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Hvorfor el-overfølsomhet og tilknyttede symptomer er forårsaket av ikke-ioniserende menneskeskapte elektromagnetiske felt: En oversikt og medisinsk vurdering

Dominique Belpomme[a,b,*], Philippe Irigaray[b]

[a] *Avdelingen for medisinsk onkologi, Paris-universitetet, Paris, Frankrike*

[b] *Det europeiske institutt for forskning på kreft og miljø (ECERI), Bryssel, Belgia*

INFORMASJON OM ARTIKKELEN

Nøkkelord:
EHS
El-overfølsomhet
Elektromagnetisk(e) felt (EMF
Ekstremt lavfrekvent(e) felt (ELF)
Mikrobølger
Patofysiologisk mekanisme
Radiofrekvens/Radiofrekvent(e) felt

SAMMENDRAG

Mye av kontroversen om årsaken til el-overfølsomhet (EHS) ligger i mangelen på anerkjente kliniske og biologiske kriterier for en allment akseptert diagnose. Imidlertid er det for tiden tilstrekkelig med data til at EHS kan anerkjennes som en særegen veldefinert nevrologisk patologisk lidelse med objektive kjennetegn. Fordi vi har vist at 1) EHS ofte er forbundet med multippel kjemisk sensitivitet (MCS) hos EHS-pasienter, og 2) at begge disse lidelsene, som kommer ulikt til uttrykk hos de enkelte personer, deler en felles patofysiologisk mekanisme for symptomforekomsten. Det ser ut til at EHS og MCS, uansett hva som er den opprinnelige årsak, kan identifiseres som et unikt nevrologisk syndrom. I denne oversikten skiller vi selve etiologien til EHS fra miljøårsakene som utløser patofysiologiske endringer og kliniske symptomer etter at EHS har oppstått. I motsetning til dagens vitenskapelig ubegrunnede påstander, tilbakeviser vi bestemt hypotesen om at en nocebo-effekt skulle kunne forklare opprinnelsen til EHS og dens manifesteringer. Vi tilbakeviser også den feilaktige forestillingen om at EHS kan reduseres til en vag og uprøvd «funksjonssvikt». Tvert imot viser vi her at det opptrer objektive patofysiologiske endringer og helseeffekter påført av eksponering for elektromagnetisk felt (EMF) hos EHS-pasienter og mest av alt hos friske forsøkspersoner, noe som betyr at for mye ikke-termisk menneskeskapt EMF er sterkt helseskadelig. I denne oversikten og medisinske vurderingen fokuserer vi på virkningene av ekstremt lave frekvenser, trådløse kommunikasjonsradiofrekvenser og mikrobølgede EMF. Vi diskuterer hvordan vi bedre kan definere og karakterisere EHS. I lys av WHOs foreslåtte årsakskriterier, viser vi at EHS faktisk er årsaksmessig knyttet til økt eksponering for menneskeskapt EMF, og i noen tilfeller for miljøkjemikalier som er i handelen. Vi appellerer derfor til alle myndigheter og internasjonale institusjoner innen helse, spesielt WHO, om raskt å vurdere den voksende pandemiske pesten som knytter seg til EHS, og å anerkjenne EHS som en i hovedsak ny reell patologi som er årsaksmessig knyttet til EMF.

1. Innledning

Vi har tidligere publisert bevis på at a) el-overfølsomhet (EHS) er en distinkt, nylig identifisert og objektivt kjennetegnet nevrologisk patologisk lidelse som kan diagnostiseres klinisk og behandles ved å bruke perifere blod- og urinmolekylære biomarkører og hjerneavbildning [f.eks. med MR-skanner] (Belpomme og Irigaray, 2020), b) EHS og multippel kjemisk overfølsomhet (MCS) kan være forbundet med hverandre hos EHS-pasienter, i det de to framtrer klinisk på liknende vis med de samme og biologiske og radiologiske unormale endringer, derfor kan EHS og MCS faktisk være to etiopatogene lidelser av et eget, vanlig patologisk syndrom (Belpomme et al., 2015, 2016), c) EHS og MCS er begge forbundet med påviselig lavgradig inflammasjon (Belpomme et al., 2015) og oksidativt stress (Irigaray et al., 2018a) med mulig påfølgende åpning av blod-hjernebarrieren (BBB) (Belpomme og Irigaray, 2020) slik som ved tilfeller av Alzheimers sykdom (Heneka og

