Electromagnetic Radiation and Epilepsy

Andy Davidson, Tetrawatch

Abstract

People report adverse health symptoms which they attribute to the radiation of microwave signals from mobile phone and TETRA masts. The validity of such reports cannot be safely rejected, either on the grounds of current safety compliance, nor on the grounds that such symptoms cannot be replicated in laboratory experiments in search of a simplified mechanism.

Biological effects caused by such electromagnetic fields (EMF) are widely attested by research, but there is lack of clarity about how this occurs and why certain individuals should be more susceptible than others. In the case of electrical hypersensitivity, and in the particular, though more rare, case of exacerbated or instigated epilepsy, there are two candidate mediating factors: (i) the direct effects of coherent frequencies, and (ii) disruption of the nitric oxide synthesis process. This paper takes the second, and examines how the potential causal link is borne out by both likely and observed subject outcomes, and by observed EMF/nitric oxide synthase interactions. There is sufficient suspicion that a mechanism lies here to help validate the personal reports, and therefore action should be taken to verify that operation of such EMF sources is not the cause, rather than requiring the affected person to prove that it is.

There have been many reports from people living in proximity to mobile phone and TETRA masts, of adverse health effects. One of these effects is increased incidence of epileptic seizures, and indeed onset of epilepsy. Many of these effects relate to what has come to be referred to as electrical hypersensitivity or EHS. These medical reports largely go uninvestigated, for two reasons:

1. the symptoms are wide ranging, from headaches and nausea to sleep disturbances and nosebleeds, but are categorised as common and minor complaints
2. there is no laboratory confirmed conclusion as to precisely why or how such effects should be induced at such low signal energies.

On the first count, the complaints may appear to be minor, since many of us have one or another of them from time to time. This ignores that they may be unusual to the people concerned, or correlate to the onset of a transmitter's operation, or relate to particular signal levels (Oberfeld *et al.*, 1998; Santini *et al.*,2002; Navarro *et al.*, 2003) or cease when operation is suspended. It also ignores the manner in which they suddenly become chronic and persistent for the people concerned, and the resultant effect on well-being.

On the second count, there has been a widely held presumption that the mechanism might be simple and single-stranded. If this were so, the cause might be isolated, it is argued, and a direct cause and effect be demonstrated *in vitro*, and that this should be easily replicated. Primary refutations of this approach include (i) the complex electromagnetic environment caused even by a single transmission base station within the natural electromagnetic environment (Silk, 2004), (ii) the effects on the whole body, or significant parts of the body (*eg* Hyland, 2003; Silk, 1999), and (iii) lag effects, where separate results from different effects from common causes have separate onsets, but combine under exposure times longer than laboratory exposure tests.

Much has been written about the difficulties of *in vivo* laboratory set-ups to test the validity of claims to EHS, even though the manifestation of EHS is itself irrefutable. Nonetheless, the symptoms are separated from the attributed electromagnetic cause by reviewers not least because either a mechanism is not apparent and there is no assured laboratory-replicability. In the case of epilepsy, once it was realised that light-pulse frequency from flashbulbs, disco strobes, television or video games could induce seizures, this was accepted, warnings made mandatory, and advice given. We now know better why the effect occurs, and in particular that frequency is a central factor.

Taking reports seriously

Technology has raced ahead in recent decades, providing much opportunity for people to resist, either because they feel they cannot keep up, because it is becoming too complicated, or because its novel nature might not be all good. Communications technology has been adopted enthusiastically by most people, with its obvious advantages. Disadvantages are mostly reported as being cost, manners/etiquette, over-independence by youngsters, bullying, commercial advantage being taken by operator companies, the unsightly physical appearance of masts *etc.* It is not therefore immediately apparent quite why a Luddite tendency should emerge, based on grounds of fears of endangerment to health. And yet the primary reason for not listening and responding to very ordinary people who report adverse health reactions when masts become operational near their homes, is that it must be some form of induced hysteria generated by fear of new technology. This would appear to be poor scientific justification for a lack of intervention or even scientific curiosity.

Are reports of increased or induced epileptic seizures in some way psychosomatic?

The obvious answer would have to be no, especially in cases of children unaware of the issues or even of the presence of roof-top antennae. For this reason alone, the reports must be given due attention.

Perhaps the causality is only partly to do with the operation of base stations? Perhaps some other environmental factor comes into play when they are introduced? The most curious aspect of the whole issue of these adverse health reactions is that there is no epidemiological investigation done in the UK at all.

