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Why electrohypersensitivity and related symptoms are caused by non-ionizing man-made electromagnetic fields: An overview and medical assessment

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ABSTRACT

Much of the controversy over the cause of electrohypersensitivity (EHS) lies in the absence of recognized clinical and biological criteria for a widely accepted diagnosis. However, there are presently sufficient data for EHS to be acknowledged as a distinctly well-defined and objectively characterized neurologic pathological disorder. Because we have shown that 1) EHS is frequently associated with multiple chemical sensitivity (MCS) in EHS patients, and 2) that both individualized disorders share a common pathophysiological mechanism for symptom occurrence; it appears that EHS and MCS can be identified as a unique neurologic syndrome, regardless their causal origin. In this overview we distinguish the etiology of EHS itself from the environmental causes that trigger pathophysiological changes and clinical symptoms after EHS has occurred. Contrary to present scientifically unfounded claims, we indubitably refute the hypothesis of a nocebo effect to explain the genesis of EHS and its presentation. We as well refute the erroneous concept that EHS could be reduced to a vague and unproven "functional impairment". To the contrary, we show here there are objective pathophysiological changes and health effects induced by electromagnetic field (EMF) exposure in EHS patients and most of all in healthy subjects, meaning that excessive non-thermal anthropogenic EMFs are strongly noxious for health. In this overview and medical assessment we focus on the effects of extremely low frequencies, wireless communications radiofrequencies and microwaves EMF. We discuss how to better define and characterize EHS. Taken into consideration the WHO proposed causality criteria, we show that EHS is in fact causally associated with increased exposure to man-made EMF, and in some cases to marketed environmental chemicals. We therefore appeal to all governments and international health institutions, particularly the WHO, to urgently consider the growing EHS-associated pandemic plague, and to acknowledge EHS as a mainly new real EMF causally-related pathology.

1. Introduction

We have previously published evidence that a) electrohypersensitivity (EHS) is a distinct newly identified and objectively characterized neurologic pathological disorder which can be clinically diagnosed, and treated using peripheral blood and urine molecular biomarkers and cerebral imaging (Belpomme and Irigaray, 2020); b) EHS and Multiple Chemical Sensitivity (MCS) are possibly associated in EHS patients, both presenting similar clinical presentation and biological and radiological abnormal changes, therefore EHS and MCS could in fact be two etiopathogenic disorders of a unique common pathological syndrome (Belpomme et al., 2015, 2016); c) EHS and MCs are both

Abbreviations: BBB, Blood brain barrier; CNS, Central nervous system; ECG, Electrocardiogram; EEG, electroencephalogram; EHS, Electrohypersensitivity; ELF, Extremely low-frequency; EMF, Electromagnetic field; EMG, Electromyogram; EMR, Electromagnetic Radiation; ESP, Electric skin potential; GSM, Global System for Mobile telecommunication; HRV, Heart rate variability; HSP, heat shock protein; IEI, Idiopathic environmental intolerance; IEI-EMF, Idiopathic environmental intolerance attributed to EMF; MCS, Multiple chemical sensitivity; MF, Magnetic field; MT, Mobile telephony; MW, Microwaves; OS, Oxidative stress; PET, Positron emission tomography; RBC, Red blood cells; RF, Radio frequency; SCBF, Skin capillary blood flow; VDT, Visual display terminal; WC, Wireless Communication; WHO, World Health Organization; WiFi, Wireless fidelity; WLAN, Wireless Local Area Network (for example WIFI).

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associated with detectable low grade inflammation (Belpomme et al., 2015) and oxidative stress (Irigaray et al., 2018a) with possible consequent blood brain barrier (BBB) opening (Belpomme and Irigaray, 2020) as in Alzheimer diseases (Heneka and O'Banion, 2007; Bell and Zlokovic, 2009; Erickson and Banks, 2013) and in other chronic pathological disorders (Patel and Frey, 2015) and d) EHS is associated with brain neurotransmitters abnormal concentrations (Belpomme and Irigaray, 2020) as in laboratory animals exposed to man-made electromagnetic fields (EMF) (Hu et al., 2021).

In a recent scientific international consensus report molecular biomarkers and imaging have been recognized to be of critical value to study EHS by many scientists (Belpomme et al., 2021). In addition, as emphasized in this report, a clear distinction has been made between the causal origin of EHS itself (its etiology) and the daily environmental causes that trigger pathophysiological changes and clinical symptoms in EHS patients after EHS has occurred (its pathogenesis). A pending question is however the role of EMF exposure, both in triggering clinical symptoms and biological changes, and in causing EHS itself. At present, the lack of clear answer to these two questions may explain why most mainstream medical, sanitary and societal bodies still believe that there is not sufficient scientific proof to assert that the clinical symptoms experienced by EHS self-reported patients are really caused by EMF exposure; nor that EHS genesis could be the consequence of excessive man-made EMF exposure. Additionally, since the World Health Organization (WHO) officially stated in 2005 (WHO, 2005) and more recently in 2014 (WHO, 2014), that EHS is a "disabling condition" associated with "non-specific symptoms that lack apparent toxicological or physiological basis or independent verification" and that there are "no clear diagnosis criteria"; it is widely accepted that EHS cannot be diagnosed medically and is not causally related to EMF exposure.

The uncertainty of provocation studies testing the existence of a positive correlative effect of EMF exposure versus sham exposure in EHS patients explain why the cause of symptomatic occurrence is still debated among scientists, some of them refuting the possibility of a causal effect of EMF in triggering symptoms not only in EHS patients (Levallois, 2002; Röösli, 2008; Röösli et al. 2010a, b) but also in healthy people (Baliatsas et al., 2015); some others postulating that EHS is of psychologic origin, i.e. a psychosomatic disease (Rubin et al., 2010, 2011); while still others contrary to the present WHO statements even question the existence of EHS itself (Leszczynski, 2021).

Recalling the historical main scientific research steps and the international institutional statements concerning EHS and MCS, we would like here to summarize how man-made EMF exposure and in some cases marketed environmental chemicals can really trigger symptoms in EHS patients, that exposure to non-thermal man-made EMF are objectively noxious for healthy people and that the etiology of EHS is in fact mainly causally related to man-made EMF exposure in genetically (or epigenetically) susceptible people.

There are indeed three scientific questions to address: a) what is the state of research on EHS pathogenesis b) how can we define hypersensitivity in EHS patients; and c) what is the etiology of EHS in genetically (or epigenetically) susceptible subjects and how it may be generated.

Before answering these questions we would like to emphasize that any causality determination must satisfy the following four WHO causality criteria: a) "the existence of biological effects and health hazards can only be established when research results are replicated in independent laboratories or supported by related studies"; b) "there is agreement with accepted scientific principles"; c) "the underlying mechanism is understood"; d) and finally "a dose-response can be established" (WHO, 2006).

Taking into account these four criteria we disclose and discuss here the present scientific state-of-the-art about the three above distinct scientific questions.

We would like as much as possible to attempt to distinguish the effect of extremely low electromagnetic frequency (ELF) (50–60 Hz), Wireless communication (WC) radiofrequency (RF) (3 kHz to 300 GHz) and WC microwave (MW) EMF (300 MHz–300 GHz); which are presently used for different societal purposes. We would like also to specify that RF/MW electromagnetic radiation (EMR) used as carrier signals (300 kHz-300 GHz) is modulated by ELF EMR (3 Hz- 3000 Hz) in order to transmit increasing amounts of information (Panagopoulos, 2019).