Forkortelser [Forkortelser er fornorsket der de er angitt her med fet skrift.]: BBB: Blod-hjerne-barrieren, CNS: sentralnervesystemet, EKG: elektrokardiogram, EEG: elektroencefalogram, EHS: el-overfølsomhet, ELF: Ekstremt lavfrekvent(e) (felt), EMF: elektromagnetisk(e) felt, EMG: Elektromyogram, **EMS**: elektromagnetisk stråling, ESP: elektrisk hudpotensial, GSM: Globalt System for Mobil telekommunikasjon, HRV: hjertefrekvensvariasjon, HSP: varmesjokkprotein, IEI: idiopatisk miljøintoleranse, IEI-EMF: idiopatisk miljøintoleranse tilskrevet EMF, MCS: Multippel kjemisk følsomhet, MF: magnetfelt, MT: Mobiltelefoni, **MB**: Mikrobølger, OS: oksidativt stress, PET: Positron-emisjonstomografi, RBC: røde blodceller, RF: Radiofrekven(t/s/er), SCBF: blodstrøm i hudens kapillærårer, VDT: Visual display terminal [gammeldags katoderørskjerm], **TK**: trådløs kommunikasjon, WHO: Verdens helseorganisasjon, WiFi: trådløst lokalnett av WiFi-type, WLAN: trådløst lokalnett (for eksempel WiFi).

* For å kontakte forfatterne: ARTAC, 57/59 rue de la Convention, 75015, Paris, Frankrike. E-postadresse: contact.belpomme@gmail.com (D. Belpomme).

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O'Banion, 2007; Bell og Zlokovic, 2009; Erickson og Banks, 2013) og ved andre kroniske patologiske lidelser (Patel og Frey, 2015), og d) EHS er forbundet med unormale konsentrasjoner av nevrotransmittere i hjernen (Belpomme og Irigaray, 2020), slik som hos laboratoriedyr utsatt for menneskeskapte elektromagnetiske felt (EMF) (Hu et al., 2021).

I en nylig publisert vitenskapelig internasjonal konsensusrapport er molekylære biomarkører og avbildningsmetoder blitt anerkjent av mange forskere for å være av avgjørende verdi ved studiet av EHS (Belpomme et al., 2021). I tillegg er det, som understreket i denne rapporten, trukket et klart skille mellom den opprinnelige årsaken til EHS som sådan (dets etiologi), og de daglige årsakene i miljøet som utløser patofysiologiske endringer og kliniske symptomer man finner hos EHS-pasienter etter at EHS har utviklet seg (lidelsens patogenese). Et spørsmål som er åpent, er imidlertid hvilken rolle EMF-eksponering spiller, både for å utløse kliniske symptomer og biologiske endringer, og for å forårsake EHS som sådan. For øyeblikket kan mangelen på et klart svar på disse to spørsmålene forklare hvorfor de fleste vanlige medisinske, helse- og samfunnsorganer fortsatt er av den oppfatning at det ikke foreligger tilstrekkelige vitenskapelig bevis for å hevde at de kliniske symptomene som oppleves av pasienter som selv rapporterer EHS, virkelig er forårsaket av EMF eksponering, og heller ikke at det foreligger tilstrekkelige bevis for at EHS-genese kan være en følge av for mye menneskeskapt EMF-eksponering. Siden Verdens helseorganisasjon (WHO) dessuten offisielt uttalte i 2005 (WHO, 2005), og mer nylig i 2014 (WHO, 2014), at EHS er en «invalidiserende tilstand» forbundet med «ikke-særegne symptomer som mangler et åpenbart toksikologisk eller fysiologisk grunnlag og uavhengig verifisering», og at det fins «ingen klare diagnosekriterier», er det en bred oppfatning at EHS ikke kan diagnostiseres medisinsk og ikke er knyttet årsaksmessig til EMF-eksponering.

Usikkerheten rundt de provokasjonsstudiene som har testet om det fins en positivt korrelert virkning fra EMF-eksponering hos EHS-pasienter sammenliknet med liksom-eksponering, forklarer hvorfor årsaken til at symptomene opptrer, fortsatt diskuteres blant forskere. Noen av studiene tilbakeviser muligheten for at EMF kan være årsaken som utløser symptomer, og da ikke bare hos EHS-pasienter (Levallois, 2002; Röösli, 2008; Röösli et al. 2010a, b), men også hos friske mennesker (Baliatsas et al., 2015). Andre postulerer at EHS er av psykisk opprinnelse, dvs. en psykosomatisk lidelse (Rubin et al., 2010, 2011), mens atter andre til og med stiller spørsmål ved eksistensen av EHS som sådan (Leszczynski, 2021), i strid med de nåværende WHO-uttalelsene.

