards from my property and 40 effective feet above my head will likely kill me. This is not hyperbole.

Commissioners, please consider the gravity of the decision you are about to make regarding CUP0022-23. The ADA *requires* you to make a disability accommodation that will prevent my further incapacitation (in a best-case scenario) and my death (in a worst-case scenario). Is it too much to ask my county government, "Please do not murder me?"

I purchased this five-acre parcel in the woods of Elmira so that I might become maximally self-sufficient, thus minimizing the necessity to make trips into town that always make me ill for hours after I return home. I garden to grow my food, and I raise livestock to grow my meat. A 140-foot cell tower erected 853 yards from my property will make it impossible for me to enjoy the zoned-use of my property. I will not be able to garden or tend my livestock. This would be, in essence, a taking of property.

Furthermore, I do not have the resources, at this point, to move from my property. I had to spend everything my husband left me, after he died, to create this small (primitive) homestead. I live in a trailer without running water. I have no shower or laundry facilities. These luxuries are simply beyond my budget. Nor do I have the physical strength at this point to move elsewhere.

Under the ADA, RA is intended to be an interactive process. Thus, for the county and this disabled individual to identify an appropriate course of action (i.e., RA) to the exercise of the County's authority relative to CUP0022-23, we are required to engage in a productive dialogue to explore possible solutions to rectify the obvious discrimination that will afflict me by placement of this tower *as currently described* in CUP0022-23.

I have no desire to restrict others' ability to benefit from cell phone coverage, which, most already enjoy here in Elmira. Yet, at this juncture in the CUP review process, we have *no evidence of a significant gap in coverage* in this location. Nor do we have any analysis of *'least intrusive means'* relative to proposed cell tower placement. These issues <u>should have been addressed</u> at the Planning Department staff level, and at the first public hearing, as the Bonner Code (12-488.C) dictates, but it wasn't.

I testified at the February 7, 2024, CUP0022-23 public hearing. My testimony was delivered over Zoom, because I can no longer enter the County Building due to the extreme WIFI levels in the building. In my testimony, I informed the Hearing Examiner, and others in attendance, about my EHS disability, history of heart attacks and strokes from EMR exposure. I explained how this proposed tower will harm me further, if not kill me.

The Bonner Code 12-223 states,

> "To grant a conditional use permit, the Zoning Commission or Hearing Examiner must find there is *adequate evidence* showing that the proposal is in accordance with the general and specific objectives of the comprehensive plan and this title, and that *the proposed use will neither create a hazard nor be dangerous to persons on or adjacent to the property.* [italics added for emphasis] (Ord. 501, 11-18-2008; amd. Ord. 661, 3-18-2022; Ord. 681, 10-12-2022; Ord. 682, 10-12-2022)

Please explain to me how the Hearing Examiner, after hearing my testimony, came to the following conclusion in her Decision Letter on CUP0022-23 issued on February 13, 2024:

> **"Conclusion 3:** The proposed use **will not** create a hazard or will not be dangerous to persons on or adjacent to the property.
>
> The decision is based upon the evidence submitted up to the time the Staff Report was prepared, and testimony received at this hearing."

Commissioners, these statements are untrue. The word adjacent can mean adjoining, and it can also mean nearby. Joe Bindert's property adjoins the proposed site; mine is nearby. Is the legitimate hazard to my person articulated by Dr. Martin Pall's expert testimony invalidated by a subtle (and unarticulated) preference of definition?

Is it in accordance with the general and specific objectives of the comprehensive plan and the Bonner Code to facilitate harm (or death) to residents on nearby properties who have a disability that will be directly and uniquely impacted by the proposed activity?

I am aware of the stipulations of the TCA relative to cell tower placement, just as I am familiar with the tenants of the ADA. Is this a situation in which two federal laws are seemingly in conflict?[1] Perhaps the answer is above our respective paygrades. I am not an attorney who specializes in cell tower laws and regulations. Are you?

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[1] *ADA is the legitimate remedy for EHS* sufferers. "Given that the FCC has declined to regulate RFs with respect to biological (non-thermal) effects, especially any impact to those that might be hypersensitive, the ADA provides a legitimate remedy to any unusual harm that might be caused to a small subset of individuals exposed to something that is

Appropriate expertise may be needed to navigate these complex regulatory waters in the absence of a contemporary wireless ordinance appropriate to these situations, something Bonner County needed on February 7, 2024. For me, CUP0022-23 is a matter of life and death.

It is not sufficient to say, "Let's wait and see what happens in other jurisdictions." As the saying goes, "Don't get by riding someone else's horse. Ride your own horse." We need a clear set of modern procedures for handling cell tower applications that are appropriate to the times we live in. Please hire an expert and 'get 'er done.' Delay invites liability rather than avoids it.

While I worked on this appeal project for the past month, it was necessary to reach out to neighbors in the Elmira area whom I did not know. I was surprised to find quite a few residents, here, tucked away in the woods, many off-grid, who live with EHS and who moved to this remote area to escape the EMR exposure of more densely populated local areas. With permission, I have included a doctor's letter from one such resident so that you understand that your decision, regarding CUP0022-23, will affect more disabled individuals than just me. Now, we have found each other; we can work together.

Commissioners, I have provided you with what you need to understand your obligations under the ADA. As such, you are now empowered. If, for some reason, you decide to vacate the CUP0022-23 using your *existing authority*, independent of the ADA, then, my request for RA may be moot.

If you determine that the applicant did not satisfy Bonner County CUP criteria, and the applicant subsequently sues the County, then, you will be judged on your application of those criteria. Our appeal document provides clarity in that regard. Unfortunately, this document is provided at great expense to this taxpayer. I was saving for a chicken coop.

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generally, genuinely, and legitimately accepted as safe for the wider population." (p. 18) USDC Massachusetts: Case 4:15-cv-40116-TSH

In conclusion, it is my hope, and my prayer, that the imminent threat to my life and livelihood represented by CUP0022-23 will cease to be a threat in the very near future.

As the saying goes, "Do what's right, and let the consequences fall on God."

My life is in your hands.

Respectfully,

Joan Esnayra

Joan Esnayra, Ph.D.

[redacted]

Seven Attachments:
Letter from Gunnar Heuser, MD., Ph.D.
Social Security Disability Notice of Award
Letter from Martin Pall, Ph.D.
Book Chapter on EHS by Martin Pall, Ph.D.
Graphic: Aerial View Distance between proposed cell tower and Esnayra residence (853 yards)
Graphic: Elevation View from top of proposed cell tower to Esnayra residence (~40 feet)
Letter from William Billica, M.D., on behalf of Elmira resident, Johany Franz.

Social Security Administration
Retirement, Survivors, and Disability Insurance
Notice of Award

Western Program
Service Center
P.O. Box 2000
Richmond, California 94802-1791
Date: November 5, 2023
BNC#: 23M1827J35077-A

0001295 00006834 2 MB 0.561 1031MACTR5PB T32 P6

JOAN G ESNAYRA
[redacted]
SANDPOINT ID 83864-1410

02015IK7500292 0001295 0000000100

We are writing to let you know that you are entitled to monthly disability benefits from Social Security beginning May 2022.

Your Benefits

The following chart shows your benefit amount(s) before any deductions or rounding. The amount you actually receive may differ from your full benefit amount. When we figure how much to pay you, we must deduct certain amounts, such as Medicare premiums and worker's compensation offset. We must also round down to the nearest dollar.

Beginning Date	Benefit Amount	Reason
May 2022	$ 1,849.80	Entitlement began
December 2022	$ 2,010.70	Cost of living adjustment

What We Will Pay

We pay Social Security benefits for a given month in the next month. For example, we pay Social Security benefits for March in April.

- Your first payment is for $27,826.50.
- This is the money you are due through October 2023.

GUNNAR HEUSER, M.D., Ph.D., F.A.C.P., F.A.C.F.E., B.C.F.E.
NeuroMed and NeuroTox Associates
A Medical Group

Fellow, American College of Physicians
Diplomate, American College of Forensic Examiners

Fellow, American EEG Society
Diplomate (McGill University), Internal Medicine

NEUROTOXICOLOGY

IMMUNOTOXICOLOGY

August 18, 2022

Re: ***JOAN G. ESNAYRA***

To Whom It May Concern:

This letter is written in support of Joan G. Esnayra. She is 58 years old and suffers from severe and disabling sensitivity to electromagnetic and transient electrical fields.

Electromagnetic fields including dirty electricity are ever present in our society and are generated by cell phone towers, cell phones, microwaves, Wi-Fi, smart meters, and also high voltage transients.