2. Historical scientific and institutional background

The term electromagnetic hypersensitivity which is commonly named electrohypersensitivity (EHS) was first proposed in 1991 by William Rea to identify the pathological condition of patients reporting health effects while being experimentally exposed to RF EMF versus sham and being compared to healthy controls in a controlled environment (Rea et al., 1991). This term was then re-used in 1997 in a report provided by a European group of scientific experts for the European Commission to clinically describe this unusual pathological condition, which posit EMF exposure as symptomatic trigger (Bergqvist and Vogel, 1997). In 2004, because of the seemingly worldwide prevalence increase in EHS, WHO organized an international scientific workshop in Prague to define and characterize EHS. Although not acknowledging EHS as being caused by EMF exposure, due to a lack of available correlation studies, the Prague working group clearly defined EHS as "a phenomenon where individuals experience adverse health effects while using or being in the vicinity of devices emanating electric, magnetic, or electromagnetic fields" (Mild et al., 2006). According to a previous 1996 WHO-sponsored International Program on Chemical Safety (IPCS)-related conference in Berlin on MCS (Report of the Workshop on Multiple Chemical Sensitivities, 1996), it was recommended to qualify such unknown new environmental pathological conditions under the term of "idiopathic environmental intolerance (IEI)". Thus, following the 2004 Prague workshop, instead of using the term EHS, it was recommended to use the term idiopathic environmental intolerance attributed to EMF (IEI-EMF) to name this particular pathological condition, because of the lack of a proven causal link between EHS and EMF exposure, and no known pathophysiological mechanism linking EMF exposure with clinical symptoms. However, because the term EHS was in common use worldwide, WHO officially acknowledged also EHS as an adverse health condition in its 2005 fact sheet N°296 (WHO, 2005); and in its 2014 fact sheet N°193 which further reports on public health and mobile phone use, claiming again a lack of proven causal link between the emission of EMF from mobile phones and health effects, and that there is no proven underlying pathophysiological mechanism accounting for such effects (WHO, 2014). But it was already shown that mobile phones and more generally WC EMFs can cause clinical symptoms (NIEHS, 1998; Chia et al., 2000; Santini et al., 2002, 2003; and others), Oxidative Stress (OS) and DNA damage (Lai and Singh, 1995; Ivancsits et al., 2002, 2003; Diem et al., 2005; Panagopoulos et al., 2007; De Iuliis et al., 2009; Phillips et al., 2009), while the biophysical mechanism of action was also already suspected (Panagopoulos et al., 2002).

Indeed since the 2005 and 2014 WHO official statements; much clinical, biological, and biophysical progress has been made to confirm previous data and to better understand the biophysical and biological processes of the noxious effects of EMFs (Panagopoulos et al., 2015a, 2021; Yakymenko et al., 2016; Lai 2019; 2021) and their pathophysiological significance on human health (Belpomme et al. 2015, 2018; Irigaray et al., 2018a); more particularly to identify and characterize EHS as a new pathological disorder (Belpomme and Irigaray, 2020). Such progress on EMF effects and EHS genesis was summarized in an international consensus meeting held in 2015 at the Royal Belgium Academy of Medicine in Brussels and published in a special issue of the journal *Reviews on Environmental Health* (Carpenter and Belpomme, 2015). Table 1 summarizes the historical scientific steps and WHO statements concerning MCS and EHS acknowledgment.

Table 1
The different historical steps to identify and qualify EHS and MCS, including WHO official statements, statements from WHO-sponsored meetings, and other scientific consensus meetings and reports.

1962	First identification and description of MCS	Randolph (1962)
1991	First identification and description of EHS	Rea et al. (1991)
1996	Berlin WHO-sponsored workshop: MCS classified as idiopathic environmental intolerance (IEI)	Report of the Workshop on Multiple Chemical Sensitivities (1996)
1997	Stockholm possible health implication of EMF exposure: a report prepared by a European group of experts for the European Commission	Bergqvist and Vogel (1997)
1999	Atlanta (US), definition of MCS:1999 consensus meeting	Bartha et al. (1999)
2004	Prague WHO sponsored workshop: identification of idiopathic environmental intolerance attributed to EMF	Mild et al. (2006)
2005	WHO fact sheet n° 292 aiming at defining EHS	WHO (2005)
2014	WHO fact sheet n° 193: EMF and Public Health; mobile phone	WHO (2014)
2015	Brussels: Fourth Paris Appeal Colloquium; a focus on EMF and EHS	Carpenter and Belpomme (2015)
2021	The critical Importance of molecular biomarkers and imaging in the study of EHS. A scientific consensus international report	Belpomme et al. (2021)

3. Symptomatic and biological triggers in EHS patients

Clinical symptoms presumably related to MW exposure were initially reported by Soviet scientists (Dodge, 1969; Carpenter, 2015). They consisted of headaches, fatigue, loss of appetite, insomnia, loss of concentration and short-term memory, transient cardiovascular dysfunction and labile emotional behavior. Some or all of these symptoms were described in particular in people exposed to microwave radar equipment. During the Soviet period, this symptomalogic description was not acknowledged by western scientists. However in a 1972 revised document the US Naval Medical Research Institute was able to count more than 2500 references on the biological and clinical response to radiofrequency radiation (RFR) or microwave radiation published up to April 1972 in the world scientific literature (Glaser, 1972).

In 1979 the clinical symptoms reported to be caused by microwaves were recorded in the framework of a new clinical syndrome named the "microwave syndrome" (Pollack, 1979). This particular clinical syndrome considered to be caused by microwaves in exposed workers was described to involve the nervous system and to be characterized clinically by symptoms such as fatigue, headaches, dysesthesia and various autonomic dysfunctions. This microwave syndrome is symptomatically tantamount to the experimentally identified pathological disorder termed hypersensitivity to EMF (i.e. EHS) by William Rea in 1991 (Rea et al., 1991).

A first approach in describing the adverse health effects possibly associated with exposure to man-made EMFs was made in Sweden in 1984 by Ulf Bergqvist, who reported in a well-documented overview article the clinical symptoms occurring in people using Visual display terminal (VDT) (Bergqvist, 1984). Recorded symptoms included eye problems, ocular disturbance with change in visual performance, musculoskeletal discomfort, facial skin rashes, stress and psychological distress involving particularly mood disturbance, and adverse pregnancy outcomes. Although it was shown that there was an increased number and mobilization of mast cells in the skin of normal volunteers using VDT or television (TV) (Johansson et al., 2001), suggesting that these adverse health effects could be EMF-related; no clear causal relationship could be established between symptom occurrence and VDT- or TV-related EMF exposure. Thus, this observational study could not relate specifically any symptom occurrence to EMF exposure.

Following this VDT study, Ulf Bergqvist and Evi Vogel, with other European scientific experts working for the European Commission conducted a multinational questionnaire-based survey and reported in 1997 that patients who claim to be EHS frequently have "neurasthenia" symptoms, headache and skin symptoms, and less frequently sleep disturbance and anxiety (Bergqvist and Vogel, 1997). However, again, these symptoms were considered non-specific and not causally related to EMF exposure. In fact, this large multinational questionnaire-based survey was unable to clinically define the real symptomatic picture presented by so-called EHS patients and its possible connection with EMF exposure.

However, in 1998, it was reported by the US National Institute of Environmental Health Sciences that health effects could be caused by exposure to powerline frequency (50–60 Hz) electric and magnetic fields (NIEHS, 1998), while in 2000 an increased prevalence of headache among mobile phone users was observed in Singapore (Chia et al., 2000). Then in 2002 Roger Santini in France described the clinical symptoms ascribed to mobile phone use in a French engineering school (Santini et al., 2002), and a year later those ascribed to Mobile Telephony (MT) based station proximity (Santini et al., 2003).

In fact, many studies focused on the symptomatic risk in ELF, RF or MW EMF-exposed people in the general population, but not specifically in EHS self-reported patients. All these general population-based studies were based on telephone survey or mailed or web-based questionnaires. Moreover, most of these studies in the general population investigated one or few self-reported symptoms such as headache (Chia et al., 2000; Milde-Busch et al., 2010; Sudan et al., 2012; Auvinen et al., 2019), tinnitus (Frei et al., 2012; Medeiros and Sanchez, 2016; Auvinen et al., 2019), sleep disturbance (Hutter et al., 2006; Mohler et al., 2012; Monazzam et al., 2014; Huss et al., 2015; Eyvazlou et al., 2016; Tettamanti et al., 2020), cognitive deficiency (Hutter et al., 2006), psychiatric symptoms (Silva et al., 2015) and microwave cataracts (Zaret, 1973). Thus they did not report a detailed description of the complete symptomatic picture of people associated with EMF exposure.

Surprisingly, only few studies have focused specifically on the description of the health symptoms in EHS self-reported patients. Most of these studies were also based on mail or web-based questionnaire and not on face-to-face questioning and examining patients. Such observational investigations concluded that symptoms are subjective, non-specific and not causally related to ELF, RF or MW EMF exposure (Levallois, 2002; Röösli, 2008; Röösli et al., 2010b; Baliatsas et al., 2014). However more recently studies allowing a more precise description of symptoms in such patients were conducted in Finland (Hagström et al., 2013) and in the Netherlands (van Dongen et al., 2014). In both studies, the percentage of women was higher in the EHS group than in the general population, suggesting some genetic susceptibility of these categories of patients, as reported in other studies including our own (Belpomme et al., 2015). In the Dutch study the number of symptoms was higher among people recruited by non-governmental organizations than in the general population (van Dongen et al., 2014), while in the Finnish study it was shown that the number of symptoms during the acute phase of EHS is higher than before its onset (Hagström et al., 2013). Table 2 summarizes all known major original published studies including our own reporting the symptomatic picture in EHS patients.