There is no other response than that attribution to base station must be false. Worse, the single most common premise, that base stations operate well within international guidelines for exposure, completely ignores the now well-known fact that the guidelines[1] expressly relate only to acute short-term effects of heating by the radiation, and neither to chronic exposure nor non-thermal biological effects. The guidelines explain that at the time of their devising, there was insufficient research material upon which to construct practical exposure levels for protection from potential longer-term, low-level influences on biological functions. Nevertheless, such effects are well-attested and expressed as such in the UK government's own advice from the Independent Expert Group on Mobile Phones and Health (IEGMP, 2000; committee chaired by Sir William Stewart, updated and reiterated NRPB, January 2005).

It is therefore entirely reasonable to take correlations of reported effects and attributed sources from base stations, where there is a common theme (such as EHS symptoms or epilepsy) and to investigate them thoroughly.

Returning to the fundamental question: can exposure to low level EMF have implications for epileptic seizures?

Possible causal relations

How can electromagnetic fields (EMF) from mobile phone or TETRA base stations impact on the human body? This is a vital question, and several routes can be reasonably pursued:

1. Since currents can be induced in the body by external varying fields, and since the body is both conductive and employs DC and AC currents at minute levels for its messaging, direct electrical interference may occur (*eg* Becker, 1985 & 1990; Lai, 1994).
2. The human body has evolved to utilise a number of frequencies key to its operations, such as 'brain waves' (which in fact permeate the body), pulse rate *etc.* It is possible that induced currents at particular frequencies present significant influences, including issues of entrainment, whereby biological frequencies 'lock on' to external drivers. (Brain entrainment happens internally between neurons, but is used via acoustic coupling — of binaural beats — for therapy or meditation.)
3. Since everything with bounded mass has its own natural or harmonic resonance frequency relating to its dimension, incident frequencies may cause resonance at atomic level, or ionic, molecular, cellular, organ, cavity (*eg* skull, eye sockets, heart chamber), extension (*eg* limbs) or whole body levels (*eg* Silk, 1999; Bruel and Kjaer, 1982).

---

[1] *ie*, from the International Commission of Non-ionising Radiation Protection – ICNIRP

4. Since resonance with biological processes naturally depends on interference with the body's active whole-body processes, investigation of the whole person is more likely to yield meaningful results than isolated tissue/culture samples, therefore ambivalent results from the latter may not be informative (Hyland, 2003).
5. Nonetheless, understanding ionic resonance and its role in chemical progression is an important factor, in the context of geomagnetic fields (Becker, 1990 citing Blackman and Liboff, and Adey).
6. The body contains many bioelectronic features, since by nature it operates and is maintained by subtle and complex circuits (*eg* Oschmann, 2000; Becker, 1989). The electronic properties of cell walls and cellular structures, the semiconductance of neuronal epithelial cells, the growth-guiding piezo-electric effect of bone, the DC and AC pathways of the nervous system, semiconductance in DNA and electrical frequency maintenance by the brain, whilst they cannot be construed as constituting a radio receiver, nonetheless imply acute sensitivity to external EMFs, and these may interact in ways novel to the normal operation of biochemical and physiological functions (Williams, 2002).
7. The body contains crystalline structures, including calcite and magnetite, which may produce piezo-electric effects under external fields (Baconnier *et al.,* 2002; Lang, 2003; Kirschvink, 1989, 1992).

This subtlety does make the scenario appear quite complex, and indeed it is. Nonetheless, any view of the body as a purely chemical entity, or even one in which the electrical activity is a minority influence, would be quite incorrect. Much research has demonstrated, not all conclusively, that chemical processes regulated by electrical stimulus are indeed disrupted by EMFs, and that these can occur most strongly within power and frequency 'windows', *ie*, outside certain ranges, the effects are less evident or absent. What has attracted most attention is the idea that EMFs may cause, promote or lead to such serious diseases cancer or motor neurone disease. These are not dealt with here, but introduced to compare how little attention has been paid to 'less important' outcomes such as EHS and epilepsy.

It may well be that directly induced currents and consequent electrical interference are a primary cause of epileptic seizures in some people, especially where the frequency of the imposed signal corresponds to trigger frequencies for the individual's epileptic characteristics. Perhaps this occurs via the hippocampus. Mobile phone radiation, especially GSM and TETRA have come into question here, since both employ time division signal structures (TDMA) that give rise to extremely low frequency pulse rates in both handsets and base stations. It is important to assert here that any argument that TETRA base stations, for example, do not pulse are purely semantic. The signal envelope (shape) is clearly modulated at 70.6Hz with 0.9Hz repetitions, and this signal is visible under simple signal rectification with a resonant circuit that is not highly tuned. GSM base stations (including DECT – digitally enhanced cordless telephones) similarly employ extremely low frequency pulse and frame rates.

