

**Essay 3 - A hypothesis that may explain why certain individuals develop untoward reactions to electrical emissions put out by devices that are low emitters of electrical magnetic fields and why there is an increased prevalence of Amyotrophic Lateral Sclerosis (ALS) among soldiers who participated in the Gulf and Iraq Wars.(1,2)**

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This hypothesis was generated by the fact that the computer that was used by the “key case” discussed in Essays 1 and 2 met all legal requirements regarding electrical emissions. This brought up the question as to whether the key patient was overly sensitive to electrical emissions.

A search of the internet in this regard turned up the fact that 1% of the population of the United States carry a recessive gene for the relatively rare syndrome of Ataxia Telangiectasia.(3,4,5) This gene increases their sensitivity to electrical emissions.(3,4,5) This supported a notion that our key patient *might* be a carrier of this gene or of other abnormal genes that have been found in ALS patients and patients with electrical hypersensitivity.(6)

While the literature search was going on, the fact surfaced that an increase in the prevalence of ALS among veterans of the Gulf and Iraq Wars had taken place. (1,2)

This information prompted the hypothesis that is being presented in this essay. **“It is that ALS is caused by electrical emissions of the type put out by computers and other common devices because there exists a population of individuals who have a genetically induced hypersensitivity to electrical emissions due to the fact that**

**they carry the recessive gene that causes Ataxia-Telangiectasia, or that they have other genes that may be related to increased sensitivity to electrical emissions.**<sup>(6)</sup>

It is a reasonable assumption that modern weaponry of the type to which our armed forces are exposed might pose electrical dangers.

What are the measures that should be taken because of this hypothesis?

First, each soldier that developed ALS should be assigned a “buddy” who had the same amount of exposure to armaments as did he. If no differences are noted, studies to test the soldiers’ resistance to electrical emissions would be in order. The amount of emission put out by the weaponry to which the soldiers were exposed can be quantitated by the SARs method which is the gold standard in this regard.<sup>(9)</sup> Less definitive readings taken by the Trifield Broadband meter which revealed electrical emissions could be used in this regard.

These meters are readily available through Amazon.com.

If both soldiers received that same amount of electrical emissions it would support the hypothesis that the soldiers with ALS had decreased resistance to electrical emissions.

The second study should be that the veterans with ALS should be tested for genes that cause increased sensitivity to electrical emission. Notable amount these genes are the genes both homologous and recessive that are present in Ataxia-Telangiectasia (A-T genes). Other genes to be tested for are those present in some patients with ALS and those that controversially suggested in chemical and electrical sensitivities. The

genetic studies are available through Great Plains Laboratory whose director is Dr. William Shaw (5) and other laboratories in this country.

The next series of experiments that should be done are to test the electrical emissions sensitivity of astrocyte cell cultures obtained from the member of the cohort with ALS. These can be made available through the stem cell methods described by Dr. Bruign before a congressional hearing.(7) Astrocyte cell cultures obtained from animal models of ALS should be used to study electrical sensitivity as well. Methods to do this have been described by Ansari and Hei.(8) Data obtained from the later studies would be helpful in the reconsideration of the acceptable doses of electrical emissions that have been suggested by Carpenter.(11) The endpoint of the above studies would be whether or not a rise in the intercellular level of glutamate occurred in the cells that were cultured.(7,9)

Finally, on another subject, wouldn't it be only fair to treat the veterans with ALS with Ceftriaxone on the empirical basis that it might possibly help them. This suggestion is based on Dr. Rothstein's work which showed that Cephtriaxone would reverse Glutamate and other enzyme changes found in ALS animal models.(9) The government thinks this might be of benefit or they would not have financed a long term multicenter double-blind study of Ceftriaxone in the treatment of ALS.(12) Since the distinct possibility that Ceftriaxone might help ALS don't the veterans who developed ALS deserve immediate treatment with Ceftriaxone based on the possibility that it might help them. This would be done without forcing them to participate in a double-blind study? Haven't they done enough for our country without there being forced to wait for the results of the double-blind study? By the time these studies become conclusive, these

veteran will probably all be dead.

I have shared all the above with a physician in the veterans administration. His answer was that he would refer the information for an opinion to the Institute of Medicine in Washington D.C. At this point, it seems that the suggestions in these musings will only be instituted when and if the effected veterans, their families, their doctors, their elected representatives and the national organizations dedicated to ALS insist on it.

In addition, cohorts of patients who have developed ALS are well known to organizations that have particular interest in this disease. These organizations by questionnaire could identify patients with ALS that have had more than usual exposure to electrical emissions due to their occupation or excessive use of computers or cell phones or possibility to hybrid automobiles.

Patients who are identified by these questionnaires would be excellent candidates for genetic studies regarding genes that confer increased electrical emission sensitivity. As mentioned before, the first genetic study might be to determine if they are carriers of the recessive A-T gene. Additional studies might be helpful in regard to other genes that are claimed to be involved in electrical sensitivity.

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