In fact, as emphasized by several scientists (Carpenter, 2015), the strongest evidence that EHS is a real syndrome similar to the microwave syndrome comes from the initial cases reported from 1980 to 2000 of acute high intensity exposure to MW EMF of healthy people, resulting in "prolonged illness" (Williams and Webb, 1980; Forman et al., 1982; Schilling, 1997, 2000; Reeves, 2000). Moreover, since it was shown that MCS is associated with EHS in near 25% of the EHS cases (Belpomme et al., 2015) and that both disorders are associated with inflammation, OS, possible BBB opening and brain neurotransmitter changes (Belpomme et al., 2015; Irigaray et al., 2018a; Belpomme and Irigaray,

Table 2
Major original published studies describing the symptomatic picture of EHS self-reported patients.

Author	Study types	Source/exposure	Total/evaluable cases
Dodge, 1969 (USA)	Observation study	MW	391 cases vs 100 controls
Rea et al., 1991 (USA)	Provocation test	0.1 Hz–5 MHz EMF exposure	25 patients vs sham and vs 25 healthy controls
Bergqvist and Vogel 1997 (International)	Nationwide questionnaire-based survey	General EMF exposure	72 EHS patients
Hillert et al., 2002 (Sweden)	Population-based questionnaires	EMF, all types	15.000 participants (general population), including 1.5% EHS patients
Navarro et al., 2003 (Spain)	Questionnaire-based survey and EMF power density measurements	WC EMFs	101 persons close to MT base station
Oberfeld et al., 2004 (Spain)	Questionnaire-based survey and EMF measurement	WC EMFs	201 persons close to two GSM 900–1800 cellular phone base stations
Schreier et al., 2006 (Switzerland)	telephone interviews cross-sectional study	50/60 Hz EMF residential/personal exposure	2048 participants, including 5% (107) EHS patients
Schüz et al., 2006 (Germany)	Questionnaire-based survey via internet	EMF, all types including mobile phone use and MT base stations.	192 persons with health complaints, including 107 EHS patients
Röösli et al., 2010a, b (Switzerland)	Population-based questionnaire and weekly measurements	EMF, all types including MT base station proximity, mobile phone and cordless phone use and, W-LAN/WiFi.	1375 participants (general population), including 8% (130) EHS patients
Johansson et al., 2010 (Sweden)	Questionnaire-based survey	EMF, all types including domestic appliance and computer and mobile phone use	45 cases with mobile phone use and 71 EHS patients compared with a 106 population-based sample and 43 controls
Kato and Johansson, 2012 (Japan)	Questionnaire-base survey	EMF, all types including medical device use, mobile phone and cordless use and proximity to MT base stations.	75 EHS patients
Hagström et al., 2013 (Finland)	Questionnaire-based survey via internet	EMF, all types (selection of 50 electrical devices).	194 EHS patients
van Dongen et al., 2014 (The Netherland)	Questionnaire-based survey via internet	EMF, all types	188 people sensitive to EMF versus 937 people non-sensitive to EMF
Nordin et al., 2014 (Sweden)	Questionnaire-based survey	EMF, all types	113 EHS patients versus 48 controls
Baliatsas et al., 2014 (The Netherlands)	Questionnaire-based survey and electronic medical records	EMF, all types including proximity to MT base stations, mobile phone use, domestic appliance and W-LAN/WiFi	5789 respondents including 514 (8.8%) cases with general environmental sensitivity and 202 cases (3.5%) with IEI-EMF (EHS) while the rest of respondents (5073 cases) were used as controls.
Belpomme and Irigaray, 2020 (France)	Face-to-face physical examination	EMF, all types	50 EHS, 50 EHS/MCS and 50 MCS people versus 50 apparently healthy people

2020); it is believed that both EHS and MCS are objective somatic disorders, which cannot be claimed to originate from non-EMF-related psychologic or psychiatric cause, and neither result from a simple undefined and unproven functional impairment (Belpomme and Irigaray 2020, 2021; Belpomme et al., 2021)–although it cannot be excluded these disorders may occur in patients with some particular psychologic traits (Frick et al., 2002).

The purpose of provocation studies is to prove that EHS patients display acute symptoms at the time they are exposed (or after they are exposed) to man-made electric, magnetic and electromagnetic sources; whatever they are, i.e. ELF, RF or MW EMFs. As indicated above, the EHS-associated neurological symptoms are identical to those described in the MW syndrome which was considered at that time as evidently caused by MW EMF in exposed workers. A major difficulty here is that EHS patients are not only associated with hypersensitivity to low intensity anthropogenic EMFs, but due to their possible association with MCS, may also be sensitive to low concentration of multiple chemicals; so both environmental stressors could trigger clinical symptoms and pathological changes in these patients at weak or even very weak environmental EMF intensity or chemical concentration. Furthermore, against all standard medical practice, the clinical symptoms reported by the EHS patients have not been considered as medically assessed and recognized, but simply considered as "self-reported symptoms", meaning they are not "functional symptoms", as it is commonly used in medicine since Hippocrates. Hence they are not accepted as a valuable clinical descriptive tool to identify and diagnose EHS, due to their reported subjectivity and reported non-specificity. Moreover, it was claimed by WHO that EHS-associated symptoms differ from one patient to the other, a claim which is not confirmed by objective clinical observation analysis. In fact, as can be soundly deduced from any face-to-face questioning and physical examination of EHS patients, there is *a priori* no medical reason to dismiss the patients's words, or to believe they make up or mistake each time they attribute their symptoms to EMF exposure (Belpomme and Irigaray, 2020).

Many of the provocation studies performed in EHS patients were of insufficient methodological quality (Rubin et al. 2010, 2011). A major criticism as emphasized in the 2021 consensus report (Belpomme et al., 2021) is that these provocation tests were done before EHS had been objectively diagnosed using biomarkers and imaging techniques. This observation in addition to the flawed method used have resulted in negative findings. We thus consider *a priori* as scientifically unjustified to speculate that the electromagnetic claims of all the patients are unfounded and that their subjective symptomatic feeling could relate to some non-EMF psychosomatic or nocebo health effects (Belpomme et al., 2021; Belpomme and Irigaray, 2021). In Table 3 are depicted some of the unsuitable methodological issues of provocation tests having provided negative results.

An additional important reason for negative results in provocation studies is the fact that in cases of chronic suffering, the patients' response to EMF exposure may be confused without clearly discriminating on/off or off/on field transition, especially when changes occur in a high rate with short-term field durations. In such cases a correct response to short time stimuli should not indeed be reasonably expected.

In fact not all provocation studies have provided negative results. Therefore, the apparently negative results could not preclude an absence of EMF trigger effects. Indeed, in well-designed provocation studies, ELF and/or WC pulsed RF or WC MW EMFs have been shown to trigger clinical and biological health effects in EHS patients. As indicated in Table 4, in such single- or double-blind provocation studies, various clinical and pathophysiological changes have been evidenced in these patients. Clinical effects include heart rate variability (HRV) and/or blood pressure variability (Havas et al., 2010; Havas, 2013; Koppel et al., 2018), altered pupillary light reflex (Rea et al., 1991), reduced visual perception (Trimmel and Schweiger, 1998), and abnormal

Table 3
Some unsuitable methodological issues in provocation tests of previously published studies having provided negative results (Belpomme et al., 2021).