Ved å hente fram de historisk sett viktigste vitenskapelige trinnene i forskningen og de internasjonale institusjoners uttalelser om EHS og MCS, ønsker vi her å oppsummere hvordan menneskeskapt EMF-eksponering, og i noen tilfeller miljøkjemikalier som er handelsvare, faktisk kan utløse symptomer hos EHS-pasienter, at eksponering for ikke-termisk menneskeskapt EMF er objektivt skadelig for friske mennesker og at etiologien til EHS hovedsakelig faktisk er årsaksmessig knyttet til menneskeskapt EMF-eksponering hos genetisk (eller epigenetisk) mottakelige mennesker.

Det er tre særlig viktige vitenskapelige spørsmål å ta opp: a) Hva er status for forskningen på sykdomsutviklingen som fører til EHS? b) Hvordan kan vi påvise overfølsomhet hos EHS-pasienter?, og c) Hva er etiologien [årsaksforklaringen] bak EHS hos genetisk (eller epigenetisk) mottakelige individer, og hvordan kan EHS skapes? Før vi besvarer disse spørsmålene vil vi understreke at enhver årsaksbestemmelse må tilfredsstille følgende fire WHO-årsakskriterier: a) «At det fins biologiske virkninger og helsefare kan bare fastslås når forskningsresultater er gjentatt i uavhengige laboratorier eller støttet av relevante studier»; b) «[Funnene] er i samsvar med aksepterte vitenskapelige prinsipper.», c) «Den underliggende mekanismen er forstått.», d) og til slutt «Det kan fastsettes en dose-respons[-forbindelse]» (WHO, 2006).

Ved å ta hensyn til disse fire kriteriene klarlegger og diskuterer vi i det følgende den nåværende vitenskapelige status om hvert enkelt av de tre vitenskapelige spørsmålene ovenfor.

Vi ønsker så langt som mulig å forsøke å skille fra hverandre virkningene fra ekstremt lave elektromagnetiske frekvenser (ELF) (50–60 Hz), trådløs kommunikasjon (TK) ved radiofrekvenser (RF) (3 kHz til 300 GHz), og trådløs mikrobølget kommunikasjon (TK MB) EMF (300 MHz–300 GHz). Disse ulike frekvensene brukes i dag til ulike samfunnsformål. Men vi ønsker samtidig å understreke at når RF/MB elektromagnetisk stråling (EMS) brukes som bæresignaler (300 kHz- 300 GHz), moduleres bæresignalet med bruk av ELF EMS (3 Hz- 3000 Hz) for å overføre stadig økende informasjonsmengder (Panagopoulos, 2019).

2. Historisk vitenskapelig og institusjonell bakgrunn

Uttrykket elektromagnetisk overfølsomhet, som vanligvis omtales som el-overfølsomhet (EHS), ble først foreslått i 1991 av William Rea for å identifisere den patologiske tilstanden til pasienter som rapporterte helsevirkninger mens de i eksperimenter i et kontrollert miljø ble eksponert for RF EMF opp mot liksom-eksponering, og ble sammenlignet med sunne personer fra en kontrollgruppe. (Rea et al., 1991). Dette uttrykket ble deretter brukt på ny i 1997 i en rapport som ble lagt fram av en europeisk gruppe vitenskapelige eksperter for EU-kommisjonen for å beskrive klinisk denne uvanlige patologiske tilstanden, og den angir at EMF-eksponering er utløseren av symptomene (Bergqvist og Vogel, 1997). På grunn av den verdensomspennende økningen i forekomster av EHS som nå skjer, arrangerte WHO i 2004, en internasjonal vitenskapelig workshop i Praha for å definere og beskrive EHS. Selv om arbeidsgruppen i Praha på grunn av mangel på tilgjengelige korrelasjonsstudier ikke anerkjente EHS som forårsaket av EMF-eksponering, definerte den uttrykkelig EHS som «et fenomen der individer opplever plagsomme helsevirkninger mens de bruker eller er i nærheten av enheter som sender ut elektriske, magnetiske, eller elektromagnetiske felt» (Mild et al., 2006). I samsvar med en tidligere konferanse i 1996 i Berlin om MCS (Report of the Workshop on Multiple Chemical Sensitivities, 1996) som var knyttet til et WHO-sponset internasjonalt program for kjemikaliesikkerhet (IPCS), ble det anbefalt å betegne slike ukjente nye miljølidelsestilstander med uttrykket «idiopatisk miljøintoleranse (IEI)». Etter Praha-workshopen i 2004 ble det derfor anbefalt å bruke betegnelsen «idiopatisk miljøintoleranse tilskrevet EMF» (IEI-EMF) for å betegne denne spesielle patologiske tilstanden, i stedet for å bruke begrepet EHS, på grunn av mangelen på en bevist årsakssammenheng mellom EHS og EMF-eksponering og mangelen på en kjent patofysiologisk mekanisme som forbinder EMF-eksponering med kliniske symptomer. [«Idiopatisk» vil si at den opptrer spontant uten å ha noen kjent, påvist årsak og uten å ha sammenheng med noen annen kjent tilstand.] Men siden uttrykket EHS var i vanlig bruk verden rundt, anerkjente WHO offisielt også EHS som en ugunstig helsetilstand i sitt 2005-faktaark Nr. 296 (WHO, 2005) og i sitt faktaark Nr.193 fra 2014, som forøvrig rapporterer om folkehelse og mobiltelefonbruk og på ny hevder at det mangler en bevist årsakssammenheng mellom utslippene av EMF fra mobiltelefoner og helsevirkninger, og at det ikke fins noen bevist underliggende patofysiologisk mekanisme som kan redegjøre for slike virkninger (WHO, 2014). Men det var da allerede påvist at mobiltelefoner og mer generelt EMF fra trådløs kommunikasjon kan forårsake kliniske symptomer (NIEHS, 1998; Chia et al., 2000; Santini et al., 2002, 2003; og andre), og føre til oksidativt stress (OS) og DNA-skader (Lai og Singh, 1995; Ivancsits et al., 2002, 2003; Diem et al., 2005; Panagopoulos et al., 2007; De Iuliis et al., 2009; Phillips et al., 2009), og den biofysisk virksomme mekanismen hadde man også allerede mistanker om (Panagopoulos et al., 2002).