Upon exposure to the above, Ms. Esnayra has developed significant and at times disabling symptoms such as debilitating headaches, with stabbing head pain, confusion, and inability to process information. In addition, she experiences chest and heart pain along with heart palpitations when exposed to even the smallest amounts of electromagnetic fields and dirty electricity.

Her symptoms started in 2012 and have continued unabated.

Joan radically changed her lifestyle and now practices strict avoidance and also shielding by using special garments and other protective devices.

Avoidance became a necessity since no treatment is available for her condition. She is being forced to live in isolation off the grid which has become necessary for her survival.

On account of her disability, Joan's husband, Dr. Craig Love, supported her financially until his death in 2017. To this end, in January 2014, they moved from urban Washington, DC. to a very remote area of BC, Canada, in order to accommodate her severe Electrical Hypersensitivity Syndrome. After Craig's death in 2017, Joan had to return to the U.S. and identify another (affordable) remote area in which to re-settle.

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Ph (310) 500-0041 Email: toxguns@netscape.net
emfdoc.com

Thus, it was necessary for her to purchase affordable raw land, in this case in Northern Idaho, and develop it so that she could live on-site in an off-grid trailer. For the past two years, she has developed the land to make it habitable as a necessary accommodation for her disability. She had to hire contractors to fell trees, install a gravel driveway, a septic system, a water well, etc. This is the reason for which she requests the disability exemption for early IRA withdrawals. She had no other choice but to draw down the funds she inherited from her husband, in order to create a safer, albeit primitive, place to live.

Her disease is expected to last her lifetime and therefore requires continuous avoidance and shielding.

In summary, Ms. Esnayra satisfies the following diagnoses of: toxic encephalopathy (349.82), non-ionizing radiation (W 90, and exposure to electric current (W 85-W 99).

As a physician and medical specialist, I certify that Joan Esnayra meets the IRC §72(m)(7) definition of disabled.

My qualifications can be viewed on my website and show that I have published peer-reviewed papers in the field of chemical and electromagnetic sensitivity. I have also been an invited speaker to international conferences on these topics. In. addition I am familiar with the scientific literature and I am in communication with international experts in the field.

Gunnar Heuser, M.D., Ph.D., F.A.C.P.

PERTINENT REFERENCES from Gunnar Heuser Curriculum Vitae

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ONLINE PRESENTATIONS

1. **Heuser, G., Heuser, SA. Multisystem impairment after EMF exposure, with special attention to the brain. International EMF Conference; September 5-7, 2019. Scotts Valley, California. Online.**
2. **Heuser, G, Heuser, SA. Functional brain scans of patients exposed to neurotoxic chemicals and/or EMF. International Electromagnetic Fields Medical Conference, Prevention, Diagnosis, and Treatment of EMF Associated Illnesses; January 28-31, 2021. Online.**

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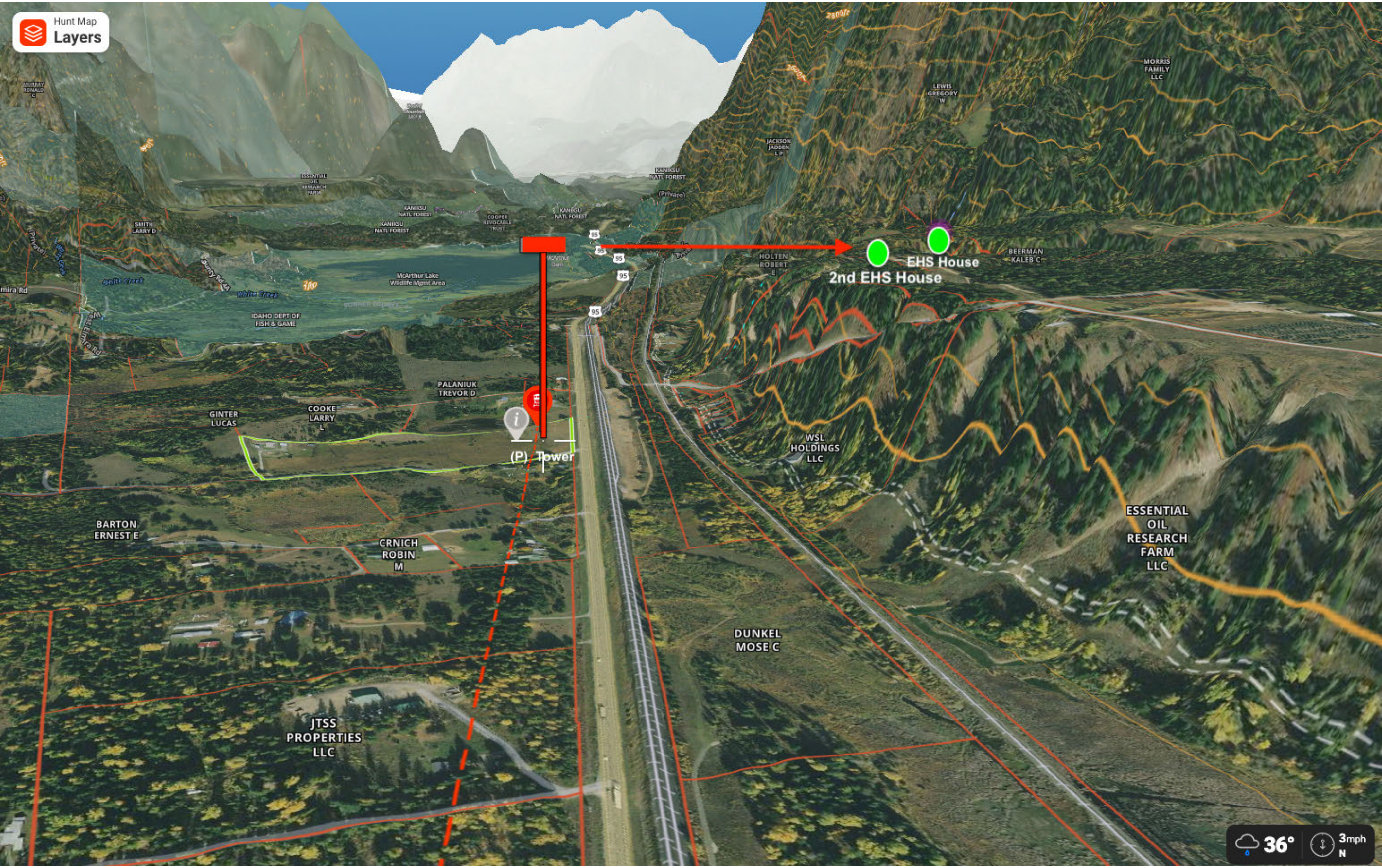
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Wildlife Mgmt Area
IDAHO DEPT OF
FISH & GAME
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CRNICH
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JTSS
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MOSE C
ESSENTIAL
OIL
RESEARCH
FARM
LLC
HOLTEN
ROBERT
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BEERMAN
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MORRIS
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LEWIS
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L P
SMITH
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KANIKSU
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White Creek
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853 yds
02/01/24, 4:49 PM
Elevation
2,260 ft
2,156 ft
Hide on map
Distance
↔ 853 yds
Elevation Gain
↗ 99 ft
Elevation Loss
↘ 0 ft
Share
Add to Folder
Edit
Delete
EHS Home
(P) 140' Tower
853yds
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Elmira Rd
Dancing Rd
Ellis Rd
Cindy Ln
95
HOLTEN ROBERT E
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GOW MICHAEL J
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BINDERT JOSEPH D
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JOHNSON VANESSA
DALY JAROD MICHAEL
PIXLER KORIN A
SCHWOERER WILLIAM A
HERCULES INVESTMENT SYNDICATE LLC
mapbox

March 11, 2024

To Whom This May Concern:

Joan G. Esnayra has alerted me to her concerns about the planned cell phone tower. She also emailed me copies of Dr. Gunnar Heuser's letter and diagrams showing the location and height of planned cell phone tower relative to where Joan Esnayra lives. I recently published a paper on electromagnetic hypersensitivity that is coming out as a chapter in a book on EMFs. It will be attached to this email.

It shows that there are five published studies that show that EHS is real and that using objective measures of sensitivity, people react in blinded studies (they don't know when they are being exposed) to EMFs at levels that do not impact most normal people. Therefore, EHS is a genuine hypersensitivity. That does not mean that everyone who claims sensitivity has EHS but it does mean that this sensitivity exists and impacts substantial numbers of people. The levels of sensitivity are quite variable, but once persons are sensitive, they are at great risk of becoming more sensitive as a consequence of EMF exposures.

