Electromagnetic field exposures act via activation of voltage-gated calcium channels.

How this leads to diverse impacts on health

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Outline:

- 26 studies show that non-thermal EMF effects are caused by activation of voltage-gated calcium channels (VGCCs)!

- *The VGCC voltage sensor has physical properties such basic physics predicts that it is extraordinarily sensitive to electrical forces produced by low-intensity EMFs. Thus both the physics and the biology strongly support this mechanism.*

- Excessive calcium in the cell produced by VGCC activation can act along three different pathways to produce diverse pathophysiological and also therapeutic effects.

- *These include oxidative stress, cellular DNA damage, cancer, widespread neuropsychiatric effects, male and female infertility and increased spontaneous abortion, widespread endocrine changes, cardiac changes on the electrical control of the heart and many others.*

It is high time to jettison the claim that there are only thermal effects.

It is also high time to do safety testing of devices exposing us to microwave frequency EMFs by biological testing.

A great puzzle:

- These EMFs are composed of low energy photons, with energy per photon too low to influence the chemistry of the body!
- *How can they influence our biology through non-thermal effects?*
- Safety standards assume that they can't - that only thermal effects need to be considered: If no thermal effects there cannot be biological effects.
- *And yet, there are thousands of papers in the scientific literature reporting biological effects of exposures well within safety standards!*
- *Solution: EMF effects shown in 26 studies to be blocked by calcium channel blockers – drugs specific for blocking voltage-gated calcium channels (VGCCs). When the VGCCs are activated, they open up a channel that allows calcium (Ca2+) to flow into the cell.*
- *Electromagnetic fields act via activation of voltage-gated calcium channels to produce beneficial or adverse effects. Pall ML. J Cell Mol Med. 2013 Aug;17(8):958-65.*

Problem 2:

For over 30 years, it has been known that *pulsed* electromagnetic fields are often much more biologically active then are non-pulsed fields.

That is inconsistent with the thermal/heating paradigm:

Pulsed fields either produce less heating or the same amount, depending on how the experiment is set up.

So we meet again the great puzzle:

How can such low intensity EMFs influence our biology - for better or for worse?

Energy per photon is too low to influence the chemistry of the body!

Can they influence our biology through non-thermal effects?

There is a substantial literature reporting that they do.

I recently solved this important puzzle:

EMFs activate voltage-gate calcium channels.

And it is the downstream effects of the increased intracellular Ca^{2+} ([Ca2+]i) that leads to the biological effects of EMF exposure.

The most central evidence:

A whole series of studies have shown that in studies of exposures to various low frequency EMFs, all of the effects produced can be blocked by calcium channel blockers - drugs that block voltage-gated calcium channels.

I will discuss first some of the evidence supporting this mechanism.
I will discuss later how this may lead to various diseases.

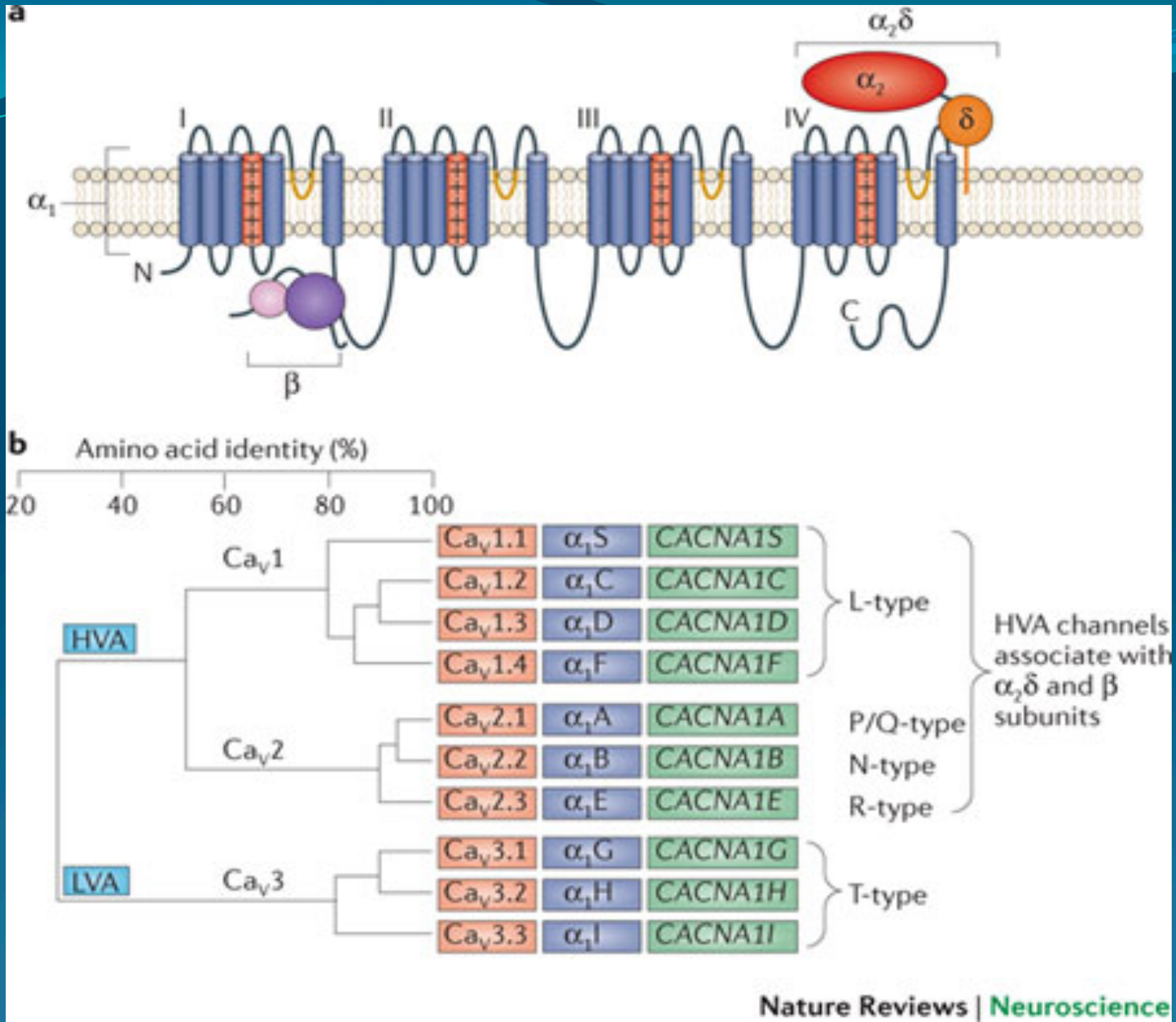
Table 1: EMF Responses Blocked or Lowered by Calcium Channel Blockers

Ref #	EMF type	Calcium channel	Cell type or organism	Response measured
2	Pulsed magnetic fields	L-type	Human lymphocytes	Cell proliferation; cytokine production
3	Static magnetic field (0.1 T)	L-type	Human polymorphonuclear leukocytes	Cell migration; degranulation
5	ELF	L-type	Rat chromaffin cells	Differentiation; catecholamine release
6	Electric field	L-type	Rat and mouse bone cells	Increased Ca2+, phospholipase A2, PGE2
7	50 Hz	L-type	Mytilus (mussel) immunocytes	Reduced shape change, cytotoxicity
8	50 Hz	L-type	AtT20 D16V, mouse pituitary corticotrope-derived	Ca2+ increase; cell morphology, premature differentiation
9	50 Hz	L-type	Neural stem/ progenitor cells	In vitro differentiation, neurogenesis
10	Static magnetic field	L-type	Rat	Reduction in edema formation
11	NMR	L-type	Tumor cells	Synergistic effect of EMF on anti-tumor drug toxicity