Thus the signal characteristics of base stations are complex, presenting to the body a combination of carrier waves in the microwave band, the interference patterns between these carrier frequencies, and the signal structures themselves, all within the context of the earth's geomagnetic EMFs, including the Schumann resonance with which our bodies appear to be closely and naturally tuned, and our own internal bioelectromagnetic-frequencies. Pinning one factor down as a predominant or prime cause for any specific effect is therefore difficult, but it is unavoidable that some biologically significant effects might well be taking place, given that our exposure to EMFs in towns and cities is running at 10^{15} times natural pre-industrial levels, and right across the EM frequency band (Philips A, Powerwatch UK).

A significant alternative approach for EMF and epilepsy

Epilepsy is caused by abnormal electric impulses in groups of neurons in the brain. Epilepsy is diagnosed under two main types, 'symptomatic' (*ie* the reason is known, *eg* head trauma) and 'idiopathic'. In cases of this latter type, the cause is unknown. However, attacks are understood to be caused by a lack of neurotransmitters that regulate the electric impulses in the brain. The resultant aberrant electrical activity produces the seizures.

Perhaps if we can say too little that is definitive and conclusive about induced current and frequency effects causing epilepsy (whilst we can say a very great deal indeed about possible consequences), we can at least point to another potential cause. If we start from the direction of the neurotransmitters, we might be able to identify the reason *why* one key neurotransmitter might be disrupted.

Nitric oxide is rapidly becoming one of the most researched molecules in the body, since it plays such a central role in so many biological processes. One of these roles is as a neurotransmitter or signalling molecule. Unlike most neurotransmitters, NO is not stored. Instead it is synthesised on demand from l-arginine by the enzyme nitric oxide synthase (NOS). The nitric oxide itself is transient, being rapidly converted into superoxide and peroxinitrite. Two forms of nitric oxide synthase that are relevant here are inducible (iNOS) and neuronal (nNOS). In both cases, where the NOS is up-regulated (*ie* it over-produces), there is a link with epileptic seizures (Itoh *et al.*, 2004).

Further, acetylcholenesterase is particularly implicated in temporal lobe epilepsy (*eg* Green at *al.*, 1989; Leonard, 1989), and this in turn is regulated by serotonin, which in turn is modified by nitric oxide (*eg* Fossier *et al.*, 1999).

Epilepsy, nitric oxide and the link to EHS

There is growing clarification about the relationship between nitric oxide and epilepsy, but at this point it is noteworthy to return to the beginning of this paper, and ask about the other reported adverse health symptoms attributed to mobile phone and TETRA masts. Grouped under the EHS heading, these include

nosebleeds, unusual headaches, itchy skin and rashes, nausea, dizziness, unusual sleep disruptions coinciding with REM sleep periods, and fatigue.

Surprisingly, if the nitric oxide synthase reaction were to be *deliberately* disturbed, all these symptoms would likely be produced, since each is underlaid by one role or another of nitric oxide, or its products, superoxide and peroxynitrite:

- nitric oxide affects blood platelets (*eg* Alonso, 2003), reducing clotting, and is also a vasodilator, and the capillaries of the nasal cavity are particularly delicate
- nitric oxide imbalance is a prime cause of headaches (as a vasodilator) (Jensen, 2003)
- nitric oxide is involved in skin mast cells producing histamine (Johansson, 2004)
- nitric oxide regulates cerebral blood flow, creating sensations of nausea and dizziness, and affects oxygenation levels on haemoglobin, in a manner similar to altitude sickness (Huber *et al.*, 2002; Dumont *et al.*, 2003)
- nitric oxide is a REM sleep regulator (Faradji *et al.*, 2000; Mann *et al.*, 1996), perhaps via acetylcholenesterase (Leonard, 1997) (which is also known to be affected by exposure to EMF) via modification of serotonin by nitric oxide (Fossier, 1999). Serotonin is a precursor of melatonin, which again has been noted to be suppressed by exposure to microwave band EMF (Cherry, 2000) in the same way as it is by visible light band of EMF; and of course melatonin is a natural sleep cycle regulator, as well as a free radical scavenger. (NO itself and its products are free radicals, and EMF has also been shown to extend the life of free radicals (Toshikawa, 2000).)
- overproduction of iNOS leads to excessive peroxynitrite, which is highly oxidative. The body appears to respond by limiting its precursor, superoxide, by closing down energy production (ATP) because this also produces superoxide. This results in the fatigue (Pall, 2003).

In fact, EHS symptomatology closely mirrors that of multiple chemical sensitivity and aspects of chronic fatigue syndrome, and these conditions are also under scrutiny regarding the role of nitric oxide (Pall, 2003).