1	Lack of precise inclusion criteria. No objective criteria based on molecular biomarkers and imaging techniques.	Röösli, 2008; Röösli et al., 2010b; Baliatsas et al., 2012; Schmiedchen et al., 2019
2	No clear consideration on medical anamnesis and degree of EHS severity.	Baliatsas et al., 2012; Schmiedchen et al., 2019
3	No consideration of an association with MCS.	Belpomme et al. 2015
4	No consideration that EHS patients are intolerant to specific man-made EMF frequencies.	Röösli, 2008; Röösli et al., 2010b; Baliatsas et al., 2012; Schmiedchen et al., 2019
5	Too short exposure duration.	Baliatsas et al., 2012; Eltiti et al., 2015
6	Symptom recording made too early.	Baliatsas et al., 2012; Schmiedchen et al., 2019
7	Endpoint criteria depending on subjective statements.	Röösli, 2008; Rubin et al., 2010, 2011; Baliatsas et al., 2012; Eltiti et al., 2015; Schmiedchen et al., 2019
8	Possible EHS-associated psychological conditioning due to past suffering.	Dieudonné, 2016
9	Possible significant EMF levels during sham exposure.	Alasdair, 2002

movement during sleep (Mueller and Schierz, 2004), which all have been established by objective clinical evaluation. In addition, pathophysiological effects include altered electroencephalogram (EEG) during sleep (Arnetz et al., 2007; Lustenberger et al., 2013), altered electromyogram (EMG) after wireless local area network (WLAN) exposure (Tuengler and von Klitzing, 2013; von Klitzing, 2021), altered skin capillary blood flow (SCBF) (Tuengler and von Klitzing, 2013; Loos et al., 2013), and electric skin potential (ESP) and conductance changes (Tuengler and von Klitzing, 2013) – these also all allow objective measurements.

Moreover, in a single EHS case double-blind experiment, EMF-related symptomatic intolerance in comparison with sham-exposure has also been reported to be induced by off/on or on/off field transition, rather than by EMF uninterrupted exposure. As the authors state, this means that "the statistically reliable somatic reactions to subliminal EMF exposure were obtained under conditions that reasonably excluded the causative effect of any psychological process" (McCarty et al., 2011).

Such positive effects recorded by provocation tests have also been independently shown in two different earlier EHS case reports (Hocking and Westerman, 2002, 2003) and more recently in two studies showing the objective WC EMF effect on HRV in EHS patients in a double-blind provocation study (Havas et al., 2010) and more generally the effects of RF/MW EMF on the blood, the heart and the autonomic nervous system (Havas, 2013). Provocation studies using similar objective endpoints were also independently provided by the two German biophysicists Andreas Tuengler and Lebrecht von Klitzing, who considered that HRV, SCBF, ESP, and EMG recordings are suitable non-invasive methods to measure EHS in EHS patients (Tuengler and von Klitzing, 2013; von Klitzing, 2021). The same authors propose to combine the continuous measurements of HRV, SCBP and ESP overtime via electrocardiogram (ECG), Doppler meter and electrode matrix recordings respectively; before, during and after EMF versus sham-exposure. This method possibly allow the distinction of EHS patients from individuals suffering from other pathological conditions (Tuengler and von Klitzing, 2013).

As summarized in Table 4, objective abnormalities include the EHS-associated acute and reversible sympathetic and parasympathetic symptoms such as HRV and pupillary light reflex, and other acute neurological symptoms such as attention/memory loss and sleep disturbance, and above all objective biophysical cerebral and transient skin parameter changes, but not all symptoms are acute and reversible. In case of no treatment and no protective measures, chronic symptoms (such as loss of immediate and retrospective memory, mental confusion, insomnia, chronic fatigue, depressive tendency with possible suicidal ideation) may persist for a long time and even become irreversible, leading in some cases, to cerebral atrophy. Such evolution may occur in the case of chronic brain vascular insufficiency caused by persisting high resistance of the brain blood flow and low pulsatility in the cerebral middle arteries (Belpomme and Irigaray, 2020).

In fact, in EHS patients there seems to be a continuum from acute to

Table 4
Provocation tests performed in EHS patients using EMF exposure versus sham-exposure and/or comparison with healthy controls resulting in a positive causal link between EMF exposure and symptoms occurrence and/or pathophysiological changes.

Study	Endpoints	Source	Type of study	EHS patients Evaluable cases	Results (effect of EMF exposure)
Rea et al., (1991) (USA)	Pupillary light reflex	ELF (1–10 kHz)	Double blind EMF v. sham provocation study	25 EHS patients' versus 25 healthy controls	16/25 EHS patients consistently report symptoms in active, but not inactive conditions, compared with 0/25 healthy controls
Trimmel and Schweiger (1998) (Austria)	Attention, perception and memory tests	ELF (50 Hz)	Double blind provocation study	36 EHS versus 30 healthy controls	Reduced performance of visual attention and perception by combining a 50 Hz magnetic field with acoustic noise exposure, compared to the effects of noise only.
Mueller and Schierz (2004) (Switzerland)	Sleep disturbance	ELF (50 Hz)	Double-blind cross-over provocation study	54 EHS cases	Cases moved away from area with maximum 50 Hz field intensity
Arnetz et al., (2007) (USA)	Sleep EEG	RF (884 MHz)	Double blind case-control study compared to sham.	38 IEI-EMF and 31 healthy controls	Exposure caused longer latency to deep sleep from sleep onset and reduced amount of cerebral slow wave
Mc Carty et al. 2011 (USA)	symptomatic responses and EMF field perception	ELF (60 Hz)	Single Blind provocation study, EMF versus sham exposure	A single female EHS case	In the first experiment, the EHS person reported somatic reactions with a significant difference with sham. In the second, she reported significantly more intense symptoms during exposure to a pulsed EMF in comparison with sham. In the third, she was not able to perceive EMF consciously.
Havas et al., 2010 (Canada)	HRV, RBC clumping	RF (2.4 GHz)	Single Blind provocation study EMF versus sham exposure	25 EHS self-reported patient	40% of EHS patients experienced some changes in their HRV during pulsed microwave exposure
Tuengler and von Klitzing, 2013 (Germany)	HRV, capillary blood flow and SEP	RF (Mobile phone)	Single Blind provocation l study	Several types of EHS patients	Modifications of biological parameters caused by EMF exposure
Koppel et al., 2018 (Estonia)	HRV	ELF (50 Hz)	Single Blind provocation study	108 EHS patients	HRV significantly lower during EMF exposure than non-exposure.
Von Klitzing, 2021 (Germany)	ECG and EMG.	RF (WiFi)	Single Blind provocation study	5 EHS patients	Modification of EMG caused by WLAN- exposure.

chronic symptoms, and from biological to health effects/disease; in case of no treatment and/or no efficient protection. We postulate two stages of EMF-related disease progression: first, where EMF-related biological effects may occur with a minimum of clinical symptoms; second, where pathophysiological changes and health symptoms predominate and lead to chronic disease. While the first step may be reversible, the second may be characterized by presumed pathological neurological lesions which may persist and be irreversible (see further).

Taking into account for all available scientific data we believe that present scientific knowledge strongly suggests that man-made EMF exposure can be causally involved in triggering harmful adverse clinical symptoms and noxious pathophysiological changes in EHS patients; and consequently that today's evidence of EMF-related multi-organic somatic effects dismisses the hypothesis of a causal psycho-pathological mechanism to account for the EHS-associated symptom occurrence.

4. Search for electrohypersensitivity characterization

There remains persisting confusion between EHS, which was acknowledged by WHO (WHO, 2005; and IEI-EMF, which was proposed one year before, during the 2004 WHO-sponsored Prague meeting (Mild et al., 2006). EHS as indicated above is presently considered by WHO as a disability condition not proven to be causally related to EMF, and so not specifically subject to medical diagnosis, treatment and prevention; while IEI-EMF is defined as an idiopathic environmental intolerance condition possibly attributed to EMF.

We have proposed to define EHS as the intra-corporal acquisition of a pathological state of hypersensitivity to man-made EMFs in genetically or epigenetically predisposed EHS persons, as is the case for man-made chemicals in MCS patients (Belpomme et al., 2021). By contrast, IEI could be defined as the environmental intolerance to man-made EMFs, chemicals or other stressors, without the necessary acquisition of a state of hypersensitivity. More precisely, we proposed to define EHS clinically and biologically as a decrease in the physiological central nervous system (CNS)-associated EMF tolerance threshold, meaning that intolerance to EMF in EHS patients could occur for weak or even very weak EMF intensities, while intolerance to EMF in non-EHS people could occur for higher EMF intensities (Belpomme and Irigaray, 2021). We thus propose that designation of EHS be restricted to the presumable pathological intra-corporal acquisition of hypersensitivity to EMF, while IEI-EMF will be *stricto sensu* defined as presumable EMF-related environmental intolerance. A similar pathophysiological process involving a decrease in the CNS-associated chemical tolerance threshold could apply to MCS, a consideration that could result similarly in chemical intolerance for weak or even very weak concentrations of multiple environmental chemicals. Note that such a proposed pathophysiological definition, based on a decrease in the environmental tolerance threshold to better define EHS and MCS, is similar to that of toxicant-induced loss of tolerance proposed by Claudia S Miller (1999) who introduced this new concept of environmental sensitivity-related diseases.