Siden WHOs offisielle uttalelser i 2005 og 2014 er det så avgjort skjedd store kliniske, biologiske og biofysiske fremskritt som bekrefter tidligere data og gjør at vi bedre kan forstå de biofysiske

og biologiske prosessene som ligger bak de skadelige virkningene fra elektromagnetiske felt (Panagopoulos et al., 2015a, 2021; Yakymenko et al., 2016; Lai 2019; 2021) og deres patofysiologiske betydning for menneskers helse (Belpomme et al. 2015, 2018; Irigaray et al., 2018a), og spesielt bedre kan identifisere og finne kjennetegn ved EHS som en ny patologisk lidelse (Belpomme og Irigaray, 2020). Fremgangen innen EMF-virkninger og EHS-genese ble oppsummert under et internasjonalt konsensusmøte som ble holdt i 2015 ved Det kongelige belgiske akademi for medisin i Brussel og publisert i en spesialutgave av tidsskriftet *Reviews on Environmental Health* (Carpenter og Belpomme, 2015). Tabell 1 oppsummerer historisk de vitenskapelige trinnene og WHO-uttalelsene som anerkjenner MCS og EHS.

Tabell 1
De forskjellige historiske trinnene for å identifisere og kvalifisere EHS og MCS, herunder WHOs offisielle uttalelser, uttalelser fra WHO-sponsede møter og andre vitenskapelige konsensusmøter og rapporter.

1962	Første identifisering og beskrivelse av MCS	Randolph (1962)
1991	Første identifisering og beskrivelse av EHS	Rea et al. (1991)
1996	WHO-sponset workshop, Berlin: MCS klassifisert som idiopatisk miljøintoleranse (IEI)	Report of the Workshop on Multiple Chemical Sensitivities (1996)
1997	Mulig helseimplikasjon av EMF-eksponering: en rapport utarbeidet av en europeisk ekspertgruppe for Europakommisjonen, Stockholm	Bergqvist og Vogel (1997)
1999	Definisjon av MCS:1999 konsensusmøte, Atlanta (USA)	Bartha et al. (1999)
2004	WHO sponset workshop: identifisering av idiopatisk miljøintoleranse tilskrevet EMF, Praha	Mild et al. (2006)
2005	WHO-faktaark n◦ 292 med sikte på å definere EHS	WHO (2005)
2014	WHO-faktaark n◦ 193: EMF og folkehelse; mobiltelefoner	WHO (2014)
2015	Kollokvium nr. 4 for Paris-appellen; fokus på EMF og EHS, Brussel	Carpenter og Belpomme (2015)
2021	Den kritiske betydningen av molekylære biomarkører og bildebehandling i studiet av EHS. En internasjonal vitenskapelig konsensusrapport	Belpomme et al. (2021)