Ref #	EMF type	Calcium channel	Cell type or organism	Response measured
11	NMR	L-type	Tumor cells	Synergistic effect of EMF on anti-tumor drug toxicity
12	Static magnetic field	L-type	Myelomonocytic U937 cells	Ca^{2+} influx into cells and antiapoptotic effects
13	60 Hz	L-type	Mouse	Hyperalgesic response to exposure
14	Single nanosecond electric pulse	L-type	Bovine chromaffin cells	Very rapid increase in intracellular Ca^{2+}
15	Biphasic electric current	L-type	Human mesenchymal stromal cells	Osteoblast differentiation and cytokine production
16	DC & AC magnetic fields	L-type	b-cells of pancreas, patch clamped	Ca^{2+} flux into cells
17	50 Hz	L-type	Rat pituitary cells	Ca^{2+} flux into cells
18	50 Hz	L-type,N-type	Human neuroblastoma IMR32 and rat pituitary GH3 cells	Anti-apoptotic activity
19	Nanosecond pulse	L-type, N-type, P/Q-type	Bovine chromaffin cells	Ca^{2+} dynamics of cells
20	50 Hz	Not determined	Rat dorsal root ganglion cells	Firing frequency of cells
21	700 to 1100 MHz	N-type	Stem cell derived neuronal cells	Ca^{2+} dynamics of cells

22	Very weak electrical fields	T-type	Sharks	Detection of very weak magnetic fields in the ocean
23	Short electric pulses	L-type	Human eye	Effect on electro-oculogram
24	Weak static magnetic field	L-type	Rabbit	Baroreflex sensitivity
25	Weak electric fields	T-type	Neutrophils	Electrical and ion dynamics

This entire table is support for the conclusion that such EMFs act biologically by activating VGCCs.



Nature Reviews | Neuroscience

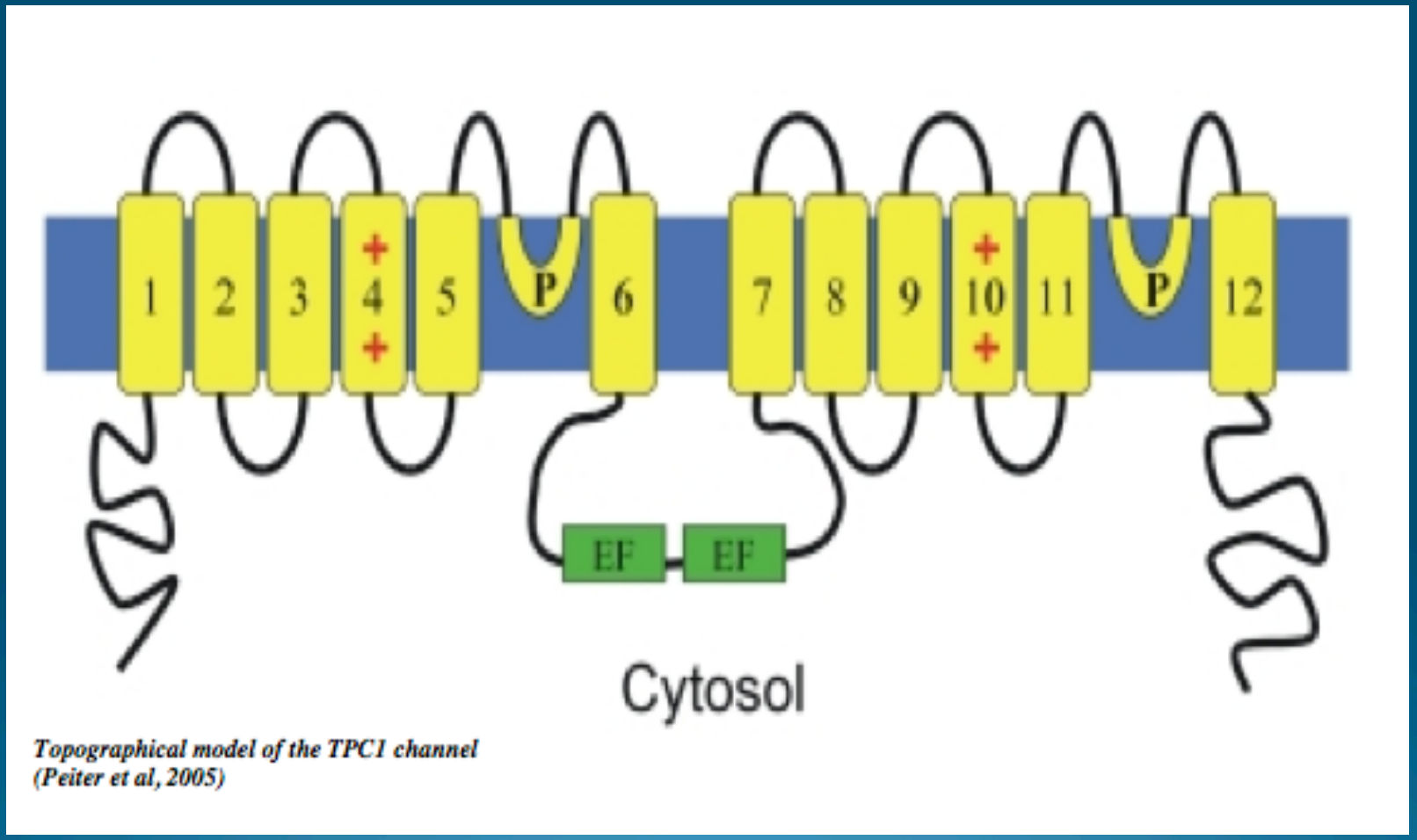
Such VGCC-like channels activated by depolarization of the plasma membrane are widely found in plants (6).

6. Biochim Biophys Acta 2000;1465:171-189.

Weak microwave field exposures are reported to often produce changes in calcium fluxes or signaling in plants (7-9), suggesting that activation of VGGC-like channels is commonly involved in producing responses to such weak fields.

7. Adv Space Res 2004;34:1566-1574; 8. BMC Plant Biol 2009,9; 9. Biotech Biotechnological Equip 2009;23:611-615.

These studies strongly suggest that weak microwave EMFs often activate VGCC-like channels in plants, acting similarly to the way such fields act in animals! While the structure of these VGGC-like channels is substantially different from those of animal VGCCs, they may function quite similarly.



Topographical model of the TPC1 channel (Peiter et al, 2005)

This channel occurs in both the plasma membrane and in the vacuolar membrane – thought to act as a dimer. Described as "half the general structure of the α-1" subunit of the VGCCs (Furuichi et al).

However, the advocates of the current safety standards, claim to this day, that there are no biophysically viable mechanisms for these weak field EMFs to produce non-thermal effects in our bodies. This claim is argued as follows (see Sheppard AR et al, Health Phys 2008;95:365-396):

While, they acknowledge that EMFs can exert forces on charged groups, they argue that weak EMFs produce only weak forces that are less than are exerted by thermal motion produced at normal body temperature. They argue, therefore, that the only effects that can be produced by weak EMFs would be dwarfed by a high background noise created by random thermal motion.