If you look at the five published studies on EHS, you will see is that one of them was published by Dr. Gunnar Heuser. That study showed that a group of his EHS patients showed very high sensitivity in their brains as compared with normal controls, using fMRI to measure that sensitivity (or lack of sensitivity). Because each of Dr. Heuser's patients that he had previously diagnosed as having EHS showed very high level of brain sensitivity, this study showed several things.

1. That EHS is a real demonstrable sensitivity.
2. Dr. Heuser is able to reproducibly and reliably diagnose EHS because if he had not been able to do so, this study could never have been done!!
3. Therefore, Dr. Heuser is one of the best people in the world at diagnosing EHS.

Consequently when Dr. Heuser writes that:

"Upon exposure to the above, Ms. Esnayra has developed significant and at times disabling symptoms such as debilitating headaches, with stabbing head pain, confusion, and inability to process information. In addition, she experiences chest and heart pain along with heart palpitations when exposed to even the smallest amounts of electromagnetic fields and dirty electricity.

Her symptoms started in 2012 and have continued unabated.

Joan radically changed her lifestyle and now practices strict avoidance and also shielding by using special garments and other protective devices.

Avoidance became a necessity since no treatment is available for her condition. She is being forced to live in isolation off the grid which has become necessary for her survival."

Dr. Heuser is one of the most reliable physicians in the world to make these judgements.

In the most recent study on how far away people are impacted by cell phone tower radiation shows that effects extend to 400 meters – over a quarter mile away. Subhan F, Khan A, Ahmed S, Malik SN, Bakshah ST, Tahir S.. 2018 Mobile antenna's and its impact on human health. J Medical Imaging and Health Informatics. 8(66):11266 - 1273. doi: 10.1166//jjmihi.22018.2296.

But this is not a normal situation. First of all, cell phone tower antennae are designed to irradiate maximally in a horizontal direction. Therefore, because Joan Esnayra lives at almost the same altitude as that of the planned antenna, she will be exposed to MUCH HIGHER exposure, possibly 100 or more times the exposure that people would get exposed to lower down. Secondly Dr. Heuser has determined that she has a very high sensitivity such that she may be hundreds of times more sensitive than are normal people. And these two numbers act multiplicatively.

I have degrees in Physics and Biochemistry and Genetics from two of the top institutions in the world, Johns Hopkins and Caltech, and this combined expertise has been instrumental in allowing me to publish a series of very influential papers on how EMFs act in our bodies. My first paper published in 2013 has now been cited over 500 times (Google Scholar).

Martin L. Pall, PhD, Professor Emeritus, Washington State University

Martin L Pall

March 1, 2024

To: Whom It May Concern

Re: Johany Franz, [redacted]

This letter is written in support of Johany Franz. She is 64 years old and suffers from severe and disabling sensitivity to electromagnetic and transient electrical fields, and with Multiple Chemical sensitivity issues. She has proven genetic issues with her body detoxification pathways.

Electromagnetic fields including dirty electricity are ever present in our society and are generated by cell phone towers, cell phones, microwaves, Wi-Fi, and high voltage Transients.

Johany already had many symptoms from this when I first started treating her in 2006. She has radically altered her lifestyle and now practices strict avoidance and uses protective devices and supplement supports. She lives in a mostly remote area and off the grid for her survival. This condition is essentially disabling and she has developed her property to be able to survive. Just a brief trip into town will make her extremely ill for several days.

For these reasons, my letter is in support of Johany Franz request to not locate a cell tower anywhere near her property. At best any tower should be several miles from her location.

This is an accommodation for her disability.

Sincerely,

William Billica, M.D.

Mechanisms Producing Sensitization Including Elevated [Ca^{2+}]i; Specific Potential Tests for EHS; EHS Prevention and Treatment
By Martin L. Pall, PhD

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Abstract:

The primary target of EMFs in the body is the voltage sensor which controls the activation of voltage-controlled calcium channels (VGCCs) in the plasma membrane of cells, with low intensity EMFs producing large increases in [Ca^{2+}]i. EHS is a genuine sensitivity condition produce via hypersensitivity of the VGCCs to activation. That hypersensitivity is thought to be produced via three calcium stimulated protein kinases which increase the sensitivity of the VGCCs. Chronic elevation of EHS is also thought to involve activity of the NO/ONOO(-) cycle. Two simple specific tests for EHS are proposed here as are six different therapeutic approaches for EHS treatment.

Introduction:

Biology, including medicine, is incredibly complex. Consequently, one can often come up with many explanations of complex findings such as EHS but it is often difficult if not impossible to determine whether such an explanation has any merit or not, without a fundamental understanding of what biological mechanisms are involved. Consequently, an understanding of the mechanism of action of electromagnetic fields (EMFs) in the body is needed and how that mechanism of action can be impacted in order to produce its hypersensitivity. Accordingly, this paper is outlined as follows:

1. How electronically generated EMFs impact the cells of our bodies.

2. EHS studies showing that it is a genuine physiological sensitivity impacting the brain, other parts of the nervous system and also other tissues including the heart and immune system.

3. EMFs cause EHS and trigger symptoms both via VGCC activation; EHS appears to be caused by hypersensitivity of the VGCCs to activation.

4. Role of protein kinases and the NO/ONOO(-) cycle in producing chronic effects.

5. Two approaches towards developing a specific test for EHS and why such tests are so important.

6. Seven proposed approaches to EHS therapy and prevention.

How electronically generated EMFs impact the cells of our bodies.

Electronically generated EMFs are distinctly different from most natural EMFs. Electronically generated EMFs are coherent, being emitted with a particular frequency, vector direction, polarity and phase and are for those reasons, coherent producing strong electric forces and time-varying magnetic forces (Pall 2021a & b). It is those forces, placed on electrically charged chemical groups in the cells of our bodies, that produce biological effects. Chapter 7 entitled "Electromagnetic Induction" of Purcell, 1985 is focused on the fact that electric currents induce EMFs in the space around them. Most natural EMFs are composed of astronomical numbers of photons, each produced by a single quantum event, where the photons are emitted in different vector directions, with different polarities and phases, often but not always with different frequencies are therefore incoherent. As a consequence, most natural EMFs produce only miniscule electric forces, typically forces no greater than 20 times the forces produced by single photons. The EMFs that I am referring to in this article are all electronically generated coherent EMFs. These are the types of EMFs that are used in all wireless communication because wireless communication always involves both emitting and receiving antennae where the emitting antenna produces a coherent EMF which acts by placing forces on mobile electrons in the receiving antenna.

I want to make one thing clear – I am not the first prominent scientist to discuss the importance of coherence of electronically generated EMFs. I cited 10 papers in Pall 2021a that each discussed the importance of coherence in various contexts. However the central importance of coherence in generating the electric and time varying magnetic forces which produce, in turn, biological effects, as discussed below has not been widely recognized in the EMF literature. Furthermore, because the ICNIRP and other international and national "safety guidelines" are based on thermal (heating) effects rather than electric & magnetic forces, they are simply fraudulent.

The Pall 2013a cited 24 different studies each showing that low intensity EMF effects could be blocked or greatly lowered by voltage-gated calcium channel (VGCC) blockers. I have since published on six additional studies showing similar findings. Those studies each show that EMFs act predominantly by activating the VGCCs. Those findings have been widely recognized in the scientific literature as shown by the fact that 434 studies cited Pall, 2013a, at this writing, as shown by Google Scholar. The VGCCs are each controlled by a structure called the voltage sensor. The voltage sensor structure is discussed below and the electric charges on the voltage sensor are thought to the primary targets of the electric forces and time varying magnetic forces produced by coherent EMFs (Pall 2021a & b).

There are 11 different voltage controlled channels each controlled by a similar voltage sensor (Table 1) are each activated by low intensity EMFs. However the calcium channels produce most the EMF effects both in animals and in plants, so that to a first approximation, other ion channel effects can be ignored.