Clearly, nitric oxide could produce all of the general adverse health effects attributed to mobile phone and TETRA base stations. Is there any evidence that by whatever mechanism, that EMFs can disrupt production of nitric oxide synthase?

The following are some suggestive abstracts of relevant studies.

Effects of electromagnetic radiation from a cellular telephone on the oxidant and antioxidant levels in rabbits.

Irmak MK, Fadillioglu E, Gulec M, Erdogan H, Yagmurca M, Akyol O

Cell Biochem Funct. 2002 Dec; 20(4):279-83

The number of reports on the effects induced by electromagnetic radiation (EMR) in various cellular systems is still increasing. Until now no satisfactory mechanism has been proposed to explain the biological effects of this radiation. Oxygen free radicals may play a role in mechanisms of adverse effects of EMR. This study was undertaken to investigate the influence of electromagnetic radiation of a digital GSM mobile telephone (900 MHz) on oxidant and antioxidant levels in rabbits. Adenosine deaminase, xanthine oxidase, catalase, myeloperoxidase, superoxide dismutase (SOD) and glutathione peroxidase activities as well as nitric oxide (NO) and malondialdehyde levels were measured in sera and brains of EMR-exposed and sham-exposed rabbits. Serum SOD activity increased, and serum NO levels decreased in EMR-exposed animals compared to the sham group. Other parameters were not changed in either group. This finding may indicate the possible role of increased oxidative stress in the pathophysiology of adverse effect of EMR. Decreased NO levels may also suggest a probable role of NO in the adverse effect.

Increase in nitric oxide and cyclic GMP of rat cerebellum by radio frequency burst-type electromagnetic field radiation

M Miura, K Takayama and J Okada

Department of Physiology First Division, Gunma University School of Medicine, Maebashi-shi, Japan.

The Journal of Physiology, Vol. 461, Issue 1: 513-524, 1993

1. Using rat cerebellum supernatant, the effects of radio frequency (RF) burst-type electromagnetic (EM) field radiation on the production of cyclic GMP were examined under various conditions. The radiation was generated by a generator coil, and set at a 10 MHz radiation frequency, a 50% burst time, a 10 kHz burst rate and a 5 V peak-to-peak generator voltage. 2. When the cerebellum supernatant was incubated with both exogenous L-arginine (nitric oxide (NO) donor) and NADPH, and irradiated by an RF burst-type EM field, the production of cyclic GMP was increased significantly from a level of 21-22 nmol min-1 (g tissue)- 1 to 25-26 nmol min-1 (g tissue)-1. By contrast, such an effect was not found when the cerebellum supernatant was irradiated by an RF volley-type EM field. 3. When neither L-arginine nor NADPH were added to the cerebellum supernatant, the production of cyclic GMP was lowered to a

level of 6 nmol min-1 (g tissue)-1 and the radiation effect was not found. When the cerebellum supernatant was chelated with EDTA, the production of cyclic GMP was lowered to a level of 7 nmol min-1 (g tissue)-1 and the radiation effect was not found. 4. Incubation with Methylene Blue, a guanylate cyclase inhibitor, lowered the production of cyclic GMP to a level of 10-12 nmol min-1 (g tissue)-1, and the radiation effect did not occur. On incubation with a NO synthase inhibitor, either NG-methyl-L-arginine or N omega-nitro-L-arginine methyl ester, the production of cyclic GMP was lowered to a level of 10- 12 nmol min-1 (g tissue)-1 or 5-9 nmol min-1 (g tissue)-1 respectively, and the radiation effect was not observed. **5. Using electrochemical NO probes, the production of NO in the cerebellum supernatant was detected. The concentration of NO increased gradually after the onset of the EM field radiation. The radiation effect persisted, and reached a maximum after the cessation of the radiation. 6. In an in vivo study, the arterioles of the frog web were dilated by the radiation, and this radiation effect was almost completely abolished by the addition of a NO synthase inhibitor. This indicates that radiation activates NO synthase and ultimately induces vasodilatation.**

Litovitz (1993) and Penafiel, Litovitz et al. (1997) have noted the reduction in another enzyme, ornithine decarboxylase, this is itself inhibited by nitric oxide, under modulated EMF.

Paredi *et al.* (**Local vasodilator response to mobile phones,** 2001) noted:

> There was a similar and significant increase in skin temperature of the nostril and occipital area on the same side as the telephone (maximal increase 2.3 +/- 0.2 degrees C at 6 min) as well as a tendency for higher nasal NO levels (maximal increase 12.9 +/- 4.9% at 10 min), whereas the MCA was significantly reduced (maximal decrease -27 +/- 6% at 15 min). Such changes were not recorded when an earpiece was used to avoid the direct exposure to the electromagnetic field. There were no changes in the skin temperature and nasal NO measured on the opposite side to the mobile phone, whereas the MCA was significantly increased (38 +/- 10%). CONCLUSIONS: Exposure to EMF produced by a mobile phone produces biological effects that can be easily measured. Microwaves may increase skin temperature and therefore cause vasodilation and reduce MCA. Further studies are needed to study the long-term effects of mobile phone use and the relation among NO production, vasodilation, and temperature.