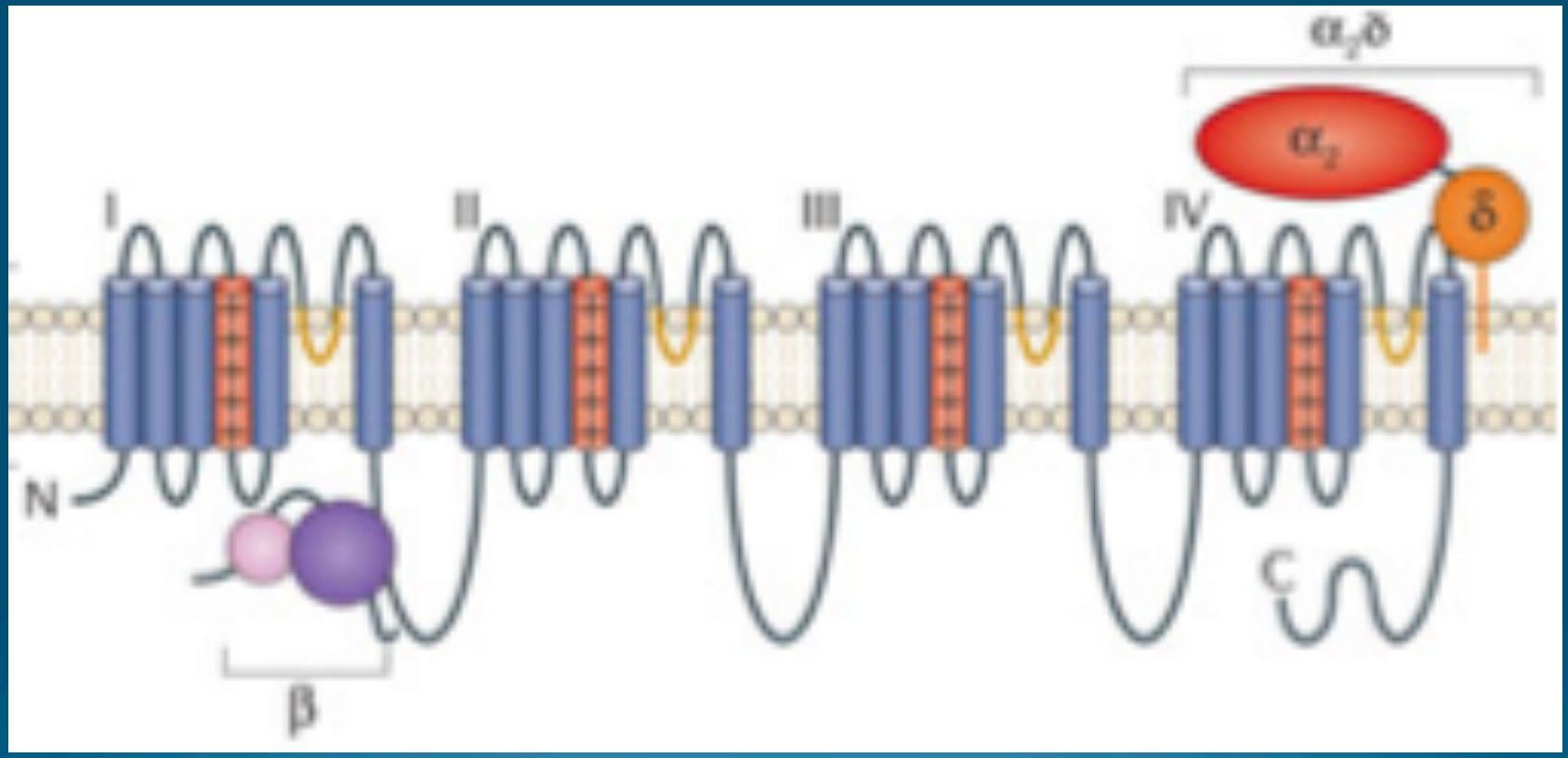
Let's look at the known properties of the VGCCs to see whether this argument holds up.

The 4 alpha helixes, each containing 5 positively charged groups, act collectively as the voltage sensor, opening the channel when they are all pushed in the same specific direction at the same time. The fields, of course, can do that because the fields produce forces on each of these charged groups in the same direction. In contrast, thermal movements are random in three dimensions, and will only extremely rarely produce movement in a specific direction at the same time.

It follows, that calculations based on the behavior of single charged groups, completely break down when considering 20 charged groups acting on 4 alpha helixes which must move in approximately the same direction in order to open the channel.

Furthermore, random thermal motion can act not only on the charged groups but also on all atoms of the transmembrane helixes.

With over 200 atoms in a transmembrane helix being jostled thermally roughly independently of each other, the chances that all four of these sensor transmembrane helixes moving together in the approximately the right direction due to random thermal motion is extraordinarily low.



Taken from Prof. Annette Dolphin, Nature Reviews Neuroscience

Coulomb's law
Gaussian units:

$$F = q\, q' / \varepsilon_r\, r^2$$

ε_r= dielectric constant

There are three factors that influence the electrical forces placed on the voltage sensor (all based on basic Physics):

1. There are 20 charges found in the voltage sensor – not just one!
2. Coulomb's law states that the forces placed on charged groups are inversely proportional to the dielectric constant of the medium where the charged groups are found. This predicts that the forces on each of these 20 charged groups will be increased by about a factor of 120.
3. The plasma membrane of the cell has a very high electrical resistance, such that Sheppard et al 2008 predict an amplification of electrical gradients of about 3000-fold across the plasma membrane as opposed to in the aqueous phase of the cell. This predicts an additional factor of about 3000 producing increased sensitivity.

In comparing the forces on the voltage sensor with the forces on singly charged groups elsewhere in the cell:

The force on the voltage sensor is approximately:

20 X 120 X 3000 = 7.2 million times stronger

This is an estimate, not a precise calculation.

Because EMF heating is produced mainly by forces on singly charged groups in the aqueous phases of the cell, *this argues that the safety guidelines/standards allow us to be exposed to EMFs that are approximately 7.2 million times too high!*

- In summary, a central role of VGCC activation in responses to low level EMFs is shown by:

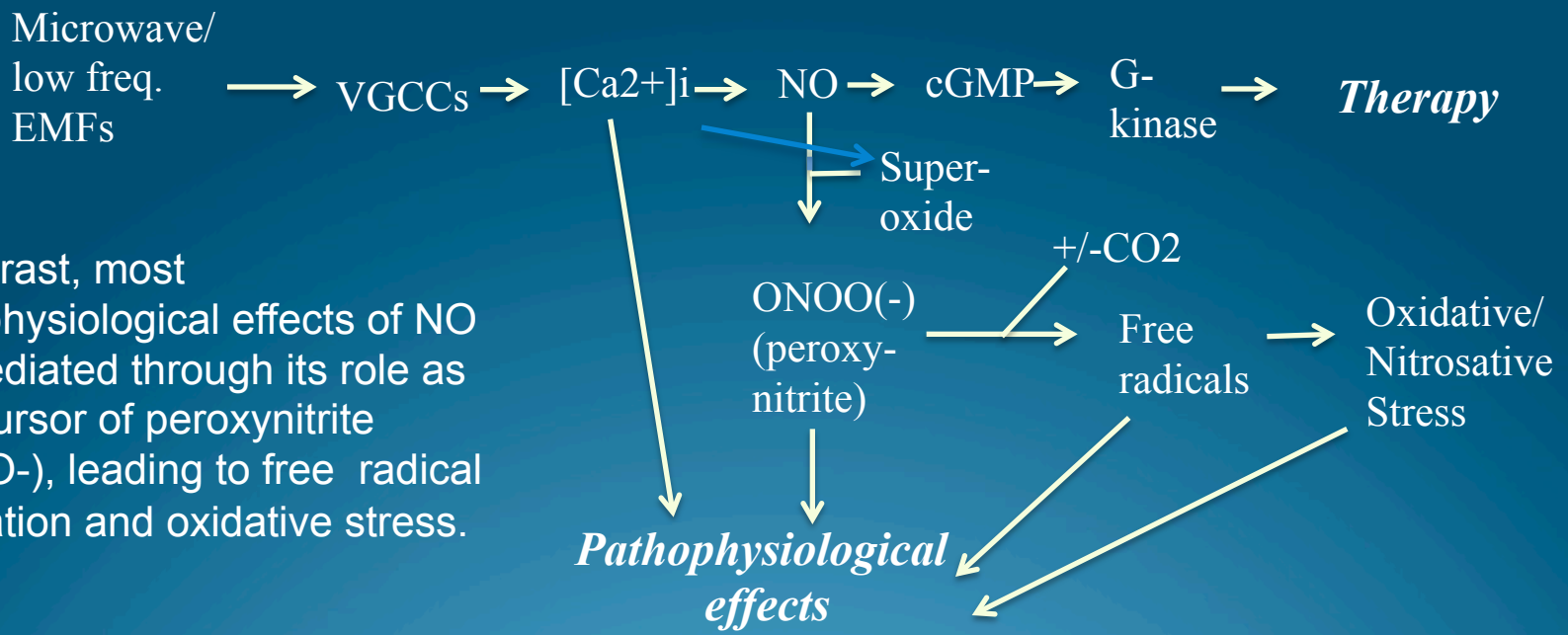
- In 26 different studies, effects of low intensity microwave/lower frequency EMFs were blocked by calcium channel blockers.
- In each of these studies, all such effects studied were blocked or greatly lowered, suggesting a widespread, perhaps universal role of VGCCs in producing such effects.
- Hundreds of studies show changes in Ca^{2+} fluxes and/or Ca^{2+} signaling following microwave EMF exposure, consistent with effects of VGCC activation.
- Pilla showed that pulsed field microwave exposure produces an almost instantaneous (<5 sec.) increase in Ca^{2+}/calmodulin-dependent nitric oxide (NO) synthesis, consistent with a direct VGCC activation response.
- VGCC activation has a universal or near universal role in converting electrical signals to chemical signals in the body.
- Low level EMFs activate VGCC-like channels in plants containing a similar voltage sensor to that found in the VGCCs.
- The properties of the voltage sensor of the VGCCs predicts that the VGCCs are exquisitely sensitive to low intensity EMFs. It is clear that VGCC activation is an exception to the claim that there cannot be a biophysically viable mechanism for low intensity EMF effects.

There can be no question that VGCCs are the major, perhaps the only targets of low intensity EMFs in the body.

How then does VGCC activation act to produce biological changes in the body?

Most physiological responses to [Ca2+]i and NO, act as follows:

NO increases levels of cGMP, leading in turn to stimulation of the cGMP-dependent protein kinase (protein kinase G).



In contrast, most pathophysiological effects of NO are mediated through its role as a precursor of peroxynitrite (ONOO-), leading to free radical generation and oxidative stress.

Arthur A. Pilla published a model of therapeutic effects of EMFs and reviewed the evidence supporting it, a model that was very similar but not identical to mine that you just saw on the preceding slide. He states in the title, abstract and first sentence of his paper that these are all non-thermal effects.

Nonthermal electromagnetic fields: from first messenger to therapeutic applications.
Pilla AA.
Electromagn Biol Med. 2013 Jun;32(2):123-36.

I proposed a similar mechanism to the Pilla mechanism for this in two papers.

Some Relevant Papers for my talk:

Electromagnetic fields act via activation of voltage-gated calcium channels to produce beneficial or adverse effects. Pall ML. J Cell Mol Med. 2013 Aug;17(8):958-65.
This paper was honored to be included on the "Global Medical Discovery" site as one of the most important medical papers of 2013.

- Pall ML. 2014 Electromagnetic field activation of voltage-gated calcium channels: role in therapeutic effects. Electromagn Biol Med. 2014 Apr 8.
- Scientific evidence contradicts findings and assumptions of Canadian Safety Panel 6: microwaves act through voltage-gated calcium channel activation to induce biological impacts at non-thermal levels, supporting a paradigm shift for microwave/lower frequency electromagnetic field action. Pall ML. Rev Environ Health. 2015;30(2):99-116.
- Pall M. L. 2009 Multiple chemical sensitivity: Toxicological questions and mechanisms. In General and Applied Toxicology, 3rd Edition, John Wiley & Sons, pp. 2303-2352.

Health Impacts by Microwave Radiation

There are multiple studies showing that each of the following responses have been reported to be produced by microwave radiation exposures.

The scientific evidence is very strong on each of the effects shown in the following table, although spokesmen for corporations will, no doubt, argue against each of them.

None of these **can be explained by heating** -- they can all be **explained by VGCC activation** and downstream effects!

Table 1. Apparent Mechanisms of Action for Microwave Exposures Producing Diverse Biological Effects (See Fig. 1)

Reported Biologic Response	Apparent Mechanism(s)	Citation(s)/Comments
Oxidative stress	Peroxynitrite & consequent free radical formation	[1-3]; detected via a large number of oxidative stress markers
Single strand breaks in cellular DNA	Free radical attack on DNA	[1-3]
Double strand breaks in cellular DNA	Same as above	Same as above; detected from micronuclei and other chromosomal changes
Cancer	Single and double strand breaks, 8-nitroguanine and other pro-mutagenic changes in cellular DNA; produced by elevated NO, peroxynitrite	[3] and this paper
Breakdown of blood-brain barrier	Peroxynitrite activation of matrix metalloproteinases (MMPs) leading to proteolysis of tight junction proteins	[3]
Male and female infertility	Induction of double strand DNA breaks; Other oxidative stress mechanisms; [Ca2+]i mitochondrial	[3]

Male and female infertility	Induction of double strand DNA breaks; Other oxidative stress mechanisms; [Ca2+]i mitochondrial effects causing apoptosis; in males, breakdown of blood-testis barrier	[3]
Therapeutic effects	Increases in $[Ca]_i$ and NO/NO signaling	[1-3; 13]
Depression; diverse neuropsychiatric symptoms	VGCC activation of neurotransmitter release; other effects?; possible role of excess epinephrine/norepinephrine	These were reported in occupational exposures [21]; also reported in people living near cell phone towers
Melatonin depletion; sleep disruption	VGCCs, elevated $[Ca]_I$ leading to disruption of circadian rhythm entrainment as well as melatonin synthesis	[3]
Cataract formation	VGCC activation and $[Ca]_I$ elevation; calcium signaling and also peroxynitrite/oxidative stress	This paper
Tachycardia, arrhythmia, sometimes leading to sudden cardiac death	Very high VGCC activities found in cardiac (sinoatrial node) pacemaker cell; excessive VGCC activity and [Ca2+]i levels produces these electrical changes in the heart	[3]

