Electromagnetic field and neurological disorders

Alzheimer’s disease,

why the problem is difficult and how to solve it.

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Introduction
Energy from Electromagnetic fields (EMF) are emitted from computers, power lines, microwave ovens and mobile phones. Also use in magnetic resonance imaging (MRI) and for therapy use. There is reliable evidence that oxidative stress has a role in pathophysiology of neurodegenerative diseases. Several biological effects has been linked to EMF. Pulsed EMF (as in WiFi, DECT and mobile phone) are more bioactive. A mechanism that is altered of EMF from WiFi exposure is the acetylcholinesterase (AChE) activity that in a study was lowered to half in the exposed group. The inhibition of the enzyme AChE has been implicated in neurodegenerative diseases (Obajuluwa et al., 2017). There are different biological effects from EMF that can be related to neurological diseases like Alzheimer’s disease (AD), amyotrophic lateral sclerosis (ALS) and demyelinating disease. The EMFs generate electrical fields and magnetic fields. EMFs biological effects of EMF found that exposure for 50 min mobile phone was associated with increased glucose metabolism and it can also change the structure of living tissues. Neurodegenerative effects can have a mechanism connected to oxidative stress. It decreases the antioxidant defense and lead to more aggressive radicals that can cause cellular damage and can result in long-term DNA-damage. The most common neurodegenerative disease, AD, is characterized by loss of nervous system cells in hippocampus and cortex. A key mechanism of neurodegeneration is oxidative injury (Terzi, Ozberk, Deniz, & Kaplan, 2016).

Implications in Alzheimers disease
AD affects our cognition. You start to forget things and your brain is not fully functional. AD is defined by abnormal protein deposits of amyloid-β (Abeta) and tau (Jack et al., 2018). AD is diagnosed by levels of Abeta and tau and mild cognitive impairment (MCI) is a precursor of AD ("Alzheimer's disease facts and figures," 2018). Non thermal effects of EMF has in 24 studies shown that voltage-gated calcium channel (VGCC) blockers reduced effects of EMF showing that the role of VGCC activation has many different effects from EMF exposure. (M. L. Pall, 2015). Non thermal effects of EMF has been found to affect refolding of a protein in a study (Mancinelli et al., 2004). Defective proteins are also part of AD pathology. Oxidative stress found to have a part of AD pathophysiology is also a mechanism that is found in studies of EMF. VGCC activation produce oxidative stress and excessive neurotransmitter release and other responses like neuropsychiatric changes, sleep disturbance, concentration dysfunction, memory changes (Martin L. Pall, 2016). A study has confirmed an earlier study that microwave radiation enhance aggregation of proteins and promotes amyloid fibril formation. This means microwave electromagnetic fields (MW-EMF) is a risk for misfolding of proteins. It can be a potential risk for amyloid pathologies like prion disease (Mancinelli et al., 2004). Risk and protection factors for AD are many, they are in the areas of biological, genetic, environmental/occupational (Electromagnetic Fields (EMF) (L)), pharmacological and lifestyle. (Krewski et al., 2017).

Another review has shown two studies with increased risk of AD from EMF and 5 studies that failed to detect an association. A meta-analysis of 14 studies showed a risk factor of 2.03. For 5 studies of AD association with Extra low frequency of electromagnetic fields, (ELF-EMF) showed a 62% increased risk of AD. Result show some evidence but are relatively inconsistent (Hersi et al., 2017). Instability of genomics is involved in the origin of AD. Gene amplification, telomere shortening and oxidative stress is shown to play a key role in AD pathology (Maes & Verschaeye, 2012). The Apolipoprotein E (ApoE) supports injury repair and lipid transport in the brain. It binds to amyloid-β peptide. That could initiate toxic events that can lead to synaptic dysfunction and neurodegeneration in AD (Liu, Liu, Kanekiyo, Xu, & Bu, 2013). One of the genes associated with AD risk is, the APOE gene (Dacks, 2018).
People has as a consequence of this an individual risk factor. The genetic risk for incidence of AD is not the same for different individuals. There is also individual differences in different persons effect of EMF, like mobile phone exposure. This is an explanation that studies that show no effects of EMF are not strong evidence for no effect and total effect is going to be underestimated. (Loughran, McKenzie, Jackson, Howard, & Croft, 2012).

Conclusion

Implications for personal early risk protection
A multifactor strategy can be used to minimize AD. You can also use strategies that focus on APOE to minimize risk of AD and mechanisms (Liu et al., 2013). This could be done by gene tests to identify risk groups early and have early interventions to minimize risk factors. Factors to change are in the area of food, exercise and environmental factors like the multidomain intervention FINGER (diet, exercise, cognitive training, and vascular risk management) that showed 25% improvement in neuropsychological tests (Tipton & Graff-Radford, 2018).

Implications for research
To find pathological processes you need to use big data and combine facts from many studies. A model for computer simulations of brain disorders has shown a computer model for pathological processes for AD (Geerts et al., 2016). This challenge for science to find a cure neurologic disease with complex and multi factorial problems and solutions need cooperation between scientists of different subjects. In interviews of scientists worldwide both academic and commercial, they agreed on solutions that try to prevent, detect early, have effective treatment. There is also a need for interactions among scientists with different knowledge working together for solving complex issues like neurological diseases and to integrate available knowledge (Sahakian, 2014). Many other factors are also of importance and need to be considered before the child is born. Alcohol use, physical shock, immune activation, toxins, isolation, DNA, nutrients, antibodies, hormones and care is shown as risk factors for AD in the next generation (Kaplan et al., 2016; Millan, 2013). If you don’t measure the exposure for EMF, this risk factor is going to be neglected in studies of AD. EMF exposure has been connected to oxidative stress from wireless communication. And for AD, oxidative stress is involved in a proposed mechanism for the connection between sleep disturbance, circadian dysfunction and AD, (Figure 1) (Musiek, Xiong, & Holtzman, 2015). Neuropsychiatric symptoms from EMF can also be involved in AD pathology.
Figure 1. Combined models by Musieks proposed mechanisms linking sleep deprivation, circadian dysfunction, and AD. Dotted arrows represent hypothetical links. Pall’s model, Mechanisms of EMF act to stimulate VGCC and produce oxidative stress (Musiek et al., 2015; M. L. Pall, 2015).

Implications for society
Lessons learned from other kind of risks with inconclusive evidence is that if you wait until you have best possible evidence you often delay actions to minimize incidence of disease or risk minimizing that could have been done with less certain evidence (Gee, 2009). EMF regulations of wireless communication and risk assessment need to be reconsidered (Martin L. Pall, 2018; Roda & Perry, 2014; Russell, 2018). In the time until necessary evidence to take precautions to minimize incidence of AD, a multifactorial strategy can be used.
REFERENCES


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