While the present medical state-of-the-art must avoid any psychological causal interpretation for EHS occurrence and symptomatic development, there remains a first-order pending question: could the provocation tests prove hypersensitivity to man-made EMFs, i.e. that EHS patients are more sensitive to man-made EMFs than non-EHS healthy subjects; and could these patients detect the presence of ELF or WC RF/MW EMFs better than other persons? Relative to these two important question it was initially believed that using provocation tests in healthy people would show less or no responses under exposure to EMF in comparison with EHS patients (Wagner et al., 2000; Kleinlogel et al., 2008; Valentini et al., 2010; Baliatsas et al., 2015). Similar results would be also expected in case-control studies (Landgrebe et al., 2008) or double blind provocation studies (Lowden et al., 2011); whereas EHS patients depending on the endpoint considered would exhibit typical responses during and/or after EMF-exposure. This is not the case. Contrary to previous supposition of none or fewer effects of man-made EMF exposure in normal healthy individuals; many provocation studies, mostly using ELF and RF non-thermal man-made EMFs in healthy volunteers, have evidenced biological effects; while most studies in EHS patients were negative for the afore-mentioned reasons. The type of EMF/EMR used in provocation studies in healthy people is indicated in Table 5. These effects consist of decreased β-trace protein (prostaglandin D synthase) peripheral blood concentration (this molecule is an endogenous sleep promoting neurohormone) (Hardell et al., 2010), alterations of sleep EEG (Mann and Röschke, 1996; Schmid et al., 2012) and resting EEG (von Klitzing, 1995; Huber et al., 2002; Ghosn et al., 2015; Loughran et al., 2019), alteration of evoked electric potentials (Carrubba and Marino, 2008) and changes of the EEG alpha rhythm (Croft et al., 2008; Vecchio et al., 2012) and of the EEG slow beta, fast beta and gamma bands (Roggeveen et al., 2015). Such exposure to ELF or mostly to RF EMF (see Table 5) have also been shown in healthy subjects to alter the brain response during a memory task (Krause et al., 2000), to affect sleep dependent performance improvement in normal subjects (Lustenberger et al., 2013), to modify the 50 Hz exposure-induced human performance and psychophysiological parameters (Crasson et al., 1999), to induce annoyance and alter well-being (Zheng et al., 2015; Miller et al., 2019), to modify smells (Carlsson et al., 2005), and to influence cognitive performance (Verrender et al., 2016). In addition it has been reported that cell phone-associated WC EMF exposure decreases slow brain potentials at the central and temporo-parieto-occipital brain region (Freude et al., 1998), increases brain glucose metabolism activity (Volkow et al., 2011) and oxygen consumption at the frontal cortex (Curcio et al., 2009), alters non-thermal RFR-induced hemoglobin deoxygenation in cell-free preparations (Mousavy et al., 2009; Muehsam et al., 2013), influences electric properties of human blood measured by impedance spectroscopy (Sosa et al., 2005), increases blood viscosity (Tao and Huang, 2011), modifies brain vascularization (Huber et al., 2002; Aalto et al., 2006), alters blood pressure-associated baro-reflex activity (Braune et al., 1998), and induces vagal nerve stimulation at ECG and EEG (Burgess et al., 2016). In addition it has been shown that cell phone-induced HRV is dependent on breath, i.e. on the inspiration/expiration ratio (Béres et al., 2018). Most of these experimental studies in healthy people are summarized in Table 5, specifying the type of EMF/EMR exposure involved.

The hypothesis that EHS patients are really more sensitive to man-made EMF than healthy people, and that they could detect the presence of EMFs better than healthy people, is challenged by biological studies (Markovà et al., 2005) as well as by epidemiological studies (Röösli, 2008) and provocation studies (Rubin et al., 2011); showing no evidence that short-term exposure to WC EMFs in EHS patients can cause self-reported symptoms, and that these patients could be able to detect ELF, RF or MW EMF better that healthy subjects.

Considering the above reported EMF-induced positive effects in healthy people, it will be extremely difficult to scientifically demonstrate the specific EMF-related hypersensitivity state in EHS patients, i.e. their sensitivity to lower intensity EMFs, using comparative methods. Therefore, research on hypersensitivity to EMFs using such clinical approach in EHS patients may remain an open question for a long time. Although the toxic pathophysiological role of EMF has been ascribed in different animal and human studies, this role has still not been studied specifically for EHS.

5. Search for etiology

The uncertain results of many provocation tests performed in EHS self-reported patients and their misinterpretation have resulted in postulating some nocebo effects; accounting for the great confusion existing presently between researchers within the scientific and medical community and consequently within the international and national medical, sanitary and societal institutions. A big mistake is that the negative results provided by these provocation studies have been

Table 5
Double or single blind provocation studies or observational studies resulting in positive EMF-associated causal link in healthy volunteers.

Authors, Year, Country	Endpoints	Type of study	Evaluable cases	Results (effect of EMF exposure)
von Klitzing L. 1995 (Germany)	Changes in resting EEG	Observational study involving low frequency (217 Hz) exposure	17 healthy students	Alteration in the range of alpha-activity during and after exposure for some hours
Mann and Roschke, 1996 (Germany)	Changes in sleep EEG	Single blind study involving RFR (900 MHz) exposure	24 healthy male volunteers	Temporal pattern of cortisol secretion differs between placebo and night exposure
Braune et al., 1998 (Germany)	Blood pressure (BP), heart rate, capillary perfusion, and subjective well-being	Single-blind placebo-controlled study involving RFR (900 MHz) exposure	7 healthy volunteers	BP associated baro-reflex with activity alteration
Freude et al., 1998 (Germany)	Slow brain potentials (SBP)	Single blind study involving RFR (916.2 MHz) exposure	16 healthy young people	significant decrease of SBP in central and temporo-parieto-occipital brain regions
Crasson et al., 1999 (Belgium)	Changes in event-related potentials (ERP) and EEG/psychophysiological and psychological behavior	Two double blind experimental studies involving 50 Hz exposure and sham	21 healthy male volunteers	Low level 50 Hz MF may have a slight influence on ERP and reaction time under circumstances of sustained attention.
Krause C.M. 2000 (Finland)	Changes in EEG (during a memory task)	Single blind study involving RFR (902 MHz) exposure	16 healthy volunteers	RFR modifies the brain responses
Croft et al., 2002 (Autralia)	effects of active mobile phone (MP) on the neurological system	Single blind cross-over study involving RFR (900 MHz) exposure	24 healthy volunteers	MP exposure affects brain functionning
Huber et al., 2002 (Switzerland)	Effect of EMF on waking regional cerebral blood flow (rCBF) and on waking and sleep EEG in humans.	Double blind study involving two types of RFR (a 'base-station-like' and a 'handset-like' signal) vs. sham control exposure	16 healthy young male right-handed subject	Pulse EMF increases waking rCBF and pulse modulation of EMF is necessary to induce waking and sleep EEG changes
Curcio et al., 2005 (Italia)	Effects of GSM on the neurological system:	RFR (902.4 MHz) exposure	20 healthy volunteers	EMF affects normal brain functioning
Carlsson et al., (2005) (Sweden)	Annoyance related to electrical and chemical factors in a Swedish general population	Cross-sectional study involving different electrical equipment.	13,604 subjects, representative of the population of Scania, Sweden	Connection between environmental annoyance, well-being and functional capacity
Huber al., 2005 (Switzerland)	Effect of EMF on waking regional cerebral blood flow (rCBF)	Double blind study involving two types of RFR (a 'base-station-like' and a 'handset-like' signal) vs. sham control exposure	12 healthy young male subjects	Only 'handset-like' RFR exposure affected rCBF
Aalto et al., 2006 (Finland)	Effects of an active mobile phone on rCBF	Double-blind, counterbalanced study design with subjects performing a computer-controlled verbal working memory task	12 healthy volunteers	EMF emitted by a commercial mobile phone affects rCBF in humans
Croft et al., 2008 (Australia)	Effects of MP on the neurological resting system	Double blind cross-over study. RFR (895 MHz) exposure versus sham.	120 healthy volunteers	Alpha power enhancement during MP exposure
Carrubba and Marino, 2008 (USA)	Evoked brain electrical potentials, EEG normal humans, and patients with epilepsy	Review on different normal human studies	Different normal human studies	Changes in brain activity
Curcio et al., 2009 (Italy)	Oxygenation of the frontal cortex by functional near-IR spectroscopy (fNIRS)	Double blind Case-control study of GSM signal (902.4 MHz) compared to sham.	31 healthy students	Slight influence in frontal cortex
Moussavy et al., 2009 (Iran)	Structure and function of hemoglobin	Experimental study involving RFR (910 MHz and 940 MHz) exposure	Human adult hemoglobin prepared from human RBC of healthy donors.	MP electromagnetic fields decreases oxygen affinity and modifies tertiary structure of hemoglobin depending on field intensity and time of exposure.
Hardell et al., 2010 (Sweden)	Effect of MP and/or cordless phone on β-trace protein blood concentration	Observational study involving RFR (MP and cordless phone)	62 health volunteers	Long term wireless phone use decreases β-trace protein
Carrubba et al., 2010 (USA)	Effects of MP (217 Hz) on the neurological system:	Double blind study	20 healthy volunteers	MP trigger evoked potentials at the frequency of 217 Hz during ordinary MP use.
Lowden et al., 2011 (Sweden)	Sleep EEG	RFR (884 MHz) exposure versus sham double blind study	48 healthy volunteers	RFR exposure increases alpha range in sleep EEG
Volkow et al., 2011 (USA)	Brain glucose metabolism (PET-scan)	Single blind study invovling 50 min cell phone (837.8 MHz) exposure	47 healthy participants	Increased brain glucose metabolism in the region closest to the antenna
Tao and Huang 2011 (USA)	Blood viscosity	Experimental study involving 1.3 T magnetic pulse to a small sample of blood	Human blood from healthy donors	After 1 min of exposure blood viscosity is reduced by 33%
Vecchio et al., 2012 (Italy)	Changes in GSM event-related desynchronisation (ERD) at resting EEG	Placebo controlled double blind study involving RFR (902.4 MHz) exposure	11 healthy volunteers	The peak amplitude of α ERD and the reaction time to go stimuli are modulated by the effect on the cortical activity
Schmid et al., 2012 (Switzerland)	Resting EEG and polysomnography cognitive/behavioral endpoints	Double blind cross-over study invoving RFR (900 MHz) exposure	30 young healthy men	pulse-modulated RFR alter brain functionning
Muehsam et al., 2013 (USA)	Structure and function of hemoglobin	Experimental study involving a pulse-modulated RFR (27.12 MHz) or a static magnetic field exposure	Human adult hemoglobin prepared from human RBC of healthy donors.	Exposure for 10–30 min to either pulse-modulated radiofrequency or static magnetic field increased the rate of deoxygenation of hemoglobin occurring several minutes to several hours after the end of EMF exposure