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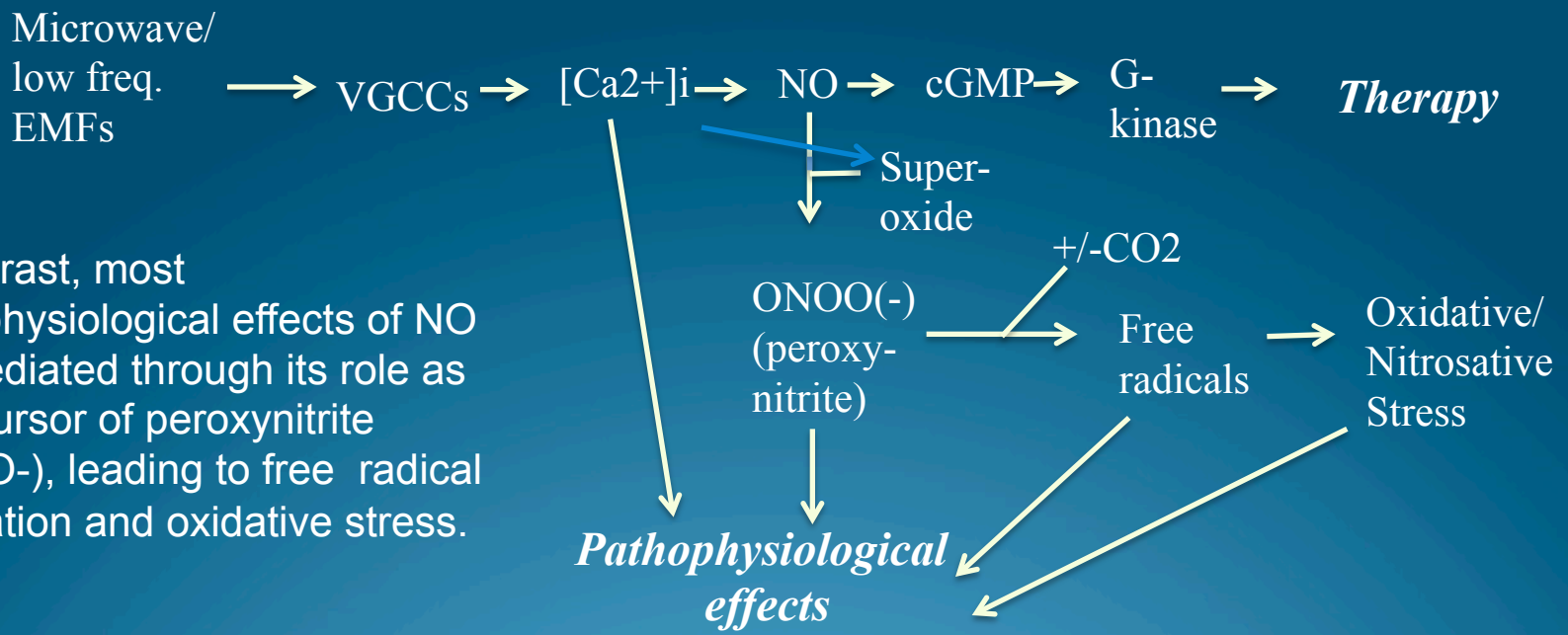
NO increases levels of cGMP, leading in turn to stimulation of the cGMP-dependent protein kinase (protein kinase G).



In contrast, most pathophysiological effects of NO are mediated through its role as a precursor of peroxynitrite (ONOO-), leading to free radical generation and oxidative stress.

These are not the only pathophysiological effects of such EMFs but these are among the best understood in terms of mechanism.

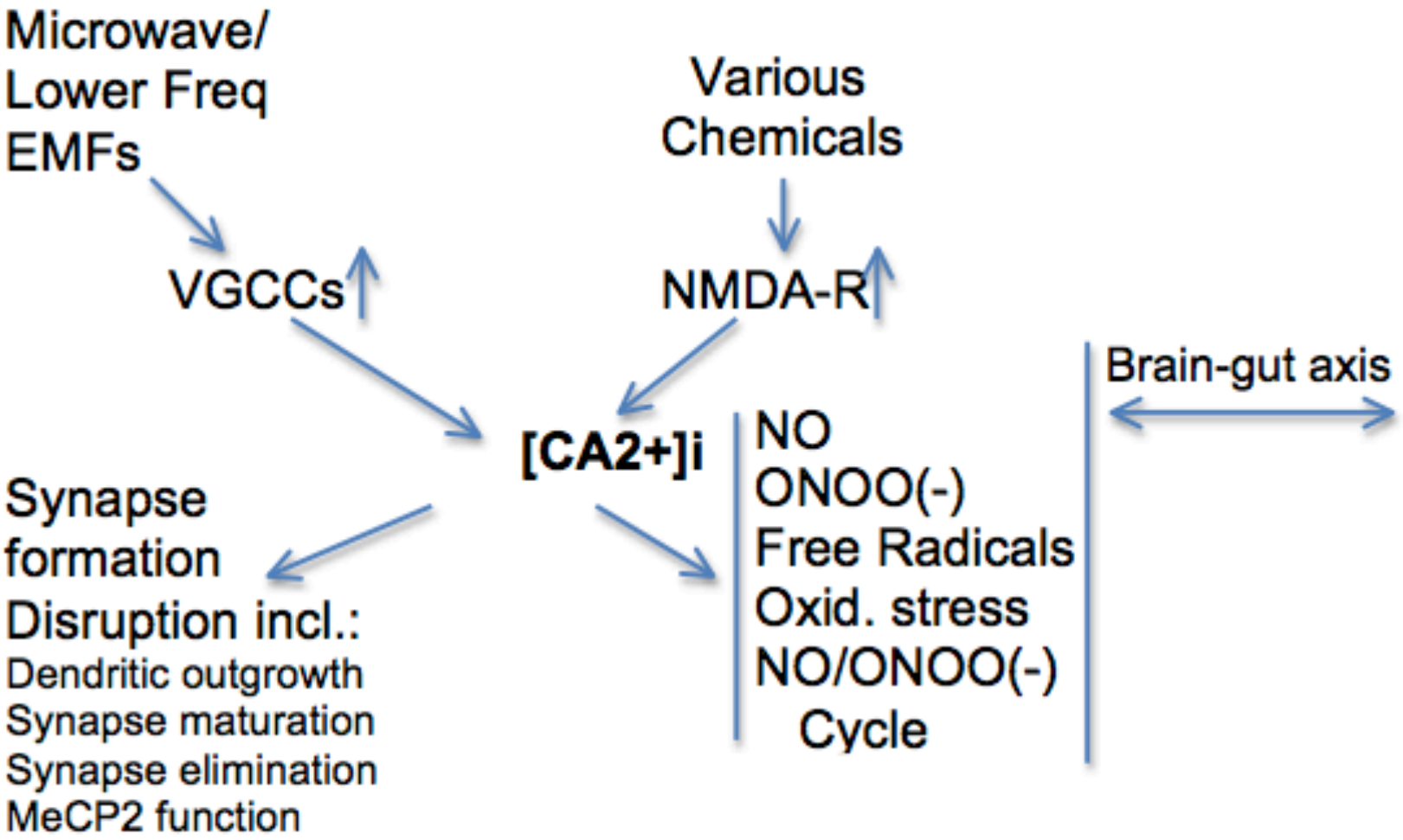
And they give you some idea of the breadth of the effects seen.

Simply based on these things that are listed in the previous table, we can say that these fields are attacking the four things that we most value as individuals and as a species:

- Our health
- Our brain function
- The integrity of our genomes
- Our ability to produce health offspring

While those four things are each of great power, there are still worse things that may be in store for us – what may be called worse case scenarios! I'll talk about 5 of these.

Worst case scenario 1:
The autism epidemic is probably largely caused by EMF exposures (although chemicals also have a role). At the AutismOne meeting Chicago, last month, I discussed 30 different types of evidence that support a pathway of action from microwave EMF exposure through disruption of synapse development in the developing brain of autism patients.


