Int J Neurosci, 2013; Early Online: 1–2

ISSN: 0020-7454 print / ISSN: 1543-5245 online
DOI: 10.3109/00207454.2013.775575

informa
healthcare

Original Article

Electromagnetic hypersensitivity syndrome revisited again

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We described experimental evidence indicating that electromagnetic hypersensitivity (EHS) was a *bona fide* neurological syndrome (that not all self-identified sufferers had a purely psychosomatic disorder) [1]. Our experimental design was tailored to the dynamical nature of EHS (that it is nonlinear, *not* linear) [1]. Rubin et al. [2] and Coggon [3] raised objections and questions. We replied to Rubin [4], but he felt we had ignored a specific question [5]. Our specific answer is addressed here because the issue is critical for proper clinical recognition and management of EHS sufferers.

Rubin asked whether our categorization of the EHS subject's symptoms as "none," "mild," and "more than mild" [1] "was in the original analytic strategy or was it decided *post hoc*?" It was *both*. Why that was so is a key point for the clinical neuroscience community because it explains why Rubin did not discover the neurological syndrome of EHS before us, even though his experiments were performed earlier (see references in [4]). In preliminary observations with the putative EHS sufferer, we observed how the subject responded to the particular field strengths and frequencies we employed as the (controlled) surrogate for typical (uncontrolled) environmental fields. We assumed that any symptoms triggered by the controlled field would be specific to the subject (not a universal reaction similar in nature and intensity to the reactions of all true EHS sufferers). We conducted preliminary studies to identify the symptoms, standardize the language that the subject would use to report the symptoms and identify the intensity of the field that produced only reversible symptoms, which was a critical aspect of our statistical design (independent trials). These steps were taken prior to the data collection we reported [1] but were *post hoc* to the process of identifying the subject's symptoms. This procedure was employed because our goal was not to predict the subject's specific symptoms, but rather to test the hypothesis that they were not explainable on the basis of a purely psychosomatic disorder. Unsurprisingly, experimental designs that force subjects to conform to *a priori* concepts of how *bona fide* EHS sufferers should respond (predefined symptoms, investigator mandated terminology for symptoms, arbitrarily chosen and poorly characterized field intensity) invariably fail (see references in [4]).

A biophysical basis for field transduction has been described [6,7]. The effects produced on brain electrical activity are known [8,9] and effective analytical methods for detecting the process are available [10–12]. These methods may permit objective characterization of the differences in the immediate early processing of information by the brain following transduction [13,14] that occurs in EHS sufferers but not in those without the disorder. Identification of a case-definition/case-selection tool for subjects with EHS [15] could then be used in connection with test results to permit clinical diagnosis of subjects with EHS.

Received 1 February 2013; revised 4 February 2013; accepted 9 February 2013.
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