3. Utløsere av symptomer og biologiske reaksjoner hos EHS-pasienter

Kliniske symptomer som ble antatt å være knyttet til MB-eksponering ble først rapportert av sovjetiske forskere (Dodge, 1969; Carpenter, 2015). De besto av hodepine, tretthet, tap av matlyst, søvnløshet, tap av konsentrasjon og korttidshukommelse, forbigående kardiovaskulær dysfunksjon og labil emosjonell atferd. Noen eller alle disse symptomene ble spesielt beskrevet hos personer som ble utsatt for utstyr med mikrobølget radar. I Sovjet-perioden ble ikke denne symptombaserte beskrivelsen anerkjent av vestlige forskere. I et dokument revidert i 1972 var imidlertid US Naval Medical Research Institute i stand til å telle mer enn 2500 referanser som var publisert frem til april 1972 i verdens vitenskapelige litteratur om biologisk og klinisk respons på radiofrekvent stråling (RFR) og mikrobølget stråling (Glaser, 1972).

I 1979 ble de kliniske symptomene som var rapportert å være forårsaket av mikrobølger, registrert som del av et nytt klinisk syndrom som ble kalt «mikrobølgesyndromet» (Pollack, 1979). Dette spesielle kliniske syndromet som man fant blant arbeidere som ble eksponert for mikrobølger, og som ble ansett for å være forårsaket av disse bølgene, ble beskrevet å involvere nervesystemet og klinisk å være kjennetegnet av symptomer som tretthet, hodepine, dysestesi og ulike slags autonome dysfunksjoner. Det som ble omtalt som mikrobølgesyndromet, er med hensyn til symptomene identisk med den patologiske lidelsen som William Rea påviste eksperimentelt i 1991 og ga benevnelsen overfølsomhet for EMF (dvs. EHS) (Rea et al., 1991).

En første tilnærming i retning av å beskrive de uheldige helsevirkningene som muligvis kunne være forbundet med eksponering for menneskeskapte elektromagnetiske felt, ble gjort i Sverige i 1984 av Ulf Bergqvist, som i en veldokumentert oversiktsartikkel rapporterte om de kliniske symptomene som oppstår hos personer som brukte [datidas] katoderør-skjermer (Visual Display Terminal, VDT) (Bergqvist, 1984). Symptomene som ble registrert, omfattet øyeproblemer, synsforstyrrelser som ga svekket synsevne, muskel- og skjelettplager, utslett i ansiktshuden, stress og psykiske plager som spesielt hadde å gjøre med stemningsskifter, og omfattet også uheldige utfall etter graviditet [fosterskader]. Selv om det ble påvist at bruk av VDT eller TV (TV) førte til økt antall mastceller i huden til normale frivillige (Johansson et al., 2001), og at [allerede eksisterende] mastceller ble mobilisert, hvilket tydet på at disse uheldige helsevirkningene kunne være knyttet til EMF, kunne man ikke slå fast at det fantes noen klar årsakssammenheng mellom det at symptomet forekom, og eksponering for EMF fra [katoderør-baserte] dataterminaler (Video Display Terminal/VDT) eller TV-skjermer. Denne observasjonsstudien kunne derfor ikke kople noe bestemt og enkeltstående symptom til EMF-eksponering.

Etter denne VDT-studien gjennomførte Ulf Bergqvist og Evi Vogel, sammen med andre europeiske vitenskapelige eksperter som jobber for Europakommisjonen, en flernasjonal spørreskjemabasert undersøkelse, og de rapporterte i 1997 at pasienter som hevder å ha EHS, ofte har «nevrasteni»-symptomer, hodepine og hudsymptomer, og – i sjeldnere grad – søvnforstyrrelser og angst (Bergqvist og Vogel, 1997). Men igjen ble disse symptomene ansett som ikke særegne og ikke årsaksmessig forbundet med eksponering for EMF. Så selv ikke denne store multinasjonale spørreskjemabaserte undersøkelsen var i stand til å definere klinisk det symptombildet som de såkalte EHS-pasientene virkelig framviste, og dets mulige sammenheng med EMF-eksponering.

I 1998 ble det imidlertid rapportert av US National Institute of Environmental Health Sciences at helsevirkninger kunne være forårsaket av eksponering for elektriske og magnetiske felt ved frekvensen (50–60 Hz) i strømnettet (NIEHS, 1998), og en økt hyppighet i forekomst [dvs. prevalens] av hodepine blant mobilbrukere ble observert i Singapore i 2000 (Chia et al., 2000). I 2002 beskrev så Roger Santini i Frankrike de kliniske symptomene som ble tilskrevet mobiltelefonbruk ved en fransk ingeniørskole (Santini et al., 2002), og ett år senere de symptomene som ble tilskrevet mobiltelefoni (MT) utfra nærhet til mobilmaster (Santini et al., 2003).