Table 1: Voltage Regulated Ion Channels in Animal and Plant Cells Each of Which Are Regulated by a Voltage Sensor and Activated By Low Intensity EMFs

Channels	Citation

L-type, T-type, N-type, P/Q-type voltage-gated calcium channels (VGCCs)	Pall, 2013
Voltage-gated sodium, potassium and chloride (anion) channels	Pall, 2018
Calcium-activated potassium channels (BKCa)	Pall, 2021a
TPC, GLR3.3, GLR 3.6 (in plants)	Pall, 2016b; unpublished

The EMF-induced forces act on the structure of the voltage sensor that is modeled below:

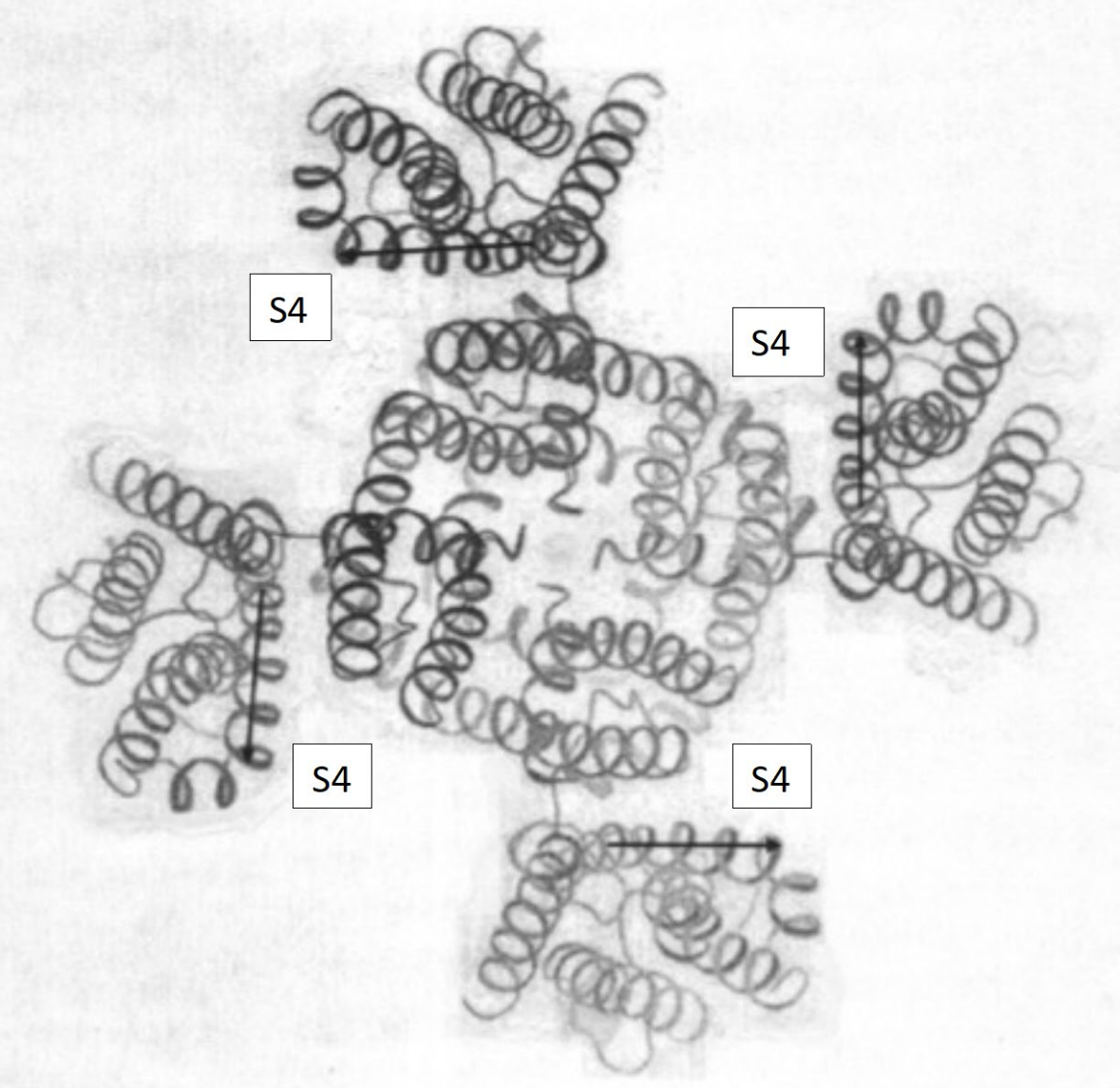

Figure 1 legend. The portion of the voltage regulated channels have a four domain structure with each domain containing six alpha helixes, designated S1 to S6. The S4 helixes in the VGCCs and other channels each contain circa 5 positive charges on arginine residue side chains. When EMFs or other forces activate these channels, they cause the S4 helixes to ratchet in the direction of the black arrows. Ratcheting involves at least 3 steps and may involve as many as 5 steps in the direction of the arrows. It is my opinion that the EMFs that are most effective in

channel activation are approximately perpendicular to the plasma membrane where the channel is located because perpendicular EMFs will produce similar forces on each S4 helix and each of them needs to ratchet out in order to activate the channel. When all four S4 helixes ratchet in the arrow direction, each will pull on the S5 and S6 helixes away from the center of the structure and after a period of a few microseconds, a conformational change can occur opening the channel in the center and allowing ions to flow. Below the structure shown (towards the cellular side of the plasma membrane is a contained aqueous phase and below that is another structure called the specificity filter which will determine what ions flow into the cell. The voltage sensor acts, therefore, independently of the ion specificity.

So why are the calcium channels the primary ones producing biological effects? Probably for two reasons. Intracellular calcium $[Ca^{2+}]i$ levels are usually maintained at levels circa 10^{-4} of the extracellular calcium levels such that there is often a 10,000-fold concentration gradient driving calcium into the cell and also circa a million-fold electrochemical gradient driving calcium into the cell. The second reason is because of the large importance of calcium influenced effects, see Fig. 2 below.

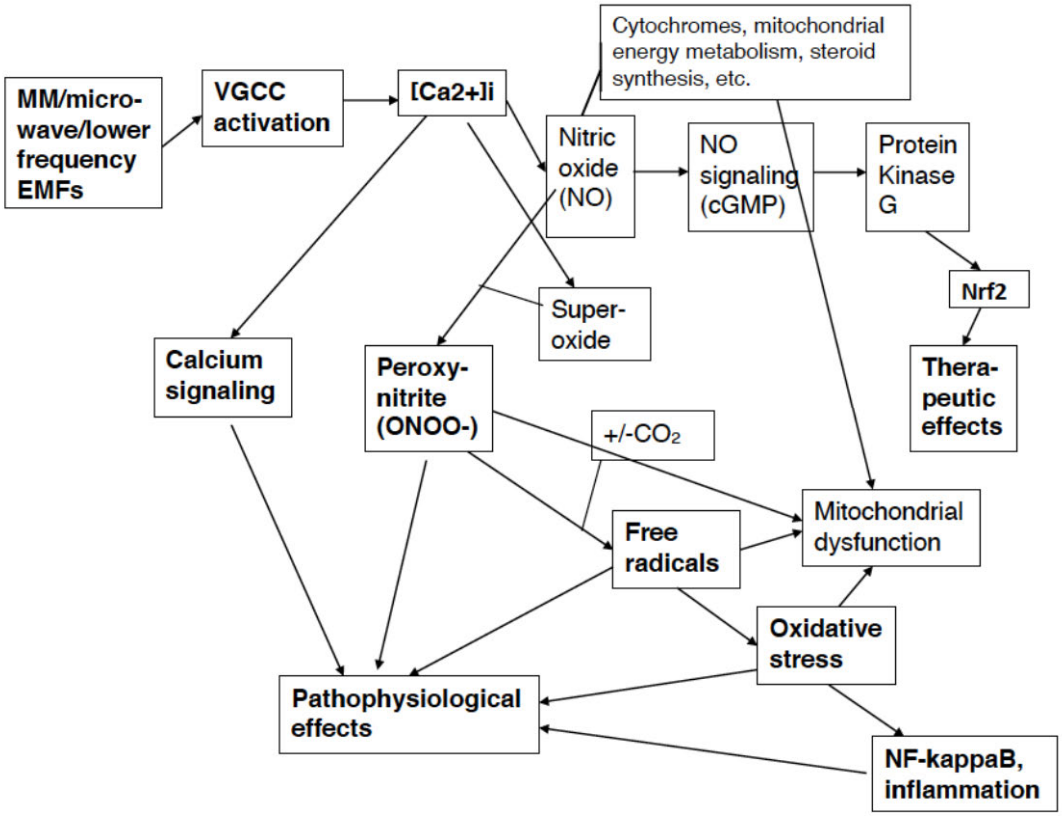

Figure 2 legend. The Figure and discussion here is taken from Pall, 2022 and earlier papers with permission. As shown (upper left), various frequencies of EMFs produce VGCC activation which produces increases in $[Ca^{2+}]i$ levels. Higher such increases produce pathophysiological effects largely through excessive calcium signaling and through the peroxynitrite/free radical/oxidative stress/NF-kappa B/inflammation pathway. Modest increases in $[Ca^{2+}]i$ levels act via nitric oxide (NO) signaling and increases in Nrf2 to produce therapeutic effects. However NO increases are NOT an unmixed blessing. NO is not only a precursor for

peroxynitrite, it also binds to and inhibits cytochromes including cytochrome oxidase and cytochrome P450s, all important enzymes (see top of Figure).