(*continued on next page*)

Table 5 (*continued*)

Authors, Year, Country	Endpoints	Type of study	Evaluable cases	Results (effect of EMF exposure)
Lustenberger et al., 2013 (Switzerland)	Brain activity during sleep EEG	Double blind cross-over study involving RFR (900 MHz) exposure	16 healthy male people	RFR affect ongoing brain activity during sleep
Ghosn et al., 2015 (France)	Changes in resting EEG effects of GSM on the neurological system	Double blind Case-control study compared with sham invoving RFR (900 MHz) exposure.	26 healthy volunteers	During exposure and post-exposure, the alpha band power is significantly decreased with closed eyes compared to sham.
Roggeveen et al., 2015 (UK)	Changes in resting EEG	Single blind, cross-over study involving RFR (1.9291–1.9397 GHz) exposure	31 young female	All brain waves except delta change significantly due to exposure of the ear, in comparison to sham, with stronger effects with ipsilateral exposure.
Burgess A.P. et al., 2016 (UK)	Resting EEG and ECG (HRV)	Blinded randomized provocation study with a standardized TETRA signal versus sham	164 police officers and 60 volunteers	vagal nerve stimulation at ECG and EEG
Verrender et al., 2016 (Australia)	Visual discrimination task and modified Sternberg working memory task,	Double blind cross-over study involving pulse modulated RFR (PMRF) (920 MHz) exposure	36 healthy volunteers	Cognitive performance is faster relative to sham in a working memory task during PMRF exposure.
Bères et aL 2018 (Hungary)	Heart rate asymmetry (HRA) and HRV parameters using repeated-measures	Double-blind crossover study involving RFR (1800 MHz) exposure	20 healthy volunteers	Increased HRV under 1:1 breathing and RFR exposure
Loughran et al., 2019 (Australia)	Changes in resting EEG	Double blind cross-over study involving RFR (920 MHz) exposure versus sham	36 healthy volunteers	Alpha activity increases during high exposure condition compared to sham

interpreted not to arise from their incorrect methodological practice (Blackman, 2009; Schmiedchen et al., 2019; Belpomme et al., 2021) but rather from some nocebo effect, considering EHS as a psychological disease (Rubin et al. 2010, 2011). Indeed the so called nocebo effect is at best a hypothesis that needs to be confirmed by suitable experimental studies (Belpomme et al., 2021; Belpomme and Irigaray, 2021). This has not occurred. To the contrary, on the basis on a limited number of interviews of EHS patients, it has been suspected that the psycho-societal behavior associated with EHS in these patients is secondary to disease occurrence and suffering, a consequence and not a cause of EHS (Dieudonné, 2016). Moreover, the molecular (Belpomme et al., 2015; Irigaray et al., 2018a; Belpomme and Irigaray, 2020) and radiological abnormalities (Heuser and Heuser, 2017; Irigaray et al., 2018b; Greco, 2020) that have been detected in EHS patients demonstrate that EHS is a neurological somatic disease not a psychological disease. Similarly, MCS has not only been shown to be associated with increased sensitivity to multiple chemicals, but also to be caused by some initial acute or subacute toxic episodes triggered by environmental chemicals – mostly synthetic – in genetically susceptible hosts (Bartha et al., 1999). Therefore neither MCS nor EHS can be considered to be of psychological origin. Also, EHS may be characterized not only as a specific state of intolerance to low intensity EMFs, but also as caused by previous excessive EMF exposure. This critical interpretation was initially provided by David Carpenter by analyzing the microwave syndrome (Carpenter, 2014, 2015). This concept was more recently developed in a review analyzing the EHS underlying mechanisms involving EMF exposure by Y. Stein and I.G. Udasin (2020).

In Table 6 the prevalence expressed in percentages of EHS people relative to the overall population is estimated to range from 0.7% to 13.3%, mainly affecting on average 3%–5% of the population in many different worldwide area or countries, meaning that millions of people may in fact be affected by man-made EMF intolerance, and often by EHS. Similar worldwide figures may account for MCS (Genuis, 2010).

From the analysis of our data and those of the scientific literature, we now consider several strong and convincing arguments that prove EHS is caused by non-thermal anthropogenic EMF exposure.

1. EHS cannot be considered to originate from a nocebo effect i.e. be a psychiatric disease; due to the findings showing its association with somatic abnormalities such as low grade inflammation, OS, and consequent disruption/opening BBB as well as in some cases with anti-myelin Po autoimmune response (Belpomme et al., 2015; Belpomme and Irigaray, 2021). EHS should be therefore considered a somatic disease. In addition we have shown it is associated approximately in 25% of the cases with MCS which is already considered as a somatic disorder (Belpomme and Irigaray, 2021). Moreover EHS is an increasing worldwide plague, hence it is reasonably expected not to be a nocebo disease.
2. EHS occurrence has appeared subsequently to artificial electromagnetic environmental pollution with a seemingly progressive increasing prevalence since the use of WC technologies (Bandara and Carpenter, 2018).
3. As indicated in Table 6 intolerance to EMF exposure including EHS occurrence is not restricted to some regional areas or to countries, but is a worldwide plague with pandemic extension, as is the case for the worldwide expansion of the EMF emitting technologies (Hallberg and Oberfeld, 2006; Bandara and Carpenter, 2018).
4. There are many independent provocation studies proving that ELF/RF/MW EMF can biologically damage the organism and are noxious agents in healthy people (see Table 5); while due to the use of incorrect methodology (see Table 4) in EHS suffering patients, there is a limited number of studies showing pathophysiological changes and symptoms induction. Therefore negative provocation studies definitely cannot exclude a causal role of EMFs in EHS patients.
5. Several main EHS-associated symptoms such as sleep disturbance (Davis, 1997), depressive tendency (Poole et al., 1993; Verkasalo et al., 1997) and suicide risk (Perry et al., 1981; Johnston, 2008) have been shown in independent epidemiological studies to result from dose-dependent EMF exposure, implying that excessive EMF exposure is the cause of these characteristic EHS-associated symptoms (Perry et al., 1981; Poole et al., 1993; Davis, 1997; Verkasalo et al., 1997; Johnston, 2008).
6. As previously reported many EHS patients are characterized by possible low grade inflammation, nitroso-oxidative stress, BBB disruption/opening and brain neurotransmitter changes (Belpomme et al. 2015, 2018; Irigaray et al., 2018a; Belpomme and Irigaray, 2020); all which have been shown in laboratory animals by different independent studies to be caused by man-made EMF exposure (Salford et al. 1994, 2003; Cao et al., 2000; Eberhardt et al., 2008; Nittby et al., 2009; Yang et al., 2012; Aboul Ezz et al., 2013; Megha et al. 2015a, 2015b; Saili et al., 2015; Hu et al., 2021).