Faktisk har mange studier fokusert på risiko for symptomer fra ELF, RF og MB hos personer i befolkningen generelt som er eksponert for EMF, men de har ikke spesifikt fokusert på pasienter som selv har rapportert om EHS. Alle disse studiene på den generelle befolkningen har vært basert på telefonundersøkelser eller post- eller nettbaserte spørreskjemaer. Dessuten undersøkte de fleste av disse studiene i den generelle befolkningen ett eller få selvrapporterte symptomer, så som hodepine (Chia et al., 2000; Milde-Busch et al., 2010; Sudan et al., 2012; Auvinen et al., 2019), tinnitus (Frei et al., 2012; Medeiros og Sanchez, 2016; Auvinen et al., 2019), søvnforstyrrelser (Hutter et al., 2006; Mohler et al., 2012; Monazzam et al., 2014; Huss et al., 2015; Eyvazlou et al., 2016; Tettamanti et al., 2020), kognitiv svekkelse (Hutter et al., 2006), psykiatriske symptomer (Silva et al., 2015) og mikrobølgestær (Zaret, 1973). Dermed har de ikke kunnet gi noen detaljert beskrivelse av det fullstendige symptombildet knyttet til EMF-eksponering hos enkeltpersoner.

Overraskende nok er det bare få studier som har fokusert spesifikt på å beskrive helsesymptomene hos pasienter som selv har rapportert at de er el-overfølsomme, dvs. har EHS. Dessuten har de fleste av disse studiene vært basert på e-post eller nettbaserte spørreskjema, og ikke på utspørring ansikt-til-ansikt og undersøkelse av pasientene. Denne typen undersøkelser av observerte symptomer har konkludert med at symptomene er subjektive, uspesifikke og ikke årsaksmessig knyttet til eksponering for EMF som er ELF, RF eller MB (Levallois, 2002; Röösli, 2008; Röösli et al., 2010b;

Tabell 2 Større og viktige publiserte studier som beskriver symptombildet ved EHS hos pasienter som selv hevder de har EHS.

Forfatter	Studiens art	Kilde/eksponering	Totalt antall/vurderbare tilfeller
Dodge, 1969 (USA)	Observasjonsstudie	MB	391 tilfeller vs 100 kontroller [dvs. i kontr.gruppen]
Rea et al., 1991 (USA)	Provokasjonstest	Eksponering for EMF 0,1 Hz–5 MHz	25 pasienter vs liksomeksponerte og vs 25 friske kontroller
Bergqvist and Vogel 1997 (Internasjonal)	Landsdekkende spørreskjema-basert kartlegging (survey)	EMF-eksponering generelt	72 EHS-pasienter
Hillert et al., 2002 (Sverige)	Spørreskjema til utvalg av befolkningen	EMF, alle typer	15 000 deltakere (vanlig befolkning), herunder 1,5% EHS-pasienter
Navarro et al., 2003 (Spania)	Spørreskjema-basert kartlegging (survey) og målinger av EMF-intensitet	EMF fra trådløs kommunikasjon	101 personer nær MT-basestasjon
Oberfeld et al., 2004 (Spania)	Spørreskjema-basert kartlegging (survey) og målinger av EMF	EMF fra trådløs kommunikasjon	201 personer nær to basestasjoner for GSM 900–1800-mobiltelefoni
Schreier et al., 2006 (Sveits)	Telefonintervjuer av et tverrsnitt [av befolkningen]	Eksponering for 50/60 Hz EMF i hjemmet/personlig	2 048 deltakere, herunder 5% (107) EHS-pasienter
Schüz et al., 2006 (Tyskland)	Spørreskjema-basert kartlegging (survey)	EMF, alle typer, herunder mobiltelefonbruk og MT-basestasjoner	192 personer med klager over helsa, herunder 107 EHS pasienter
Röösli et al., 2010a, b (Sveits)	Spørreskjema til utvalg av befolkningen og ukentlige målinger	EMF, alle typer, herunder nærhet til MT-basestasjon, bruk av mobil- og snorløs telefon og W-LAN/WiFi	1 375 deltakere (vanlig befolkning), jerunder 8% (130) EH-pasienter
Johansson et al., 2010 (Sverige)	Spørreskjema-basert kartlegging (survey)	EMF, alle typer, herunder bruk av elektrisk utstyr i hjemmet, datamaskin, og mobiltelefon	45 tilfeller med mobilbruk og 71 EHS-pasienter sammenliknet med et utvalg fra vanlig befolkning på 106 og 43 kontroller
Kato and Johansson, 2012 (Japan)	Spørreskjema-basert kartlegging (survey)	EMF, alle typer, herunder bruk av medisinsk utstyr, bruk av mobil og snorløs fasttelefon, og nærhet til MT-basestasjoner.	75 EHS-pasienter
Hagström et al., 2013 (Finland)	Spørreskjema-basert kartlegging (survey) via internett	EMF, alle typer (utvalg av 50 elektriske artikler)	194 EHS-pasienter
van Dongen et al., 2014 (Netherland)	Spørreskjema-basert kartlegging (survey) via internett	EMF, alle typer	188 personer føsomme for EMF mot 937 personer ikke-følsomme for EMF
Nordin et al., 2014 (Sverige)	Questionnaire-based survey	EMF, alle typer	113 EHS-pasienter mot 48 kontroller
Baliatsas et al., 2014 (Netherland)	Spørreskjema-basert kartlegging (survey) og elektronisk medisinsk journal	EMF, alle typer, herunder nærhet til MT-basestasjoner, mobiltelefonbruk, elektrisk utstyr i hjemmet og W-LAN/WiFi	5 789 respondenter herunder 514 (8,8%) tilfeller med allmenn miljøfølsomhet og 202 tilfeller (3,5%) med IEI-EMF (EHS), mens resten av respondentene (5 073 tilfeller) ble brukt som kontroller.
Belpomme and Irigaray, 2020 (Frankrike)	Fysisk undersøkelse ansikt-til-ansikt	EMF, alle typer	50 EHS, 50 EHS/MCS and 50 MCS people versus 50 apparently healthy people