As can be seen from Fig. 2, The two main pathophysiological pathways of action following electronically generated EMF exposure are the excessive calcium signaling pathway and the peroxynitrite/free radical/oxidative stress/NF-kappa B/inflammation pathway. Both of these are involved in many but not all pathophysiological EMF effects. In contrast, relatively modest increases in $[Ca^{2+}]i$, act via nitric oxide (NO) signaling, cGMP and elevated Nrf2 to produce therapeutic effects. The therapeutic pathway and the peroxynitrite/oxidative stress/inflammation pathway produce opposite effects and as shown in Pall 2022, each of these pathways inhibit the other. One way of interpreting EHS is that EHS shifts the EMFs needed to move from the therapeutic pathway to the pathophysiological pathway to much lower exposure levels.

Each of the findings discussed above are essential for understanding EHS.

EHS studies showing that it is a genuine physiological sensitivity impacting the brain, other parts of the nervous system and also other tissues including the heart and immune system.

I'm going to discuss a number of studies, each of which provided clear evidence that EHS clearly occurs in some individuals who have reported that they react to EMF exposure. I'll start with the first published of these, the 1991 Rea et al study. In that study Dr. William Rea and his colleagues initially started with a group of 100 possible EHS patients and did preliminary studies on them using 3 min. low intensity, non-pulsed microwave EMFs. They measured five different objectively measurable potential response to exposure: Blood pressure; pulse rate, body temperature (thermoregulation), pupillary constriction and electrical resistance (measured by a polygraph). They identified 25 patients who showed highly consistent responses and compared them with normal controls in blinded studies. Each of these end points were objectively measurable. They found very highly consistent differences between the two groups. "EHS" people showed highly consistent changes in each of these 5 parameters with EMF exposure but not otherwise, whereas normals showed no changes with exposure.

Havas published three studies that were methodologically similar to Rea et al, 1991 but looked at very different biological end points. Havas, 2006 showed that both diabetes and multiple sclerosis effects were produced in EMF exposed apparent EHS patients. Havas 2008 followed up on the diabetes findings of Havas 2006, showing that brittle diabetes was produced by EMF exposures from dirty electricity. Havas et al 2010 added cardiac effects to the experimentally demonstrated EMF effects in EHS patients, showing that cordless phone radiation could produce instantaneous tachycardia when the phone was turned on, responses that were instantaneously turned off when the cordless phone radiation was turned off. Each of these studies involved objectively measured responses and blinded radiation exposures, responses that did not occur in normal controls.

In some ways, the Heuser & Heuser, 2017 study was the most important of these. It showed that in a series of 10 apparent EHS patients, that blinded low intensity EMF exposures produced hypersensitive activation of certain neural circuits in the brain, as measured by fMRI, responses

that were missing in normal controls. This directly showed hypersensitivity in the brains of EHS patients but not in controls.

The McCarty et al 2011 paper was done using a specific EHS patient who had apparent highly consistent perceived responses to low intensity EMFs. Unlike the other five studies, discussed above which each measured objectively measurable EMF responses in the body, the McCarty paper looked only at perceived responses. It was done, as it had to be, using double blinded conditions so that neither the experimenter nor the patient knew when EMF exposure were occurring. McCarty et al, 2011 also showed that this specific patient was a genuine EHS sufferer, responding with high level consistency when EMF exposed, but not otherwise.

These six studies clearly show that EHS is a genuine EMF sensitivity where unlike normal controls, EHS people respond to low intensity EMF exposures, but not otherwise.

There are three other papers that I want to discuss in this section. The Greco, 2020 study showed that using a method called ultrasonic cerebral tomosphygmography that there were large regions of the brains in EHS and also multiple chemical sensitivity (MCS) patients which differed with extremely high levels of statistical significance with those same brain regions in normal controls. These findings clearly show that large regions of the brain in these two sensitivity conditions, differ from normal controls.

The Belpomme et al, 2021 meta review, showed that in addition to the specific changes described above, there were many non-specific physiological/biochemical changes that occurred as well. Such non-specific changes are of great importance because they show the large diversity of measurable changes that occur in EHS.

The Puri et al, 2020 paper was preceded by other studies showing that lymphocytes isolated from apparent EHS patients showed substantial increases in $[Ca^{2+}]i$ when exposed to low intensity EMFs whereas lymphocytes from normal showed much lower increases. The Puri et al, 2020 paper showed that when patient were treated with low-dose immunotherapy ascertained by provocation neutralization, this decreased the increases in $[Ca^{2+}]i$. These findings show that lymphocyte responses to EMFs in EHS patients become hypersensitive to EMFs, that EHS hypersensitivity may be produced by hypersensitivity of the VGCCs and that these measured responses may be useful in assessing patient response to a threapy

Evidence that EHS is caused by EMF exposure and possibly also by chemicals

Is EHS caused by EMF exposure? There were several important studies suggested in the very important EUROPAEM EMF review (Belyaev et al, 2016) suggesting EMF causation. Belyaev et al 2016 cited five studies where DECT phone or Wi-Fi exposures and the Molla-Djafari review summarized 30 different studies of mobile phone base station exposures including several reporting EHS-like effects. Havas, 2019 describes the long history of EHS starting with telephone operators in the early 20th century, later in radar operators in the 1940s and subsequently and then much more widely as diverse electronically generated EMFs spread almost like wild fire in countries around the world. Her paper entitled “Electrohypersensitivity

(EHS) is an Environmentally-Induced Disability that Requires Immediate Attention" cites a NASA study (Petrov, 1970) documentation much of the early history.

Carpenter, 2015 described a series of healthy people who were exposed to a single high level EMF exposure and developed apparent EHS. Symptoms included chronic headaches, irritability, emotional lability, decreased libido, and memory dysfunction often lasting for years. Hedendahl et al, 2015 described somewhat similar EHS-like symptoms in two 15 year old male students and a 47 year old female teacher exposed to Wi-Fi radiation.

Lebedeva & Sulimova, 1994 in an experimental human study showed that low intensity millimeter wave EMFs produced long-term stress-like changes in the hunan brain. The neurological effects of these changes are very similar to those widely reported to occur in EHS patients exposed to EMFs.

The most extensive reviews of the epidemiological literature on EMF causation of EHS are the two studies of Professor Emeritus Karl Hecht (2001, 2016). These both reviewed the Eastern European studies, mostly Russian language occupational exposure studies dating from around 1970 through 1990. These were all done during a period when there were no problematic EMF exposures in the general population such that effects of occupational exposures could be studied cleanly. What those extremely extensive studies showed is that EHS (and EHS was originally called microwave syndrome, see Carpenter, 2015) developed over months and years of occupational exposures, becoming more severe with time and with less spontaneous recovery with increased time of exposure when people were later removed from their EMF exposures.

The last epidemiological studies I will discuss here are the Conrad, 2011 and Lamech, 2014 on smart meter radiation. Both of these studies, despite using quite different methodologies, found large increases in EHS following smart meter installation. Conrad, 2011 also found that people who before the smart meters were installed had mild EHS symptoms often reported much more severe symptoms following smart meter radiation exposure.

There are also experimental studies of EMFs producing EHS-like changes in animals and again, the experimental basis of these studies is important. Burachas and Mascoliunas, 1989 described sensitivity changes in the compound action potential (CAP) in the frog sciatic nerve following MM-wave exposures. A second study by Chernyakov et al [53] also reported sensitivity changes using a different frog nerve using different MM-wave exposure protocols.

A fourth MM-wave animal study, discussed above in this section, also suggests possible EHS-like effects in animals. This is the Potekhina et al [54] study in the rat which found that non-pulsed MM-wave exposures produced rapid changes in the heart beat of animals. Exposures of three hours or more started to produce apparent sudden cardiac death in these exposed rats. I cited four reviews on EHS in Pall, 2021a, each reporting that among the most common sensitivities in EHS patients are neurological/neuropsychiatric sensitivity and cardiac sensitivity. Given that Havas et al, 2010 found that EMFs cause almost instantaneous changes in the heartbeat in some EHS patients, the Potekhina et al [54] rat study shows that low intensity EMFs cause EHS-like cardiac effects.

Pall 2021a reviewed studies showing that EMF impacts on the heart beat were mediated by their impact on the pacemaker cells in the sino-atrial node of the heart and.concluded that "The reason the pacemaker cells of the sinoatrial node of the heart may be particularly sensitive to EMFs is because they contain particularly high densities of T-type VGCCs, with both T-type and L-type VGCCs having essential roles in producing the pace making activity."