Table 6
Estimated prevalence of people with self-reported intolerance to EMF and/or EHS in different countries.

Author, Year, Country	Year of results	Sample Size	People Contribution Rate (%)**	Estimated % of People with EHS
Hillert et al. (2002), Sweden	1997	15,000 (19–80)[a]	73	1.5
Palmquist et al. (2014), Sweden	2010	3406	40	2.7
Schreier et al. (2006), Switzerland	2004	2048 (>14)[a]	55.1	5
Röösli et al., 2010a, Switzerland	2008	1122 (30–60)[a]	37	8.6
Röösli et al., 2010b, Switzerland	2009	1122 (30–60)[a]	37	7.7
Blettner et al. (2009), Germany	2004	30,047	58.6	10.3
Kowall et al. (2012), Germany	2004	30,047	58.4	8.7
Kowall et al. (2012), Germany	2006	30,047	58.4	7.2
Levallois et al. (2002), USA	1998	2072	58.3	3.2
Korpinen and Pääkkönen, 2009, Finland	2002	6121	40.8	0.7
Eltiti et al. (2007), UK	2005	3633	18.2	4
Meg Tseng et al. (2011), Taiwan	2007	1251	11.5	13.3
Schröttner and Leitgeb (2008), Austria	2008	460	88	3.5
Furubayashi et al. (2009), Japan	2007	2472	62.3	1.2
Baliatsas et al. (2014), Netherlands	2011	5789	39.6	3.5
van Dongen et al., 2014, Netherlands	Before 2013	1009	60	7

[a] When provided age of included patients is indicated in brackets. **Contribution rate is the percentage of people having answered positively to the survey.

7. Most EHS patients present in their past medical history excessive exposure to WC RF/MW EMFs, and/or ELF EMFs, confirming that exposure to anthropogenic EMF may be a main plausible causal factor in inducing EHS (Belpomme and Irigaray, 2020).
8. Many independent *in vitro* and *in vivo* studies demonstrate that man-made EMFs can interact with endogenous physiological electric fields which control cellular biological functions in normal organism (Weisenseel, 1983; Nuccitelli, 1988, 2000; Borgens, 1988; Blanchard and Blackman, 1994; Shi and Borgens, 1995; McCaig and Zhao, 1997; McCaig et al., 2005; Yao et al., 2009; Del Giudice et al., 2011; Funk, 2015). When applied to the whole human organism, man-made EMFs distort the physiological endogenous EMFs. They also distort the corresponding cellular functions which results in adverse biological/health effects via EMF/tissue interaction at a molecular level (Blank, 2005; Vander Vorst et al., 2006). This is particularly the case for human brain, heart and muscles all being involved biologically and symptomatology in EHS, a finding confirming the multi-target causing role of man-made EMF-exposure (Frey, 1993; Vander Vorst et al., 2006).
9. It has been shown that man-made EMFs and their corresponding EMR are completely polarized and coherent, and thus differ physically from natural EMF/EMRs which are non-polarized. This key-difference may account for their harmful and toxic effects on biomolecules, cells and tissues, in contrast to natural EMFs, which are necessary for life (Panagopoulos et al., 2015a; Panagopoulos, 2017, 2019, 2021).
10. The pathophysiological mechanism by which polarized and coherent (man-made) EMFs may cause neurotoxic effects is now evidenced. Many *in vitro* and *in vivo* animal (Bas et al., 2009; Sonmez et al., 2010; Yang et al., 2012; Aldad et al., 2012; Deshmukh et al., 2013; Balassa et al., 2013; Furtado-Filho et al., 2015; Megha et al., 2015a; Zhang et al., 2015; Odaci et al., 2016; Sırav and Seyhan, 2016), and human studies (Gandhi et al., 1996; Cardis et al., 2008; Dasdag et al., 2012; Belpomme et al., 2018) evidence the neurological and mainly brain noxious effects of man-made non-thermal or micro-thermal EMFs.
11. At the molecular level it has been shown that non- or micro-thermal low-intensity/long duration EMF exposure act directly on DNA, not only by inducing DNA strand breaks or DNA fragmentation (Lai and Singh, 1995, 2004; Phillips et al., 2009; Panagopoulos, 2019; Lai, 2021), but also by inducing chromosome alteration (Sekeroglu et al., 2012, 2013) and chromatin modification (Belyaev and Kravchenko, 1994; Belyaev, 2005). In addition following genetic damage (Lai, 2021 appendix 1 and 2) and/or epigenetic changes (Blank and Goodman, 1999; Belyaev, 2005: Belyaev et al., 2006; Leone et al., 2014; Dasdag et al., 2015a; Dasdag et al., 2015b), EMF exposure could induce gene regulation changes (Lai, 2021 appendix 3) and protein misfolding (Millenbaugh et al., 2008). In fact, multiple cell targets following external application of EMF – mostly RFR and MW EMF – to the whole organism should be considered in different tissues including the brain. It is still unclear whether these different genetic and/or epigenetic mechanisms are involved in EHS genesis, but as shown in many studies, cell free radicals production following ELF or RF EMF exposure (Lai 2019) may take part in these alterations. We have shown that in 80% of the cases of EHS patients EHS is associated with the production of reactive oxygen species (ROS) and/or reactive nitrogen species (RNS) free radicals, suggesting that EMFs could be indirectly involved in EHS genesis (Irigaray et al., 2018a).

Furthermore, it has been shown that EMFs can interact directly with DNA in a specific magnetic field responsive domain in the HSP70 promoter to induce rapid synthesis of heat-shock proteins, a finding which can account for the anti-inflammatory response reported to occur in healthy people (Lin et al. 1999, 2001; Blank and Goodman, 1999, 2011; Blank, 2005); a result we have also shown to occur in EHS patients (Belpomme et al., 2015).

12. All these different findings clearly argue for a causal role of EMF in inducing EHS directly or indirectly via ROS and/or RNS. Although EMF exposure appears to be the main cause of EHS and can explain the pathophysiological change and the symptomatic occurrence, the specific mechanism of EHS genesis, i.e. the occurrence of a decrease in the EMF intolerance threshold is still hypothetical (see further). In addition, in some EHS cases MCS may precede the occurrence of EHS. Thus we have hypothesized that chemicals may also be implicated as causing agents in EHS genesis in a limited number of cases (11%) (Belpomme and Irigaray, 2020). Additionally in conjunction with the causal role of EMF and/or chemicals there may be some independent risk factors associated with EHS genesis, such as a preexisting depression, a psychiatric comorbidity (Meg Tseng et al., 2011), a previous brain trauma, a possible acquired immunosuppression-associated opportunistic infection, or a congenital malformation; which could further the EMF- and/or chemical-related EHS genesis in genetically and/or epigenetically predisposed individuals. Future research must focus on these different risk

factors with appropriate epidemiological studies and suitable bioclinical methods.