Baliatsas et al. al., 2014). Nyere studier som tillater en mer presis beskrivelse av symptomer hos slike pasienter, ble imidlertid utført i Finland (Hagström et al., 2013) og i Nederland (van Dongen et al., 2014). I begge disse studiene var prosentandelen kvinner høyere i EHS-gruppen enn i den generelle befolkningen, noe som tyder på en viss genetisk følsomhet for disse pasientkategoriene, slik det er rapportert i andre studier, inkludert vår egen (Belpomme et al., 2015). I den nederlandske studien var antallet symptomer høyere blant personer som var rekruttert av frivillige organisasjoner enn i befolkningen generelt (van Dongen et al., 2014), mens det i den finske studien ble vist at antall symptomer i den akutte fasen av EHS er høyere enn før utbruddet (Hagström et al., 2013). Tabell 2 oppsummerer alle kjente store originale, publiserte studier, herunder vår egen rapportering av symptombildet hos EHS-pasienter.

Som flere forskere har understreket (Carpenter, 2015), kommer faktisk det sterkeste beviset på at EHS er et ekte syndrom som ligner på mikrobølgesyndromet, fra de første tilfellene som ble rapportert fra 1980 til 2000 av akutt høyintensiv eksponering for mikrobølget EMF hos friske mennesker, og som resulterte i «langvarig sykdom» (Williams og Webb, 1980; Forman et al., 1982; Schilling, 1997, 2000; Reeves, 2000). Siden det dessuten ble påvist at EHS i nesten 25 % av EHS-tilfellene er forbundet med MCS (Belpomme et al., 2015), og at begge lidelsene er forbundet med inflammasjon, oksidativt stress (OS), mulig åpning av blod-hjerne-barrieren (BBB) og forandringer i hjernens nevrotransmittere (Belpomme et al., 2015; Irigaray et al., 2018a; Belpomme og Irigaray, 2020), antas det at både EHS og MCS er objektive somatiske lidelser som man ikke kan hevde stammer fra årsaker som ikke er knyttet til EMF, men altså skulle være psykiske eller psykiatriske, og heller ikke at de skyldes noen enkel udefinert og ikke påvist funksjonsnedsettelse (Belpomme og Irigaray 2020, 2021; Belpomme et al., 2021) – selv om det ikke kan utelukkes at disse lidelsene kan forekomme hos pasienter med visse særegne psykiske egenskaper (Frick et al., 2002).