Akoev et al 1995 found EHS-like effects following low intensity MM-wave exposures on the activity of electroreceptors of skates (the article cited here is an English language study, published in an international journal that appears to be similar or identical to the Russian language article also discussed in Pall 2021a).

Bellono et al [60] showed that the electroreceptor is the VGCC Ca(V)1.3. What that Akoev and Bellono studies clearly show is that EHS-like effects can be produced via sensitization of the VGCCs following previous VGCC activation following EMF exposure.

Waldmann-Selsam 2019 showed that a very sensitive EHS patient who also had a parathyroid deficiency when exposed to extremely low EMF levels, had a very large drop in extracellular, including plasma levels of calcium. These drops must then be produced hyperactivation of a plasma membrane calcium channel, because the only place the extracellular calcium can go so quickly is into the cells of the body. The parathyroid defiency means that this EHS patient is unable to control extracellular calcium levels because control of extracellular calcium levels is the central function of the parathyroid gland, In principle, it might be possible that some channel other than the VGCCs could be involved here. The fact that we know that EMFs activate VGCCs in mammalian including human cells and that EMFs cause the VGCCs in the electroreceptor of the skate to become hypersensitive to activation argues against another channel being involved. So does Ockham's razor. The next section asks the question of how EMF exposures can produce hypersensitivity of the VGCCs?

EHS is caused by sensitization of the VGCCs. Central role of three protein kinases in the sensitization mechanism

The predominant mechanism by which regulatory changes in specific proteins is via protein phosphorylation produced by protein kinases. Consequently I searched under voltage calcium channel and protein kinase in Google Scholar and found over two dozen such studies, seven of which are cited here.(McCarron, et al, 1992; Greuter et al, 2008; Kemp & Hell, 2000; Zamponi et al, 1997; Sculptoreanu, et al, 1993; Welsby et al, 2003; Jahn et al, 1988). These studies show that three protein kinases phosphorylate the VGCCs, making them much more sensitive to activation. Those protein kinases are CamKII, which is directly activated by calcium and protein kinase A and protein kinase C which are indirectly activated by calcium; calcium also has a direct role in activating protein kinase C. There are phosphorylation sites on both the alpha-1 subunit and the beta subunit of the L-type VGCCs and there is also a phosphorylation site on the T-type VGCCs. We have then, for the first time a clear plausible mechanism for the main properties of EHS.

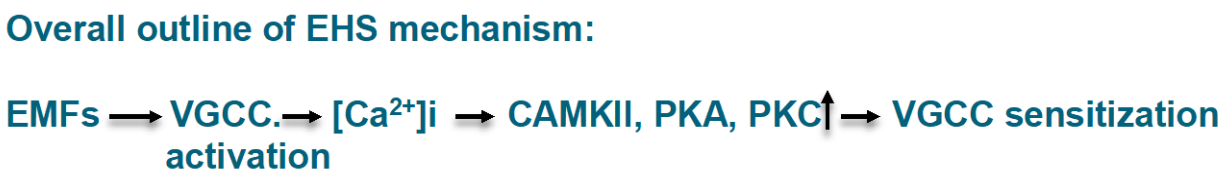

Figure 3.

The increased VGCC sensitization will act in multiple situations to produce EHS but the most important of these is likely to be via increased long-term potentiation and decreased long-term depression, making many synaptic connections in the central nervous system more sensitive, what is called neural sensitization. Consequently, one approach to EHS treatment which I believe is being discussed elsewhere in this volume is to decrease neural sensitization. I will also discuss one possible therapeutic approach to accomplish this below.

One additional point here. De Luca et al, 2011 showed that a genetic polymorphism producing lower enzymatic activity of an enzyme involved in chemical detoxification was associated with higher incidence of EHS. This finding strongly suggests that chemicals can cause EHS, not just EMFs. This is not surprising. Many chemicals act to produce higher activity of the NMDA receptors and the first thing that occurs on such NMDA receptor activation is increased $[Ca^{2+}]i$.

Probable roles of the NO/ONOO(-) cycle in EHS

It is rare for biological models to include all the complexities of the biology. Consequently, the model of EHS, outlined in Fig. 3, while it clearly makes very useful predictions, notably how previous EMF exposures (and possibly chemical exposures) can cause EHS and how, subsequently, how low intensity EMF exposures produce various EMF sensitivity responses. See Fi. 4.

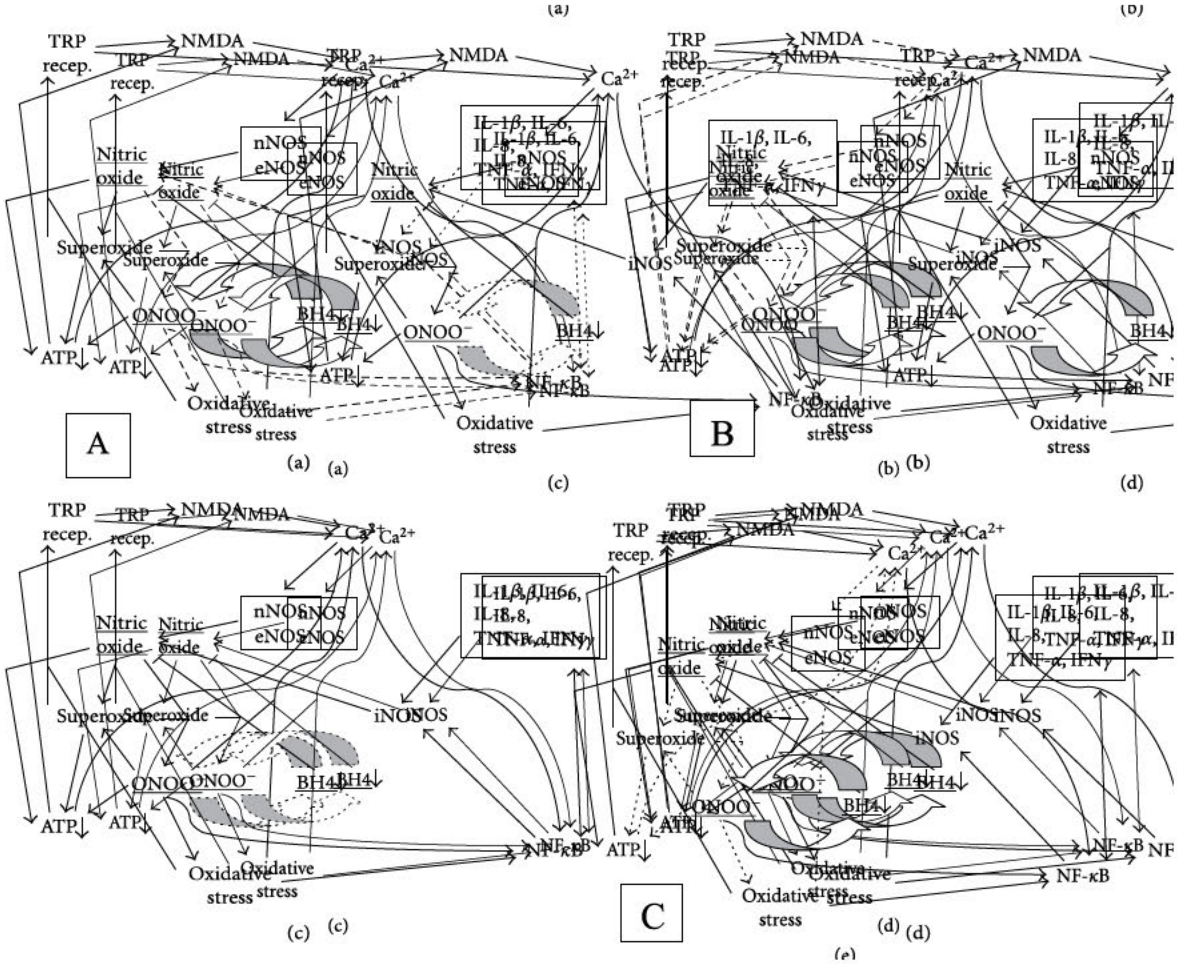

Figure 4 A, B, & C. Three underlying cycles of the NO/ONOO(-) cycle. The dashed or dotted arrows are the ones you should pay closest attention to for the individual sub-cycles. A is focussed peroxynitrite, free radicals, oxidative stress, NF-kappa B inflammation and consequences of inflammation. B & C are each focused on Ca^{2+} (actually $[Ca^{2+}]i$) and various other cycle elements. These are taken from Pall 2013b&c with permission and much further information on the cycle can be obtained from each of those papers. One point needs to be emphasized – everthing in a vicious cycle is both a cause and effect of the cycle.