Comment: What they did not test as an hypothesis, is the reverse: that the EMF causes the vasodilation through stimulation of iNOS, causing the skin temperature change as a secondary effect.

Enhanced expression of neuronal nitric oxide synthase and phospholipase C-gamma1 in regenerating murine neuronal cells by pulsed electromagnetic field

Kim SS, Shin HJ, Eom DW, Huh JR, Woo Y, Kim H, Ryu SH, Suh PG, Kim MJ, Kim JY, Koo TW, Cho YH, Chung SM (2002), *Exp Mol Med*., 34(1):53-9

Pulsed electromagnetic field (PEMF) has been shown to improve the rate of peripheral nerve regeneration. In the present study we investigated the expression of neuronal nitric oxide synthase (nNOS) and phospholipase C-gamma1 (PLC-gamma1) in regenerating rat laryngeal nerves during the exposure to PEMF after surgical transection and reanastomosis. Axons were found to regenerate into the distal stump nearly twice faster in PEMF-exposed animals than in the control. Consistently, motor function was better recovered in PEMF-treated rats. The expression of nNOS and PLC-gamma1 was highly enhanced in the regenerated nerves.

The relevance in this next study is that superoxide dismutase is the enzyme that mops up superoxide as a product of nitric oxide synthesis to prevent oxidative damage:

Effect of electromagnetic field produced by mobile phones on the activity of superoxide dismutase (SOD-1) and the level of malonyldialdehyde (MDA)--in vitro study

Stopczyk D, Gnitecki W, Buczynski A, Markuszewski L, Buczynski J. *Med Pr.* 2002;53(4):311-4 (Poland)

The aim of the study was to assess in vitro the effect of electromagnetic field produced by mobile phones on the activity of superoxide dismutase (SOD-1) and the level of malonyldialdehyde (MDA) in human blood platelets. The suspension of blood platelets was exposed to the electromagnetic field with the frequency of 900 MHz for 1, 3, 5, and 7 min. Our studies demonstrated that microwaves produced by mobile phones significantly depleted SOD-1 activity after 1, 5, and 7 min of exposure and increased after 3 min in comparison with the control test. There was a significant increase in the concentration of MDA after 1, 5, and 7 min and decrease after 3 min of exposure as compared with the control test. **On the grounds of our results we conclude that oxidative stress after exposure to microwaves may be the reason for many adverse changes in cells and may cause a number of systemic disturbances in the human body.**

Enhancement of nitric oxide generation by low frequency electromagnetic field.

Yoshikawa T, Tanigawa M, Tanigawa T, Imai A, Hongo H, Kondo M.

First Department of Internal Medicine, Kyoto Prefectural University of Medicine, Kawaramachi Hirokoji, Kamigyou-ku, 602-8566, Kyoto, Japan

Oxidative stress is implicated in the intracellular signal transduction pathways for nitric oxide synthase (NOS) induction. The electromagnetic field (EMF) is believed to increase the free radical lifespan [S. Roy, Y. Noda, V. Eckert, M.G. Traber, A. Mori, R. Liburdy, L. Packer, The phorbol 12-myristate 13-acetate (PMA)-induced oxidative burst in rat peritoneal neutrophils is increased by a 0.1 mT (60 Hz) magnetic field, FEBS Lett. 376 (1995) 164-6; F.S. Prato, M. Kavaliers, J.J. Carson, Behavioural evidence that magnetic field effects in the land snail, Cepaea nemoralis, might not depend on magnetite or induced electric currents, Bioelectromagnetics 17 (1996) 123-30; A.L. Hulbert, J. Metcalfe, R. Hesketh, Biological response to electromagnetic fields, FASEB 12 (1998) 395-420]. We tested the effects of EMF on endotoxin induced nitric oxide (NO) generation in vivo. Male BALB/C mice were injected with lipopolysaccharide (LPS) intraperitoneously (i.p.), followed by the exposure to EMF (0.1 mT, 60 Hz). Five hours and 30 min after the LPS administration, mice were administered with a NO spin trap, ferrous N-methyl-D-glucaminedithiocarbamate (MGD-Fe). Thirty minutes later, mice were sacrificed, and their livers were removed. The results were compared to three control groups: group A (LPS (-) EMF(-)); group B (LPS(-) EMF(+)); group C (LPS(+) EMF(-)). The ESR spectra of obtained livers were examined at room temperature. Three-line spectra of NO adducts were observed in the livers of all groups. In groups A and B very weak signals were observed, but in groups C and D strong spectra were observed. The signal intensity of the NO adducts in Group D was also significantly stronger than that in Group C. EMF itself did not induce NO generation, however, it enhanced LPS induced NO generation in vivo.