Microwave/
Lower Freq
EMFs
Various
Chemicals
VGCCs
NMDA-R
[CA2+]i
Brain-gut axis
NO
ONOO(-)
Free Radicals
Oxid. stress
NO/ONOO(-)
Cycle
Synapse
formation
Disruption incl.:
Dendritic outgrowth
Synapse maturation
Synapse elimination
MeCP2 function

Worse case scenario number 2: Neuropsychiatric effects of the microwave EMF exposures:

Table 4. Commonly Reported Neuropsychiatric Symptoms following Microwave EMF Exposure

Symptom(s)	#s of studies reporting
Sleep disturbance/insomnia	16
Headache	15
Depression/depressive symptoms	11
Fatigue/tiredness	11
Dysesthesia (vision/hearing/olfactory dysfunction)	10
Concentration/attention/cognitive dysfunction	9
Memory changes	8
Dizziness	8
Irritablity	8
Loss of appetite/body weight	8
Restlessness/tension/anxiety/stress/agitation/feeling of discomfort	6
Nausea	6
Skin tingling/burning/inflammation (dermographism)	6

Worse case scenario number 3: Sterility and spontaneous abortion, reproduction goes to zero.

We know that male and female infertility are increasing as is spontaneous abortion and we know that these can each be caused by microwave EMFs. Most extensive evidence on male infertility (easiest to study) but also evidence on the other two (Cell Biochem Biophys 2013;65:85-96; Andrology 2014;2:491-501; Reprod Toxicol 2013;36:1-5; J Environ Health Sci Eng. 2015 Apr 21;13:34. doi: 10.1186/s40201-015-0193-z).

Magras and Xenos (Bioelectromagnetics 1997;18:455-461) showed that pairs of mice mated at two exposure levels near an “antenna park” of large numbers of broadcasting antennae (but still within safety standards) went through only two (higher exposure) or four matings (lower exposure) in less than 5 months before they became completely sterile.

Worse case scenario number 4: Huge numbers of germ line mutations.

We know that:
1.Microwave fields are genotoxic – produce widespread DNA damage in cells.
2.Germ line cells are heavily impacted by these EMFs.
3.There have been only 3 studies of mutations in germ line cells following microwave/RF EMF exposures, to my knowledge (all 3 in males), with each of the 3 reporting mutational increases: Sarkar et al, Mutat Res 1994;320:141-147; Aitken et al, In J Androl 2005;28:171-179; De Iuliis et al, PloS One 2009;4(7) e6446

<u>We could be destroying our biological inheritance</u>.

Worse case scenario number 5: Epidemic of premature Alzheimer's disease. We are seeing an unexplained epidemic of premature Alzheimer's disease.

We know that:

1.Epidemiological studies have shown that occupational exposures to extremely low frequency EMFs, such as from our power lines, increases Alzheimer's incidence. Also know that extremely low frequency EMFs act like microwave EMFs – both act via VGCC activation!

2.High levels [Ca2+]i have important roles in Alzheimer's.

3.Alzheimer's typically have very long latency periods – 20, 25 or 30 years from the time the process starts until symptoms become apparent.

4.Jiang et al showed that young rats exposed to multiple short pulsed microwave EMFs, developed oxidative stress, high amyloid beta (Aβ protein) levels as well as cognitive and memory impairment in middle age – Alzheimer's like changes.

Electromagnetic pulse exposure induces overexpression of beta amyloid protein in rats. Arch Med Res. 2013 Apr;44(3):178-84.

Before leaving this area, I want to reconsider the issue of carcinogenesis of low intensity microwave/lower frequency fields.

The recent 2014 Canadian Report, supporting current safety standards, states that "There is no viable biophysical mechanism" for carcinogenesis by such low intensity fields. We have already discussed the general issue of biophysical plausibility, but we need to consider the specific issue with regard to cancer.

It has been shown that NO and peroxynitrite/oxidative stress/free radical elevation are central to the mechanism of inflammatory carcinogenesis and that these act via the mechanisms proposed in the previous table for low intensity EMF carcinogenesis.
Mol Cell Biochem (2013) 378: 291-8; *Antioxid Redox Signal* (2006) **8**: 1033-45; *Carcinogenesis* (2003) 24: 235-41; *Biol Chem* (2006) 387: 365-72.

Therefore there is a biophysically viable mechanism for carcinogenesis by these low intensity microwave/lower frequency fields.

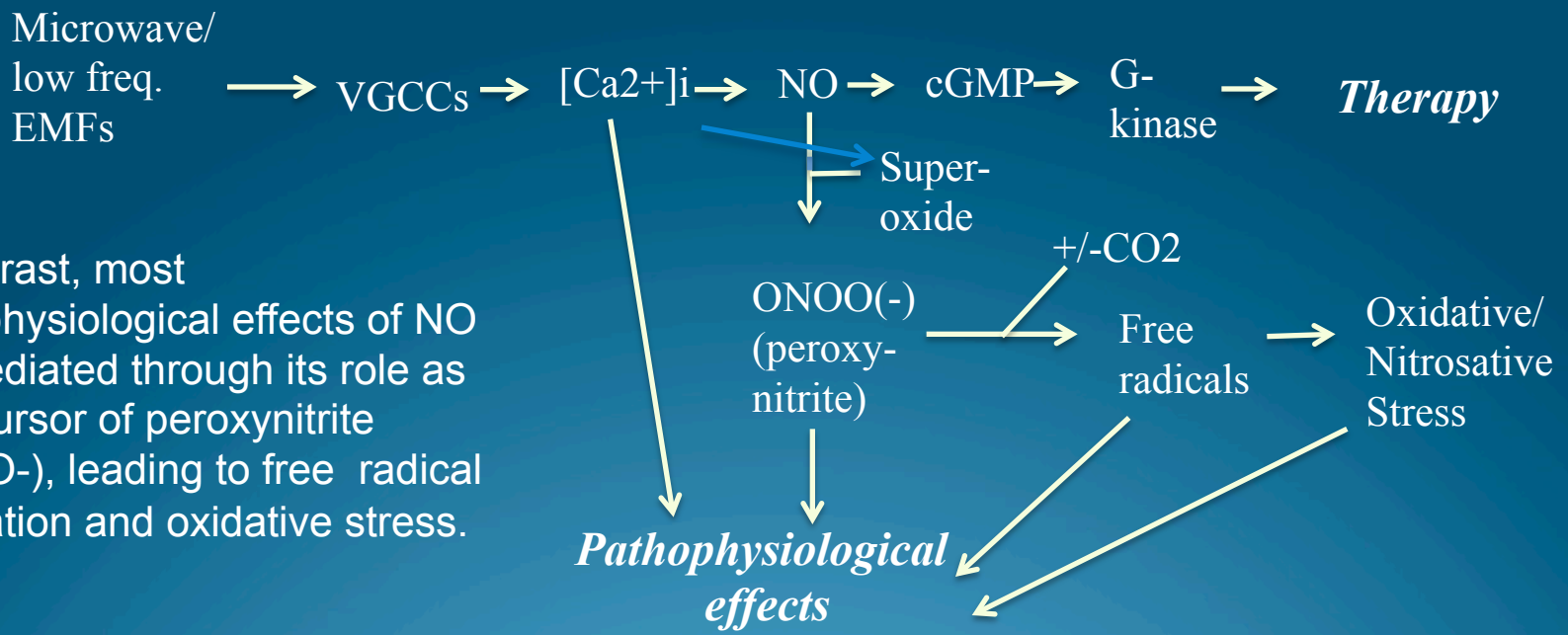
It may also be of interest to also compare ionizing radiation effects with those of microwave fields. It has also been shown that free radicals formed through Compton scattering by ionizing radiation have essential roles in ionizing radiation carcinogenesis, demontrating similarities between microwave EMF carcinogenesis and ionizing radiation carcinogenesis.