But that does not mean that model is complete. It is my opinion that what is called the NO/ONOO(-) cycle is likely to have an important role in generating the chronic nature of EHS. The NO/ONOO(-) cycle is a primarily local biochemical vicious cycle which is made up of five underlying cycles, each of which are described in Pall 2013b&c. The only element in common in each of the five underlying cycles is elevated peroxynitrite. Three of the five underlying cycles most relevant here are shown below in Fig. 4 A, B & C.

The most important evidence that the NO/ONOO(-) cycle is involved in EHS is that both Igaray et al 2018 and Belyaev et al, 2016 have each shown that both 3-nitrotyrosine, a specific biomarker of peroxynitrite and oxidative stress are elevated in EHS, Because peroxynitrite is the

common element in all five cycles, its chronic elevation in EHS is strong evidence for NO/ONOO(-) cycle involvement.

Two possible simple, specific tests for EHS

We have already discussed three possible specific tests for EHS or possibly EHS and/or MCS. These are the tomosphygmography test of Greco, 2020, the fMRI study of Heuser & Heuser, 2017 and the lymphocyte $[Ca^{2+}]i$ of Puri et al, 2020. The problem with each of these is that they are each very expensive to do, require specialized equipment in order to do them and each cannot be done simply by sending one or a few blood-derived samples to a clinical lab. Belpomme and his colleagues have shown that there are other physiological/biochemical changes that occur in EHS patients as has Belyaev et al, 2016 in the EUROPAEM review and while most of these are non-specific, they do show that EHS is a disease of pathophysiology. Some of them might be EHS-specific if they were measured before and after EMF-provocation. I'm going to suggest two possible simple inexpensive EHS tests.

Waldmann-Selsam 2019 showed that a very sensitive EHS patient who also had a parathyroid deficiency when exposed to extremely low EMF levels, had a very large drop in extracellular, including plasma levels of calcium. These drops must then be produced hyperactivation of the VGCCs in the cells of the body produced be EMF exposures. EHS people with normal parathyroid function are predicted to release large amounts of parathyroid hormone following EMF exposure in order to maintain extracellular calcium levels within or close to the normal range. My guess is that parathyroid hormone levels will increase within about 10 minutes of EMF exposure, but timing of such increases needs to be established experimentally. Because parathyroid levels can be measured by an available clinical test, it may be possible to test large numbers of people for EHS simply by taking blood samples before and after EMF provocation. Provocation studies have been successfully done, as shown earlier, so it is not impossible to assess parathyroid changes following provocation as a means of assessing EHS severity
.
The origin for second suggested possible EHS is, no doubt, going to sound peculiar. I was with my female partner at the Shakespeare festival in Ashland Oregon and we went to a bank there for her to get some cash. I was talking to a woman who was helping people with banking problems but who had no problems to deal with at that time. She apparently knew that she had EHS and she was diagnosed, according to her, by someone who showed that the electrical resistance of her body was very low. She told me that the people in the low EMF zone in West Virginia knew about this. Unfortunately, I did not get her contact information nor contact information of the person who diagnosed her. So I called my scientific contact in West Virginia, Dr. Bertil (Bert) Schou and he told me that he had not heard about this but that EHS people served as much better antennae than do normals. Because antenna function is strongly related to electrical conductivity that observation is consistent with the claim I heard in Ashland. Furthermore, I found this claim to be credible based on the fact that most of the electrical resistance of the body is produced by the high electrical resistance of the plasma membranes in the cells of the body, resistance that is greatly lowered by the activation of VGCCs or other voltage-gated ion channels. So is there any published evidence that supports this interpretation? The answer is yes and it comes from the Rea et al, 1991 study of EHS. Rea et al, 1991 showed that people with apparent EHS had much

decreased electrical resistance in their bodies following EMF exposure, as measured by polygraph test.

One can buy ohm meters for circa $30 each and by connecting each of the two electrodes to a short section of copper pipe, one can measure body electrical resistance between one hand and the other. I suggest putting both hands into salted water to avoid any possible effect of perspiration.

The development of such a test is of critical importance in many ways in addition to just determining whether specific people have EHS and also how severe a specific case may be.

Looking at groups of people suspected having EHS because of EMF exposures or other reasons. I suggest looking at each of the following and it may well be the case that each of you may suggest still other groups of people.

Airline personnel and also frequent flyers
Air traffic controllers
People living near cell phone towers (mobile phone base stations).
Police who have high EMF exposures in police cars
Havana syndrome people
The homeless
Drug addicts
Migraine sufferers with and without aura
Athletes who have been saved from apparent sudden cardiac death
Digital dementia sufferers

Assessing the efficacy of possible treatments through a quantitative measure of EHS sensitivity. This was already shown to be promising in the Puri et al, 2019 paper. Given the challenge of quantifying the severity of EHS such a simple test for EHS will be invaluable in assessing the effectiveness of possible EHS treatments.

For both of the reasons discussed in the previous two paragraphs, a simple, inexpensive test for EHS is essential for assessing EHS properties in groups of people and for developing effective EHS therapies.

EHS Therapy: Various Approaches

I am not going to discuss here the low-dose immunotherapy ascertained by provocation neutralization therapy because of my limited understanding of it and refer the reader to Puri et al, 2020 to access that literature.

1. Correct three common deficiencies.

   There are two common deficiencies in human populations which may be predicted to exacerbate EHS, magnesium and vitamin D deficiencies. These should therefore to the extent possible be corrected. Magnesium deficiency raises the activity of the NMDA

receptors and also raises the synthesis of the VGCCs. Each of these effects raise $[Ca^{2+}]i$. I like magnesium citrate or malate and possibly in the evening magnesium taurate. Magnesium oxide should be avoided because of poor absorption.

Vitamin D deficiencies greatly exacerbate much of the pathophysiology produced by EMFs. These are also common in our societies because we spend so much time indoors, away from the UV light in sunlight and these problems are still much more serious in dark skinned people. Doses on the order of 5000 IU per day are recommended by physicians I have heard speak in recent meetings.

EMFs themselves produce deficiencies in nocturnal melatonin which act, in turn, to exacerbate EMF-induced pathophysiology. It follows that melatonin supplements may be taken just before bedtime.

2. EMF avoidance and use of shielding.

EMF avoidance is, of course becoming increasingly difficult as the telecom industry puts out more devices as well as more pulse modulated "smart" devices, producing ever increasing human EMF impacts. Somewhat similarly, we have ever increasing amounts of dirty electricity in our power wiring, producing ever greater biological impacts. So our challenge for avoidance becomes ever greater.

Let me mention a few things I have done to minimize my exposure. I use a wired connection to the internet with Wi-Fi being turned off both on my computer and my modem – you can use a cable modem where there is no Wi-Fi. I use a wired keyboard, mouse and printer. Wired connections are always much better, faster and more secure in addition to the human health advantages.

I have a smart meter on my house but I use particularly good shielding on the inner wall adjacent to the smart meter to protect myself (more about shielding later).

I do not have either a cell phone or cordless phone because each of these are problematic. I use an old fashioned corded phone. With the exception of a phone available in Europe, all cordless phone bases irradiate 24 hours per day. This is an unnecessary horrible design because the bases don't need to irradiate except when sending or receiving a call. If you must have a cordless phone keep the base far away from where you sleep and preferably anywhere else you spend a lot of time.

If you must carry a cell phone, you can buy cases which are shielded on one side but not the other. By carrying your cell phone in such a case with the shielding towards your body, that helps shield your body but not the body of others around you. Cell phones are continually sending off pulses to locate the nearest cell phone tower (mobile phone base station) such that where you carry your cell phone influences human biological effects. These are reported to include such effects as male fertility, breast cancer, cardiac effects and rectal cancer. If you don't need your cell phone to be active all the time, the best thing to do is to have it in airplane mode and carry it fully shielded while taking it out and

checking it, when needed. There are air tube earbuds which can be used to lower head EMF exposures when on a cell phone call – my understanding is that different cell phones often require different air tube earbuds.

The telecom industry often assures us that cell phones cannot produce human biological effects. However, there are studies showing that there are selective effects where the ipsilateral side of the head (where people use their cell phones) is much more affected than the opposite (contralateral) side of the head. These include brain cancer, long cell phone call-associated headaches, changes in blood flow in the brain, cellular DNA damage and tinnitus.