Further similar work continues at the Laboratory of Ecology, Ukraine.

Is Nitric Oxide Involved in the Anticonvulsant Action of Antiepileptic Drugs?

Katarzyna Wojtal, Anna Gniatkowska-Nowakowska, Stanislaw J. Czuczwar

Experimental data indicate that nitric oxide (NO) may play a role in the pathophysiology of epilepsy. It is also possible that NO-mediated events are involved in the expression of the anticonvulsant action of some antiepileptics. The aim of this review was to assemble current literature data on the role of NO in the anticonvulsant action of antiepileptic drugs (AEDs). The influence of various NO synthase inhibitors (NOSI) on antiseizure activity of AEDs was tested in many animal experimental models of epilepsy (electrically and

pharmacologically evoked seizures, sound-induced convulsions, amygdala-kindled seizures). Although some NOSI were able to modify the anticonvulsive properties of AEDs, the involvement of NO pathway in the mechanisms of action of AEDs in most cases does not seem probable, since the effects of NOSI were not reversed by L-arginine, a NO precursor.

There are thus clear indications that there is a noted response in nitric oxide synthesis under certain conditions of pulse or amplitude modulated microwave radiation. We also have the results of the TNO study of 3G signals, in the Netherlands (Zwamborn *et al.*, 2003), indicating adverse reactions to microwave EMF of mobile phones, including cognitive function, which is perhaps influenced by nitric oxide it its role as vasodilator of brain blood vessels. (NO is noted for example as therapeutic in treatments for Alzheimer's disease.) Similarly, we have the latest results from Austria indicating that exposure to EMF from actual mobile phone masts alters the EEG of sensitive individuals in blind trials (Oberfeld, 2005). The human body does respond to signals such as from mobile communications masts.

Conclusion

From the above discussion it should be apparent that whilst the mechanisms, the reasons for individual susceptibilities, and the predictability of adverse reactions to microwave communications have not been elucidated, it is *unreasonable* to suppose that reports of adverse reactions at this level are insupportable on grounds of compliance by transmitters to safety guidelines for *acute thermal effects* of EMR.

It is also entirely reasonable that given the connections between epilepsy (for example), nitric oxide synthesis and related chemical processes, and the physiological influence of EMR, this remains a rational contributor to cause and effect in a susceptible individual. Other effects noted above may play an assistive or separate role in triggering seizures.

Therefore, when such an effect as increased (or onset of) seizures is observed in clear correlation to the operation and location of a communications base station, the attribution should be taken seriously unless it can be redirected to any better known direct cause that also correlates with the operation of the base station. The most reasonable response to test cause and effect would be to terminate operation of the base station for the benefit of the individual, and should this have no effect at all, only then seek an alternative explanation. Such an action is not difficult, and can be done in controlled conditions with accompanying environmental EMF monitoring. If by way of objection this might be regarded as unethical experimentation, it calls into question the circumstances whereby these adverse health reports are allowed to go uninvestigated.

A short sample bibliography

Alonso D, M W Radomski (2003), 'Nitric oxide, platelet function, myocardial infarction and reperfusion therapies', *Heart Fail Rev* 8(1): 47-54

Baconnier S, Lang SB, Polomska M, Hilczer B, Berkovic G, Meshulam G (2002), 'Calcite microcrystals in the pineal gland of the human brain: First physical and chemical studies', *Bioelectromagnetics*, 23(7):488-495

Becker R O (1989), *The Body Electric*, Quill

Becker R O (1990), *Cross Currents: The perils of electropollution, the promise of electromedicine*, Tarcher/Penguin

Bruel and Kjaer (1982), *Human Body Vibration: Technical Review*, Denmark

Cherry N (2000), 'EMR Reduces Melatonin in Animals and People', Lincoln University, Canterbury, New Zealand

Dumont L, Lysakowski C, *et al.* (2003), 'High altitude cerebral oedema', *Ann Fr Anesth Reanim* 22(4): 320-4

Faradji H, Rousset C, Debilly G, Vergnes M, Cespuglio R (2000), 'Sleep and epilepsy: A key role for nitric oxide?', *Epilepsia,* 41(7):794-801

