Autism and EMF PT1

Martha R. Herbert*, Cindy Sage
Research Program Neurology, Massachusetts General Hospital, Harvard Medical School, Boston USA

Pathophysiology Abstract

ASCs are defined behaviourally; however, they involve multilevel disturbances in their underlying biology that parallels the physiological impacts of to EMF/RFR exposures dramatically. Many pathophysiological mechanisms are apparent including: oxidative stress, free radical damage, cellular stress proteins, malfunctioning membranes, antioxidant deficiencies such as glutathione, elevated intracellular calcium may be a result of genetics or downstream inflammation from environmental exposures.

Acronyms: Electromagnetic Frequency (EMF), Radiofrequency radiation (RFR), Autism Spectrum Conditions (ASCs), Extremely Low Frequency (ELF)

1 Background

Of course, reviewing the similarities does not imply causality. Also, the physiological processes affected my EMF/RFR are affected by other environmental factors and present in a plethora of chronic illnesses. However, it does not need to be a unique contributor to ASCs to contribute to allostatic load (system overload) and dysfunction. As ASCs, due to their biological sensitivities may be more likely to be affected by EMF/RFR and precautionary measures are recommended. There are no clear unifying mechanisms for genetic syndromes or potential environmental contributors associated with ASCs. Such as the over 800 genes associated with over 100 genetic syndromes that frequently accompany ASCs, and the environmental factors such as toxicants, Vit D deficiency, failure to take prenatal vitamins, stress and air pollution during pregnancy.

Studying RFR may help understand the behavioural features of ASCs, such as a higher level of immune abnormalities correlates with more aberrant behaviours. **To move beyond studying correlations into identifying mechanisms is studying the relationship between systemic pathophysiology and nervous system electrophysiology.** The brain is affected by EMF/RFR because it is a tissue-based organ as well as an information processing system. Researchers have been looking at mechanisms that occur early and cause permanent changes, which would be effective if the main influence is genetic. However, there is evidence emerging that ASCs may be more state-like than trait-like. Children who display 'intermittent autism' could be teetering on the brink of minimally adequate interface of metabolic and electrophysiological dysfunction. The reduction of allergens, infection and pesticides as well as RFR/EMF could help to achieve more 'good days' and fewer 'bad' ones. It is critical to understand that a large number of environmental factors converge on a much smaller amount of sensitive physiological mechanisms.

2 Pathophysiology Parallels

The belief that ASCs stem from a genetic effect leads to the implication that differences occur in the way the brain is formed, and that all 'malfunction' stems from 'malformation'. The question is not about environmental impacts affecting brain development and therefore function is not in dispute. Rather, the influence of the environment on neurodevelopmental conditions. Studies linking ASCs to Down and Rett syndromes have found the cause to be closer to

immune genes than neurodevelopmental genes. Thus it could be stated that genetics increases risk, but the dysfunction is likely at the physiological level.

Oxidative stress is believed to be a common attribute among ASCs. It is a common consequence from exposure to chemical toxicants, nutritional insufficiencies and genetic vulnerabilities. EMF/RFR are also associated with oxidative damage. This is catalysed via the Fenton Reaction (iron converts hydrogen peroxides into hydroxyl free radicals) which then kill organelles and cells by damaging macromolecules. Also note that the effects have been reduced via supplements in rats (Vit AE and C) post 900 MHz exposure.

De Novo mutations are being found increasingly among ASCs and are more likely to be passed by the father than mother to the child. This is likely linked to the EMF/RFR effects of sperm damage. Not only is there an increased risk of DNA damage, there is likely damage to the DNA repair mechanisms.

3 Conclusions

The pathophysiological parallels between ACSs and EMF/RFR was discussed through DNA damage, immune and blood -brain barrier disruption, cellular and oxidative stress, calcium channel, disturbed circadian rhythms, hormone dysregulation and degraded cognition, sleep autonomic regulation and brainwave activity all share commonalities.

The disruption of fertility and reproduction associated with EMF/RFR may be related to the increased incidence of ASCs. All of this argues for the reduction of exposures now and better coordinated research.

EMF and Autism PT 2

Behaviours Abstract

Behaviours from ASCs could emerge from changes to the electrophysiological oscillatory synchronization. EMF/RFR could contribute to these changes by causing 'allostatic overload' leading to increased risk and worsening of biological symptoms. Changes in brain and autonomic nervous system electrophysiological function and sensory processing predominate leading to possible seizures and probable sleep disruption. Contrarily, mitigating exposure may improve symptoms by reducing deterrents of physiological repair. Calcium channels are a probable vulnerable mechanism at risk for both environmental agents and genes associated with autism. The increases in reported ASCs and roll out of new wireless technologies are a coincidence and need to be aggressively investigated. Current evidence is sufficient to warrant new public exposure standards.