Let's talk about EMF shielding. It is my opinion that the most important effect of shielding is to disrupt the coherence of the EMFs, with the tiny metal fibers in shielding cloth materials and much more tiny graphite fibers and metal flecks in shielding paint acting in this way. Aluminum foil has been used but it reflects the EMFs so that areas receiving such reflective EMFs may have high exposures. I suggest crumpling aluminum foil used ib this way such that reflections will have lower coherence.

Dr. Panagopoulos has argued that we should not be doing shielding because it prevents our bodies from being exposed to the Schumann resonance of the earth (frequency circa 7.8 Hz. I agree with his concern but not with his conclusion. I believe that earthing, the favorable effects produced when people walk bare footed on the earth or otherwise ground their bodies is due to the Schumann Resonance as suggested by Sinatra et al, 2017. Elhalel et al, 2019 showed that very low intensity EMFs near the Schumann resonance frequency, 7 to 9 Hz, lowered the peak VGCC-dependent calcium transients in the heart by about 40%, producing cardiac protection. I know of health care providers who have claimed that devices emitting the Schumann resonance are helpful in the treatment of EHS and the EUROPAEM guidelines suggest using the Schumann resonance in EHS treatment (Belyaev et al, 2016). There is then a simple technological fix for the Panagopoulos concerns – use inexpensive electronic devices that emit the Schumann resonance for EHS treatment. Such devices may also be useful in many other situations to lower EMF effects.

3. Use of VGCC calcium channel blockers for treatment of EHS.
You would think with the beautiful mechanism discussed above, where VGCC sensitization can be produced through the action of three protein kinases each activated directly and/or indirectly by $[Ca^{2+}]i$ that there would be a lot of evidence showing that VGCC calcium channel blockers act to lower EHS effects. But there is actually very little. I've been contacted by two people with apparent EHS each of whom reported that they had been treated for hypertension with one of the VGCC calcium channel blockers and experienced substantial lowering of their EHS symptoms along with their hypertension symptoms. The blocker used in the first case was diltiazem. I don't know which blocker was used in the second. We need clearly some studies on effects of VGCC channel blockers on EHS patients, preferably using a test of EHS severity, to monitor effectiveness. I suggest starting with diltiazem and with nitrendipine, with the latter being the most effective such blocker in Alzheimer's treatment (Novotny et al,

2018; Pall, 2022). There is some reason to question whether any of the dihydropyridine blockers including nitrendipine will be effective in EHS treatment. Jahn et al, 1988 showed that CamKII, protein kinase C and protein kinase A each phosphorylate near the binding site of dihydropyridine binding to the VGCCs, presumably lowering effectiveness of the dihydropyridine blockers. However CamKII, protein kinase C and protein kinase A each have roles in Alzheimer's and it is possible that nitrendipine may be more active than the other dihydropyridine blockers in treating Alzheimer's, because nitrendipine blocking may be less affected by phosphorylation near the bonding site. In any use of VGCC calcium channel blockers, it is essential to avoid excessive hypotensive effects so low doses may be preferable. It may be predicted that EHS brains may be especially sensitive to lowered oxygen availability. There is one common food that contains calcium channel blockers and may, therefore be useful for EHS treatment: garlic. Garlic contains substantial amounts of two calcium channel blockers (Neuhaus-Carlisle et al, 1997), allicin and trans-ajoene and garlic is also active in raising the levels of Nrf2 (discussed in the next topic). Consequently, there may be two reasons why eating garlic may be useful. Allicin is also found in green onions, shallots and Chinese garlic chives but not in bulb onions. Because garlic is reported to lower hypertension in a systematic review Xiong et al, 2015), these findings suggest that the VGCC calcium channel blocking by garlic may occur at substantial levels in humans.

4. Raising Nrf2 in EHS therapy.

The NO/ONOO(-) cycle has a highly probable role in maintain the chronicity of EHS. The complexity of the cycle makes lowering the cycle a challenging process. However, it has been argued that raising the level of Nrf2 is nature's way of preventing or treating NO/ONOO(-) cycle diseases because many of the elements in the cycle, including the most central one peroxynitrite, are lowered by raising Nrf2 (Pall & Levine, 2015). There are many health promoting factors which raise Nrf2 as shown in Fig. 5, each of which may be useful in therapy. For those who are limited to conventional pharmaceuticals for raising Nrf2, you may consider off label use of methylene blue which has been shown to raise Nrf2.

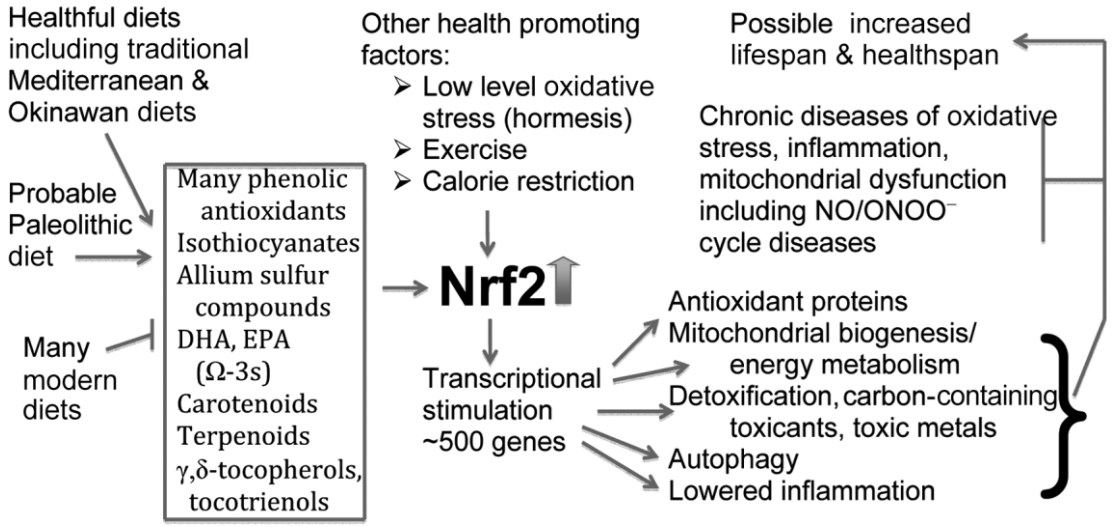

Figure 5. Taken with permission from Pall & Levine, 2015.

5. Some months ago, I was reading a book authored by Eric Kandel, a Nobel Laureate, entitled "The Disordered Mind: What Unusual Brains Tell Us About Ourselves." In it Kandel discusses, in the context of PTSD, that when an event occurs bringing to the surface the symptoms recalling a much earlier stressful event, the synaptic connections producing those symptoms are weakened such that they can become disrupted by the beta blocker propranolol. When I went into the scientific literature, I found there was a much larger scientific literature on this than what Kandel cited. So it may be possible to disrupt specific synaptic connections previously strengthened by long term potentiation, when they are active (see Brunet et al, 2018). There is no reason to think that the role of the beta adrenergic receptors in learning and memory is PTSD-specific (O'Dell et al, 2015). It may be possible, therefore, to *selectively unlearn* the long term potentiation that leads to neural sensitization following EMF exposure (or, for that matter, chemical exposure in MCS) by using a beta blocker before an exposure of a sensitive individual to EMFs or chemicals. Studies on this could be done either in conjunction with a series of provocations, as were the Brunet et al, 2018 PTSD studies or by allowing EHS patients to use propranolol or other beta blocker when the individual knows from previous experience that they need to go to a location where a particular exposure is likely to produce a sensitivity response. This, of course would be an off label drug usage. Beta blockers such as propranolol have substantial side effects and they should only be used, in my opinion, shortly before an EHS patient will be exposed to an EMF.

6. There are a number of products on the market that are claimed to help protect us from EMF effects. Some of them produce an EMF of their own but more commonly, these products apparently have within them, crystals or other highly structured materials that may reflect incoming EMFs, producing other, much weaker EMFs coming towards our bodies from a different direction. The voltage sensor must have all four S4 helixes ratcheting out away from the center of the closed channel, in order to activate the channel. I believe that these devices may work by producing another EMF that causes one or more of the helixes to ratchet back towards the center of the closed channel, thus blocking the opening of the channel. I have neither time nor space here to document this opinion. I am not endorsing any of these devices, but it is my opinion that they may be worth trying in EHS therapy.

7. I am indebted to Mr. John Costa for suggesting this. High doses of vitamin K2, have been reported to lower VGCC activation as well as destructive downstream effects of [Ca2+]i elevation (Xu et al, 2022; Ferland, 2012) and may, therefore be useful in treating EHS, as suggested by Mr. Costa.

The author declares no conflict of interest.

This paper is dedicated to the memory of Dr. Peter Ohnsorge, a great man and wonderful friend.

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