Fossier P, Blanchard B, Ducrocq C, Leprince C, Tauc L, Baux G (1999), 'Nitric oxide transforms serotonin into an inactive form and this affects neuromodulation', *Neuroscience,* 93(2):597-603

Gilad E *et al.* (1998), 'Melatonin inhibits expression of the inducible isoform of nitric oxide synthase in murine macrophages: role of inhibition of NFκB activation', *FASEB Journal*, 12:685-693

Gilberta M E (2001), 'Does the Kindling Model of Epilepsy Contribute to Our Understanding of Multiple Chemical Sensitivity?', *Annals of the New York Academy of Sciences* 933:68-91

Green R C, Blume H W, Kupferschmid S B, Mesulam M M (1989), 'Alterations of hippocampal acetylcholinesterase in human temporal lobe epilepsy', *Ann Neurol*., 26(3):347-51

Huber *et al.* (2002), 'Electromagnetic fields, such as those from mobile phones, alter regional cerebral blood flow and sleep and waking EEG', *J Sleep Res* 11(4):289-295

Hyland G, (2003), 'How Exposure to GSM & TETRA Base-station Radiation can Adversely Affect Humans', available variously: May 2003 update: http://www.carolinelucasmep.org.uk/interests/pdf/Hyland_TETRA_May2003.pdf

IEGMP (2000), *Mobile Phones and Health*, (the 'Stewart Report')

NRPB (2005), *Mobile Phones and Health*, Documents of the NRPB: 15, 5, 2004

Irmak M K, Fadillioglu E, Gulec M, Erdogan H, Yagmurca M, Akyol O (2002), 'Effects of electromagnetic radiation from a cellular telephone on the oxidant and antioxidant levels in rabbits', *Cell Biochem Funct*.; 20(4):279-83

Itoh K, Watanabe M, Yoshikawa K, Kanaho Y, Berliner L J, Fujii H (2004), 'Magnetic resonance and biochemical studies during pentylenetetrazole-kindling development: the relationship between nitric oxide, neuronal nitric oxide synthase and seizures', *Neuroscience*, 129(3):757-66

Jensen R (2003), 'Peripheral and central mechanisms in tension-type headache: an update', *Cephalalgia* 23 Suppl 1: 49-52

Jeyarasasingam G, Yeluashvili M, Quik M (2000), 'Nitric Oxide Is Involved in Acetylcholinesterase Inhibitor-Induced Myopathy in Rats', *Journal of Pharmacology and Experimental Therapeutics*, Vol. 295, Issue 1, 314-320

Johansson O (2004), 'Screen dermatitis and electrosensitivity: Preliminary observations in the human skin', *Electromagnetic Environments and Health in Buildings* (ed. D Clements-Croome), Spon Press, London & New York, pp 377-389

Kelm M (2003), 'The L-arginine-nitric oxide pathway in hypertension', *Curr Hypertens Rep* 5(1): 80-6

Kim S S, Shin H J, Eom D W, Huh J R, Woo Y, Kim H, Ryu S H, Suh P G, Kim M J, Kim J Y, Koo T W, Cho Y H, Chung S M (2002), 'Enhanced expression of neuronal nitric oxide synthase and phospholipase C-gamma1 in regenerating murine neuronal cells by pulsed electromagnetic field', *Exp Mol Med*., 34(1):53-9

Kirschvink J L (1989), 'Magnetite Biomineralization and Geomagnetic Sensitivity in Higher Animals: An Update and Recommendations for Future Study', *Bioelectromagnetics*, 10 (1989):239-259

Kirschvink J L *et al.* (1992), 'Magnetite biomineralization in the human brain', *Proceedings of the National Academy of Sciences*, 89 (1992):7683-7687

Lai H (1994), 'Neurological effects of microwave irradiation', in: *Advances in Electromagnetic Fields in Living Systems,* Vol. 1, JC Lin (ed.), Plenum Press, New York, pp. 27-80

Lang S B (2003), 'Calcite Microcrystals in the Pineal Gland of the Human Brain: Second Harmonic Generators and Possible Piezoelectric Transducers', Lecture, Monash University 16th January
(http://www.spme.monash.edu.au/seminars/s160103.html)

Leonard T O, Lydic R (1989), 'Alterations of hippocampal acetylcholinesterase in human temporal lobe epilepsy', *Ann Neurol*.;26(3):347-51

Leonard T O, Lydic R (1997), 'Pontine nitric oxide modulates acetylcholine release, rapid eye movement sleep generation, and respiratory rate', *J Neurosci*. 15;17(2):774-85

Litovitz T A, Krause D, Mullins JM (1993), 'The role of coherence time in the effect of microwaves on ornithine decarboxylase activity', *Bioelectromagnetics* 14: 395-403