Advocates of only heating effects, *emphasize the correct fact that the individual microwave photons have insufficient energy to perturb the chemistry of our bodies* and they infer from this that these photons cannot cause cancer or many other pathophysiological responses. But what the Canadian panel and others fail to realize is that the microwave fields as a whole, acting through downstream effects of VGCC activation, lead to high densities of intracellular free radicals and can produce, therefore, similar effects on the body to those produced by ionizing radiation exposure.

There is even an argument that low intensity field exposures of microwave/lower frequency radiation ***may be*** more dangerous than are similar intensity ionizing radiation effects, because of amplification mechanisms and NO-independent excessive calcium signaling mechanisms.

Most physiological responses to [Ca2+]i and NO, act as follows:

NO increases levels of cGMP, leading in turn to stimulation of the cGMP-dependent protein kinase (protein kinase G).

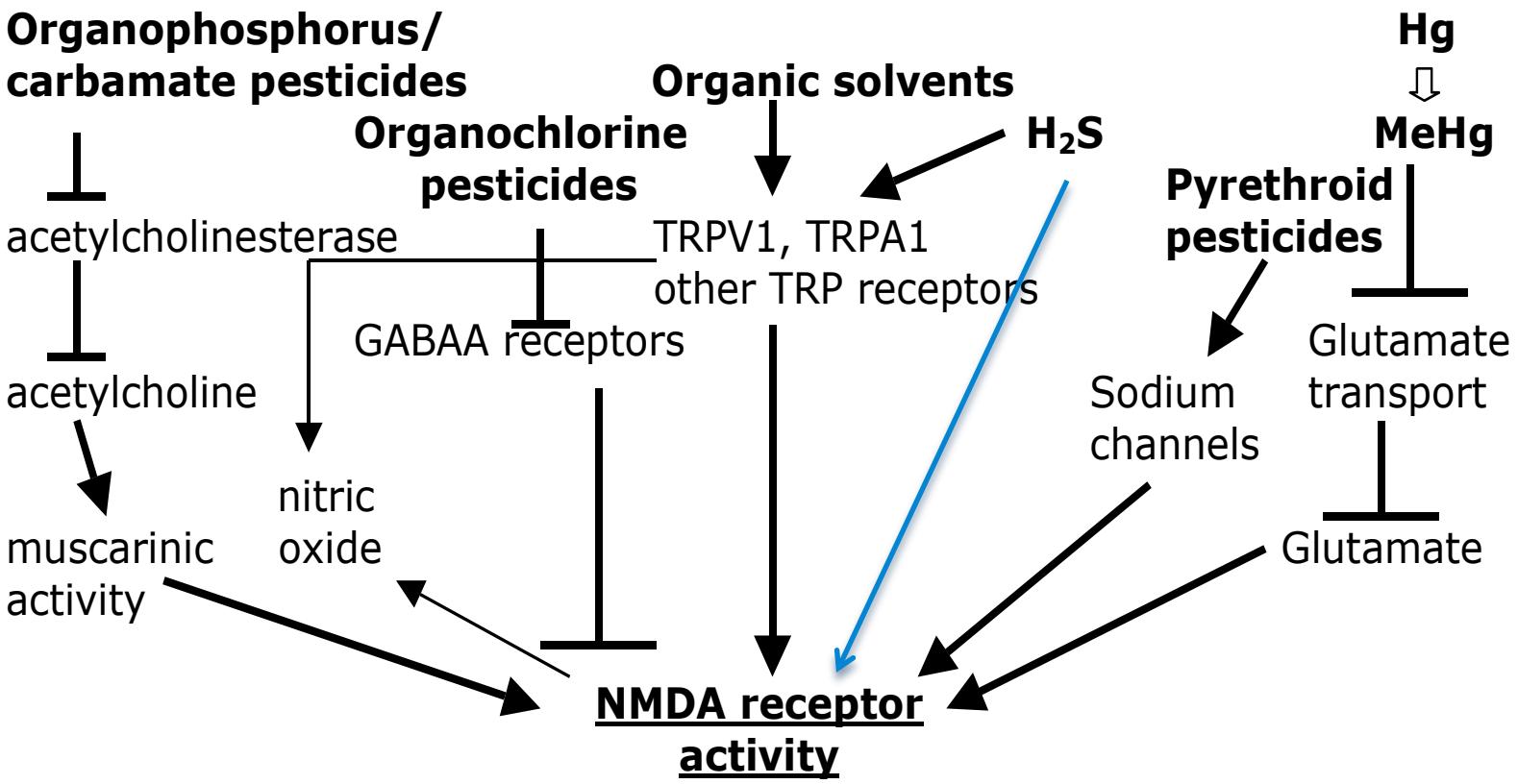


In contrast, most pathophysiological effects of NO are mediated through its role as a precursor of peroxynitrite (ONOO-), leading to free radical generation and oxidative stress.

Now, let's talk about electromagnetic hypersensitivity (EHS). Cases of EHS are thought to be caused by previous exposures to EMFs, particularly microwave/radiofrequency EMFs.

Here, one of the main sources of information on possible mechanism of EHS is what we know about the mechanism of multiple chemical sensitivity. EHS and multiple chemical sensitivity have many things in common: Cases of each can be initiated by previous exposures, of chemicals in the case of MCS and EMFs in the case of EHS with such exposures causing, then high level sensitivity responses. They are often comorbid (that is occurring in the same individuals). They both involve symptoms coming from the brain and other symptoms coming from peripheral tissues. In both, there is a lot of variation in symptoms from one individual to another, consistent with a primarily local mechanism with variable tissue distribution.