6. Hypothetical biophysical mechanisms specifically involved in EHS genesis

There are some further indications supporting the hypothesis of a particular biophysical mechanism, accounting specifically for a causal role of EMF in inducing hypersensitivity:

(a) due to the presence of electromagnetic receptors, as in bacteria and many animals, humans are all sensitive to EMFs, but normally not hypersensitive. Such receptors have been identified as "cryptochroms" in animal retina (Gegear et al., 2010; Grehl et al., 2016) and as "magnetosomes" in the human brain (particularly in the hippocampus) and in the meninges (Kirschvink et al., 1992a; Dunn et al., 1995; Maher et al., 2016). Magnetosomes are located mainly in areas thought to correspond to the observed EHS-associated pathophysiological abnormalities and clinical symptoms (hippocampus and meninges) in EHS patients. These latter receptors have been shown to contain ferrous magnetite (graigite) and maghemite crystals (Kirschvink et al., 1992a) which have been thought to sense EMFs. Moreover, biogenic magnetite has been shown to be associated with ferromagnetic resonance and to absorb EMFs, hence it can be a mechanism capable of producing some biological response under the influence of EMF (Kirschvink et al., 1992b; Johnsen and Lohmann, 2005). Since these receptors are basically constituted of minerals they are thought to sense not only natural ELF, i.e. the Earth's magnetic field but also man-made polarized static ELF EMF and man-made ELF-associated RFR. Humans may have indeed a geomagnetic sensory neurologic system as do many other animals. But most of them are not consciously aware of the Earth's magnetic field that is encountered in everyday life (Wang et al., 2019). Possibly they have lost this shared magnetic sensory system due to the development of some hypothetical adaptive protection systems. The alteration (or destruction) of this putative anti-EMF adaptive neurologic system by excessive man-made EMF exposure (see further) may explain occurence of hyper-sensitivity to EMF by restoring the remnant primordial sensing effect of magnetosomes. Restoration of other hypothetical EMF sensing receptors might be involved to account for the particular state of EHS.

(b) At a molecular level it has been theorized that the voltage-gated ion channels (VGICs) in cell membranes could be a possible target for polarized and coherent (man-made) EMFs (Bawin and Adey, 1976; Liburdy, 1992; Walleczek, 1992; Balcavage et al., 1996; Panagopoulos et al., 2002, 2015b, 2021). It has been proposed that biogenic magnetite, under the influence of EMF can open such VGICs (Kirschvink et al., 1992b; Johnsen and Lohmann, 2005). But the VGICs physicochemical process which mainly involves calcium ions (Bawin and Adey 1976; Liburdy, 1992; Walleczek, 1992; Pall, 2013), has been thought to be applied to all cells in the organism. Therefore, it cannot explain the unique sensing mechanism/effect of EHS and the particular EHS-associated pathophysiological changes observed in the CNS; specifically in the hippocampus and the meninges. Other EMF-induced mechanisms/effects may be involved;

(c) It has been shown in laboratory animals that EMFs and/or chemicals can particularly damage neurons (Frey, 1993; Redmayne and Johansson, 2014; Megha et al. 2015a, 2015b), and change the neurotransmitter and synapse-related protein concentrations particularly in the hippocampus (Bas et al., 2009; Leone et al., 2014; Teimori et al., 2016; Tan et al., 2019). Moreover, neurons are more vulnerable to EMF-induced apoptosis than other cells in the organism (Salford et al., 2003; Joubert et al., 2008; Sonmez et al., 2010; Zuo et al., 2014; Odaci et al., 2016; Eghlidospour et al., 2017). Since as previously defined EHS appears clinically to be an acquired and persisting state, our hypothesis is that man-made EMFs and/or marketed chemicals in EHS patients may have permanently altered or destroyed neurons of the adaptive protective system, and their neuronal circuits in the brain, possibly in the hippocampus (Belpomme and Irigaray, 2020). This is a path for further biophysical and pathophysiological research efforts in order to better characterize (hyper)sensitivity of EHS and/or MCS, to eventually validate our proposed hypothesis via further specific CNS neurological investigations.

7. Discussion

By using several biomarkers in the peripheral blood and urine, and suitable cerebral imaging techniques (Irigaray et al., 2018b; Belpomme and Irigaray, 2021), we have previously evidenced that EHS is a brain pathological disorder which can be objectively diagnosed and treated. Moreover, it has been shown that, although they differ in their etiology and pathogenesis, both EHS and MCS share a similar clinical and biological signature, so they must be considered medically as parts of a particular unique environmental intolerance-related neurological syndrome (Belpomme et al., 2015). This is what many scientists recently agreed to in a scientific consensus report stipulating the critical role of biomarkers and imaging to study EHS (Belpomme et al., 2021). Our finding on EHS mainly based on the use of biomarkers and suitable imaging techniques must however be confirmed by other studies. But we show here that the present research progress results in the acknowledgment of EHS as a real pathological disorder caused by EMF exposure. Indeed further research efforts should be made to prove definitely the causal role of EMFs in triggering EHS-associated symptoms and EHS genesis itself. However, the different and independent data that we have provided fulfill the causation criteria proposed by WHO (WHO, 2006) because a) they include a dose-response effect of the main EHS-associated symptoms in epidemiological studies, b) they testify that the biological changes of *in vitro* and *in vivo* laboratory animals exposed to man-made EMFs are similar to what is observed in EHS patients, c) they also evidence an EHS-associated non-thermal or micro-thermal pathophysiological mechanism accounting for symptom occurrence, and d) above all they fully obey the general scientific principles used by different independent research teams. The data therefore supports the role of man-made EMFs as a causal agent of EHS. In addition, it is clearly demonstrated in different independent studies using provocation tests, that EMFs are noxious for healthy people. Consequently, there are sufficient established facts to strongly recommend protective measures against the present man-made electromagnetic pollution, using the precautionary principle to protect in particular pregnant women, infants, children, teenagers and young adults in all countries worldwide.

Given the seven billion people worldwide – most using cordless phones and/or mobile phones, Wi-Fi, and other wireless devices – and given the present and future development of 5G (Hardell and Nyberg, 2020; Hardell and Carlberg, 2020; Pall, 2021), it is expected that the prevalence of EMF intolerance and EHS will significantly increase worldwide in the next few years. However, because the figures indicated in Table 6 are estimations based on no objective criteria for identifying EHS (Hallberg and Oberfeld, 2006), we believe these data require confirmation by more objective evaluations. Although the reported EHS prevalence figures are only estimations, it is expected that EMF intolerance and EHS prevalence will continue to grow, in as much as the manufacturers of WC technologies and chemical industries will continue developing their products.

As reported in this overview, since the 2005 and 2014 WHO official publications, much progress has been made in the identification and understanding of EHS (and MCS) as pathological disorders and the bioclinical health effects of man-made EMFs and/or chemicals on the

organism. But EHS and MCS have still not been adequately acknowledged by WHO. The non-thermal or micro-thermal health effects of man-made EMF exposure evidenced in animals as well as in humans and their physico-chemical mechanisms of action (Pall, 2013; Yakymenko et al., 2016; Belpomme and Irigaray, 2020; Panagopoulos et al., 2021) should be considered by WHO. Contrary to the unrealistic claims by the International Commission on Non-Ionizing Radiation Protection (ICNIRP), who still denies the existence of non- or micro-thermal biological and toxic health effects of man-made EMFs (ICNIRP, 1998, 2010, 2020), we emphasize again that critical research progress has recently been made, making non- and micro-thermal EMF effects today a common acknowledgment among scientists and civil society people, as testified by many international scientific appeals calling for a reasonable limitation of electromagnetic pollution and the deletion or a *moratorium* of 5G development (Hardell and Nyberg, 2020; Hardell and Carlberg, 2020; Pall, 2021). Indeed, it appears that the non- or micro-thermal EMF-related toxic health effects (in addition to the MCS-related environmental effects) are the cause of EHS pathogenesis and etiology, as is also a possible cause of cancer (Hardell et al., 1995; IARC, 2002; Belpomme et al., 2007; IARC, 2013; Hardell et al., 2013).

Furthermore, the health care needs of people with environmental sensitivities such as EHS or MCS should be determined and developed in the present socioeconomic environment and medical challenge (Gibson et al., 2015).

Today's level of scientific knowledge engenders a great ethical responsibility of scientists and governments and of national and international health bodies to uncover the adverse health effects of the increasing man-made EMF exposure and warn on the emerging and growing worldwide EHS and MCS global plagues. This means that suitable public health measures must urgently be taken to recognize EHS and MCS as new pathologies and decrease EMF-exposure.

We therefore strongly ask WHO to add EHS and MCS in the future versions of the WHO International Classification of Diseases on the basis on their clinical and pathophysiological identification, just as has already been done for other recognized diseases.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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