4 Background

Mitochondrial dysfunction is common and occurs easily due to their vulnerability and their reliance on their membranes for optimal functionality. As is, electromagnetic radiation can be propagated through the mitochondrial reticulum. This has a higher refractive index and can propagate EMF within the network. When the equilibrium of ROS scavenging and ROS generation in perturbed, the mitochondrial network locked to on remain low-frequency, high amplitude oscillatory mode. This leads to suppression of electrical excitability of Ca++. As well as reduction of mitochondrial cristae, DNA damage, swelling, changing lipids etc. It can also lead to melatonin dysregulation, but this can be treated with supplements. Exposure of cultured cortical neurons to EMF led to increased 8-hydroxyguanine in neuronal mitochondria (a biomarker of DNA oxidative damage).

There is an increased prevalence in ASCs in children born prematurely. Premature infants are exposed to a high ELF-EMF environment. Melatonin treatment significantly prevented the increase of malondyaldehyde and xanthine oxidase in rat brains. Also of note: melatonin levels of neonates has been shown to be reduced in isolettes, and when removed from this environment returned to normal levels. Therefore, a possible treatment for some aspects of pathways could be melatonin. Melatonin has been shown to be reduced in ASCs. Abnormalities in melatonin related genes may cause low levels of melatonin, and treatment with supplements has resulted in significantly improved sleep duration and onset. This has only been shown as related to a small percentage of ASCs.

Melatonin, glutathione and autism have many studies connecting them individually, but none for all three combined. Of pertinence is tryptophan hydroxylase (THP2) - the rate limiting enzyme for serotonin synthesis- is vulnerable to oxidation.

There is a high presence of immune disturbances among ASCs to the point where there is a discussion of classifying ASCs as a neuroimmune disorder. Whether EMF/RFR exposure impacts causally or serves to aggravate symptoms is yet to be determined.EMF/RFR effects on immune response have already been documented and are determined to lead to allergy symptoms. Infection during pregnancy has also shown to have an increased health risk for the foetus at the brain development stages. Various studies have attempted to identify brain relationships with ASCs behavioural features in receptor, neurotransmitter and interneuron abnormalities that could account for the increased excitation/inhibition ratio. Documenting a range of

abnormalities including altered cellular packing in the limbic system, reduced dendritic arborization etc. The presumption that changes occurred prior to birth has shifted with the knowledge that there are ongoing cellular processes extending well into adulthood. Neuroinflammation and oxidative stress play key roles in the development of and symptoms of ASCs. The questions arise: How well will synaptic signals be generated? How well will immune-activated and thereby distracted glial cells be able to modulate synaptic and network activity? We know the cortical innate immune response increases neuronal excitability and can lead to seizures. That inflammation can play a role in epilepsy. We know less about the pathophysiological dysfunction and how they modulate the brain's electrophysiology.

They are using EEGs for possibly determining discriminators for diagnosis of ASCs. Symptomatic level issues with sensory processing are common in ASCs including: hypersensitivity to external stimuli, hyposensitivity to internal sensation and difficulty localizing sensations (including pain). Showing longer and slower latencies of response in auditory stimulus. Relevant because exposure to EMF/RFR and noncoplanar polychlorinated biphenyls (not sure how they are created or natural presence in body) affect the calcium signals. EM fields have a strong effect on cell membranes. Protein strands are on cell membranes and conduct triple action amplifiers as signal detectors, signal amplification and signal transduction to the cell interior.

5 Conclusions

Cell phone exposure has been linked to ADHD-like behaviour in mice offspring due to altered fetal brain development. Exposures have outpaced precautions and there is concern that it will cause health problems. Global exposures have been increased and even as associated health problems are becoming evident there are no global monitoring systems to observe these changes. There is a parallel growth trend between cell phone use and autism birth rates. EMF/RFR exposure during pregnancy may send spurious signals to developing brain cells during pregnancy, which could lead to changes in brain development. There is obvious support for early health interventions in early childhood for the prevention of adult diseases.

The present evidence is sufficient for a call to action and further coordinated research in currently known pathways of effect are required. **When risk factors are largely avoidable and preventable, ignoring clear clear evidence of large-scale health risks to global populations poses unnecessary and unacceptable risks.**