Mann K, Roschke J (1996), 'Effects of pulsed high-frequency electromagnetic fields on human sleep', *Neuropsychobiology* 33: 41-47

Miura M, Takayama K, Okada J (1993), 'Increase in nitric oxide and cyclic GMP of rat cerebellum by radio frequency burst-type electromagnetic field radiation', *The Journal of Physiology*, Vol. 461, Issue 1 513-524

Navarro E A *et al.* (2003), 'About the Effects of Microwave Exposure from Cellular Phone Base Stations: a first approach', *Electromagnetic Biology and Medicine*, 22: 161-169

Oberfeld G *et al.* (1998), 'The Microwave Syndrome: A preliminary Study in Spain', *Arch. Environ. Health*, 53:236-238

Oberfeld G (2005), 'Strahlung von Mobilfunksende-Anlagen beeinflussen Gehirnströme', Salzburger Landeskorrespondenz, 27 April

Oschmann J (2000), *Energy Medicine: The scientific basis*, Churchill Livingstone

Pall M (2003), 'Elevated Nitric Oxide/Peroxynitrite Theory of Multiple Chemical Sensitivity: Central Role of N-Methyl-D-Aspartate Receptors in the Sensitivity Mechanism', *Environmental Health Perspectives*, Vol. 111, 12, pp 1461-146

Paredi P, Kharitonov S A, Hanazawa T, Barnes P J (2001), 'Local vasodilator response to mobile phones', *Laryngoscope*, 111(1):159-62

Penafiel M L, Litovitz T A, Krause D, Desta A, Mullins J M (1997), 'Role of modulation on the effect of microwaves on ornithine decarboxylase activity in L929 cells', *Bioelectromagnetics* 18:132-41

Przewlocka B, Lason W, Van Luijtelaar E L J M, Coenen A M L, Przewlocki R (1996), 'The role of nitric oxide in genetic model of absence epilepsy in rats', *Neuroscience Research Communications*, 18 (2), 125-131

Raevskii K S, Bashkatova V G, Vanin A F (2000), 'The role of nitric oxide in brain glutaminergic pathology', *Vestn Ross Akad Med Nauk.* (4):11-5

Santini R, Santini P, Danze J M, Le Ruz P, Seigne M (2002), 'Symptoms experienced by people in vicinity of base station : I. Incidences of distances and sex', *Pathol. Biol.*

Silk A (2004), *Mobile Communications and Interactions with Further RF & EMF Signal Systems*, submission to All-Party Mobile Group (ApMobile)

Silk A (1999), 'Mobile Phones and Human Exposure', *Optometry Today*, April 23

Stopczyk D, Gnitecki W, Buczynski A, Markuszewski L, Buczynski J (2002), 'Effect of electromagnetic field produced by mobile phones on the activity of superoxide dismutase (SOD-1) and the level of malonyldialdehyde (MDA)', *Med Pr.* 53(4):311-4

Testylier *et al.* (2002), 'Effects of exposure to low level radiofrequency fields on acetylcholine release in hippocampus of freely moving rats', *Bioelectromagnetics* 23:249-255

Williams J M (2002), *Thermal and Nonthermal Mechanisms of the Biological Interaction of Microwaves*, arXiv

Wojtal K, Gniatkowska-Nowakowska A, Czuczwar S J (2003), 'Is Nitric Oxide Involved in the Anticonvulsant Action of Antiepileptic Drugs?' *Polish Journal of Pharmacology*, 55, 535-42

Yoshikawa T, Tanigawa M, Tanigawa T, Imai A, Hongo H, Kondo M (2000), 'Enhancement of nitric oxide generation by low frequency electromagnetic field', *Pathophysiology*;7(2):131-135

Zwamborn A P M, Vossen S H J A, van Leersum B J A M, Ouwens M A, Mäkel W M (2003), 'TNO study on the effects of GSM and UMTS signals on well-being and cognition', Netherlands Organisation for Applied Scientific Research (TNO)

2005, Andy Davidson BA MA MBA

The author is not a scientist, only a researcher and investigator with special interest in the influences and biology of electromagnetic fields from mobile and wireless technology. As an involved observer, the author has also conducted a local mast survey in Sussex. This paper does not purport to break scientific ground, nor does it establish new findings. Its purpose is to draw together findings that have special relevance in seeking new understanding of observed effects, and redress the bias against investigation of increasingly prevalent phenomena.

Tetrawatch

Tetrawatch is an informal response to national concerns about the implementation of TETRA technology, arising largely in response to the widespread reporting of adverse reactions. The research remit of Tetrawatch extends to its context among similar sources of EMFs.