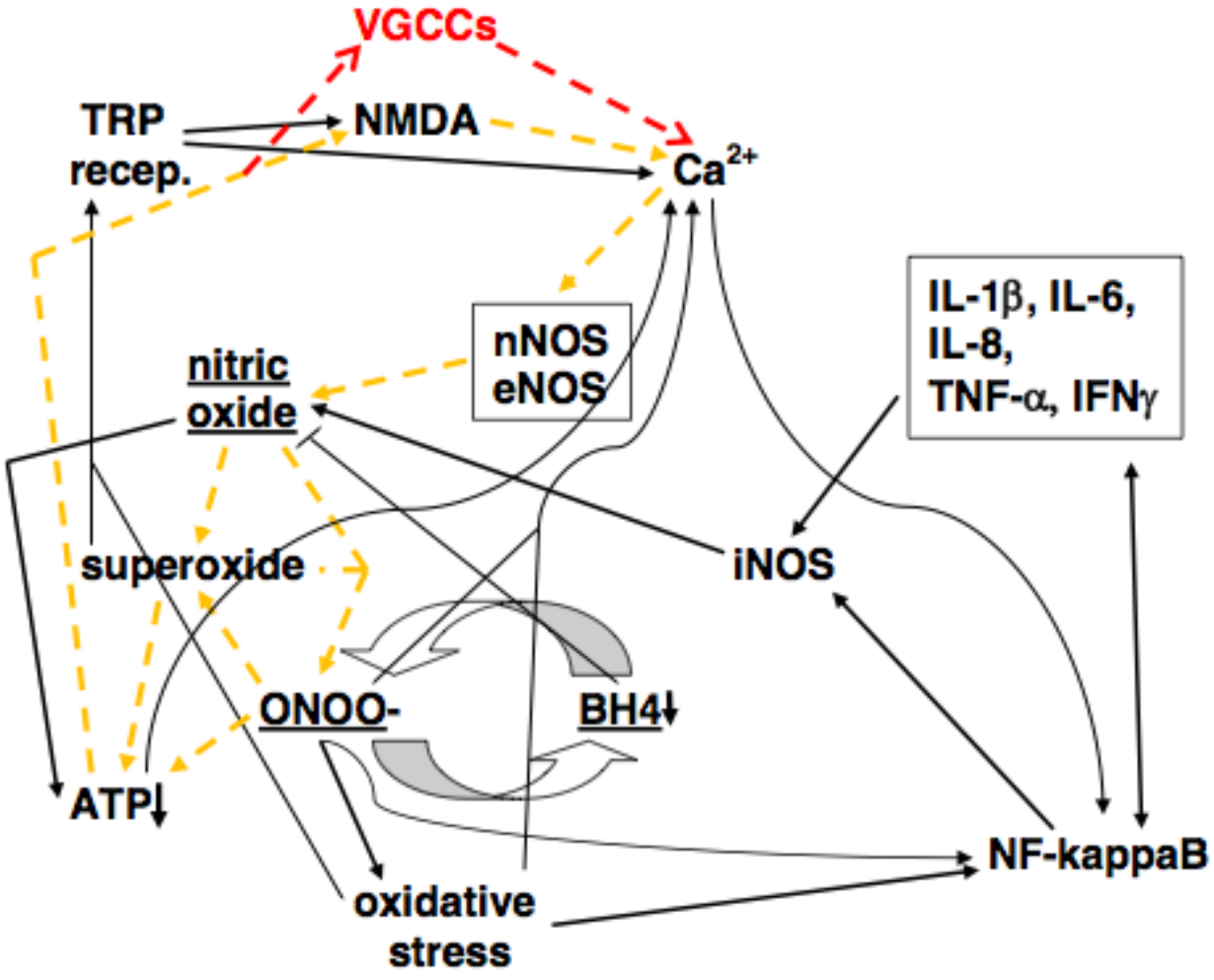
Chemical Action in MCS



There are a whole series of similarities between the NMDA receptors and the L-type VGCCs:

1. Both open up an ion channel when activated.
2. Both channels stay open a relatively long time period compared with other channels.
3. Both allow substantial amounts of calcium to flow into the cell.
4. The effects of both are thought to be mediated by excessive intracellular calcium [Ca2+]i.
5. Both lead to the production of large amounts of NO, due to the action of two calcium-dependent NO synthases, with the NO often leading to production of peroxynitrite.
6. Both have been shown to be able to stimulate long-term potentiation, the process in the central nervous system involved in learning and memory by producing neural sensitization.

It may be proposed, therefore, that all of these related similarities have roles in allowing each of them to produce the high levels sensitivities that we call multiple chemical sensitivity (MCS) or EHS. If there is one thing that is critical that you take away from this talk, it is that the similar properties of the NMDA receptors and the L-type VGCCs are almost certainly behind the two types of sensitivity!!!


VGCCs
TRP recep.
NMDA
Ca2+
nNOS eNOS
IL-1β, IL-6, IL-8, TNF-α, IFNγ
nitric oxide
superoxide
iNOS
ONOO-
BH4
ATP
NF-kappaB
oxidative stress

We think that the etiologic mechanisms of multiple chemical sensitivity is centered on two interrelated mechanisms:

1. What is called the NO/ONOO(-) cycle, a primarily local biochemical vicious cycle that is initiated by various triggers, including those acting via increased NMDA activity, and propagates itself over time.

2. And another related mechanism proposed to be involved in chemical sensitivity by Dr. Iris Bell and by others, neural sensitization caused by what is known as long-term potentiation. This can also involves NMDA receptor activity and several other mechanisms that are part of the NO/ONOO(-) cycle. Both 1 and 2 are discussed on some detail in my MCS toxicology review.

EMF exposures, by activating the L-type VGCCs should also be able to do both of these. We have already said there is a literature that the L-type VGCCs can initiate long-term potentiation, just as the NMDA receptors can. Similarly they produce large increases in intracellular calcium levels and those and downstream effects of them can act to initiate the NO/ONOO(-) cycle.

Accordingly, in the brain, EHS may be produced as follows:

- Microwave EMFs are more active in activating VGCCs in some regions of the brain than in others.
- In those regions where they are most active, they will raise [Ca2+]i, NO and peroxynitrite, starting the NO/ONOO(-) cycle going.
- That will make that area still more sensitive to additional exposures because the cycle is already started causing greater sensitivity than before.
- This will also stimulate long-term potentiation causing the synapses to become hypersensitive - therefore you have still additional sensitivity.
- Protein kinase C is also stimulated by previous exposure and the NO/ONOO(-) cycle, causing VGCCs to be still more sensitive to stimulation.

There can also be sensitivities developing in peripheral tissues that have high levels of VGCCs, such as in the cardiac pacemaker cells and in some of the endocrine cells. Here the mechanism is probably similar to what goes on in the brain except possibly there is no long-term potentiation mechanism involved.

You can get, therefore hypersensitivity in the heart (EMF-induced tachycardia) but also in some cases hypersensitivity of some of the endocrine tissues - both of these have been reported by Dr. Magda Havas.

One of the questions that should be raised, if we have time, is how so many “expert panels” have come to the conclusion that all we have to worry about is heating effects of microwave and lower frequency EMFs, when there is so much evidence to the contrary.

I looked carefully at the recent Report of the Canadian Panel of Experts which came to this same conclusion about a year ago to determine how this is possible:
https://rsc-src.ca/sites/default/files/pdf/SC6_Report_Formatted_1.pdf
Scientific evidence contradicts findings and assumptions of Canadian Safety Panel 6: microwaves act through voltage-gated calcium channel activation to induce biological impacts at non-thermal levels, supporting a paradigm shift for microwave/lower frequency electromagnetic field action. Pall ML. Rev Environ Health. 2015;30(2):99-116.

Overall conclusions:

1 The heating/thermal paradigm of action of microwave and lower frequency EMFs should be replaced by VGCC activation. We have 7 types of evidence, each clearly showing that VGCC activation is the predominant mechanism of action of such EMFs, possibly even being the whole mechanism in mammals.

2 The voltage sensor of the VGCCs has physical properties which predict that it is exquisitely sensitive to EMFs.

3 A large numbers of repeatedly reported microwave health effects can be understood as being caused by downstream effects of VGCC activation: oxidative stress; Ca^{2+} flux/signaling changes; cellular DNA strand breaks; therapeutic effects; cancer; diverse neuropsychiatric effects; male and female infertility; breakdown of the blood-brain barrier; neuroendocrine effects including melatonin deficiency; cardiac pacemaker effects leading to arrhythmia and sudden cardiac death.

4 It can be seen, from the above, that low level EMF exposures attack each of the 4 things we often value most as individuals and as a species: Our health, our brain function, the integrity of our genomes and our ability to produce healthy offspring.

5 The mechanism of EHS may be similar to that of MCS, with the main targets of EMFs being the L-type VGCCs.

6 Biologically based safety standards must be developed; the best way to do this is to study the response of cells in culture to EMFs, cells that have high densities of VGCCs.

7 With ever increasing exposures to microwave EMFs, 5 worse case scenarios each suggest that such exposures can rapidly lead to the crash of human civilization.