Hensikten med provokasjonsstudier er å bevise at EHS-pasienter fremviser akutte symptomer på det tidspunktet de blir eksponert (eller etter at de har blitt eksponert) for menneskeskapte elektriske, magnetiske og elektromagnetiske kilder, uansett hvilke disse er, dvs. om de er ELF, RF eller MB EMF. Som vi har pekt på over, er de EHS-tilknyttede nevrologiske symptomene identiske med de som ble beskrevet for mikrobølgesyndromet blant eksponerte arbeidere, og som den gang ble ansett som åpenbart forårsaket av mikrobølget EMF. En stor vanskelighet her er at EHS-pasienters overfølsomhet ikke bare er forbundet med lavintensitets menneskeskapte EMF, men siden de kan ha en mulig forbindelse til MCS, kan de også være følsomme for lave konsentrasjoner av en rekke kjemikalier, slik at begge miljøstressorene kan utløse kliniske symptomer og patologiske endringer hos disse pasientene ved svake, eller til og med svært svake, intensiteter av EMF i miljøet, eller ditto kjemiske konsentrasjoner. Dessuten har, i strid med all standard medisinsk praksis, de kliniske symptomene som er rapportert for EHS-pasientene, ikke blitt ansett som medisinsk vurderte og anerkjente, men i stedet er de ganske enkelt blitt betraktet som «selvrapporterte symptomer» med den følge at de dermed ikke skulle betraktes «funksjonelle symptomer», slik det har vært vanlig å gjøre i medisinfaget siden Hippokrates. Følgelig er symptomene på grunn av at rapportene er subjektive og at de ikke er spesifikke [dvs. særegne for EHS], ikke blitt akseptert som verdifulle klinisk beskrivende hjelpemidler for å identifisere og diagnostisere EHS. Videre er det blitt hevdet av WHO at EHS-tilknyttede symptomer er forskjellige fra en pasient til en annen, noe som er en påstand som ikke bekreftes ved objektiv analyse av kliniske observasjoner. Som man kan utlede av en hvilket som helst ansikt-til-ansikt-utspørring og fysiske undersøkelse av EHS-pasienter, foreligger det faktisk ingen medisinsk grunn til *a priori* å avvise pasientenes ord, eller til å tro at de finner det på eller tar feil hver gang de knytter EMF-eksponering til symptomene sine (Belpomme og Irigaray, 2020).

Mange av de provokasjonsstudiene som er utført på EHS-pasienter har vært av utilstrekkelig metodologisk kvalitet (Rubin et al. 2010, 2011). En alvorlig kritikk er, slik det er understreket i konsensus-rapporten fra 2021 (Belpomme et al., 2021), at disse provokasjons-testene ble gjort før EHS var objektivt diagnostisert ved bruk av biomarkører og bildeteknikker. Dette poengterte forholdet har, i tillegg til feilene ved den metoden som har vært brukt, resultert i negative funn. Vi anser det derfor *a priori* som vitenskapelig ganske uberettiget og spekulativt å gå ut fra at alle pasientenes påstander om helseplager fra elektromagnetiske felt er ubegrunnede og at deres subjektive opplevelse av symptomer kan knyttes til noen slags psykosomatiske eller nocebo-forårsakede helse-virkninger som ikke er forårsaket av EMF (Belpomme et al., 2021;

Tabell 3 Noen uegnede metodologiske sider ved tidligere publiserte provokasjonstester som har gitt negative resultater (Belpomme et al., 2021).

1. Mangel på presise inklusjons-kriterier. Ingen objektive kriterier basert på molekylære biomarkører og bildeteknikker.	Röösli, 2008; Röösli et al., 2010b; Baliatsas et al., 2012; Schmiedchen et al., 2019
2. Ingen klar vurdering av medisinsk sykehistorie og alvorsgraden av EHS.	Baliatsas et al., 2012; Schmiedchen et al., 2019
3. Ingen vurdering av mulig forbindelse med MCS.	Belpomme et al. 2015
4. Ikke med i vurderingen at EHS-pasienter kan være ømfintlige for spesifikke menneskeskapte EMF frekvenser.	Röösli, 2008; Röösli et al., 2010b; Baliatsas et al., 2012; Schmiedchen et al., 2019
5. For kort eksponeringstid.	Baliatsas et al., 2012; Eltiti et al., 2015
6. Registrering av symptomer foretatt for tidlig.	Baliatsas et al., 2012; Schmiedchen et al., 2019
7. Kriterier for endepunkter valgt utfra subjektive utsagn.	Röösli, 2008; Rubin et al., 2010, 2011; Baliatsas et al., 2012; Eltiti et al., 2015; Schmiedchen et al., 2019
8. Psykisk betinging kan skyldes tidligere lidelser som EHS-pasient.	Dieudonné, 2016
9. Betydelig høye EMF-nivåer kan ha oppstått under liksom-eksponering.	Alasdair, 2002

Belpomme og Irigaray, 2021). I Tabell 3 er gjengitt noen av de uegnede metodologiske trekkene ved provokasjonstester som har gitt negative resultater.

Nok en viktig årsak til negative resultater i provokasjonsstudier er det faktum at i tilfeller av kronisk lidelse kan pasientenes respons på EMF-eksponering bli tilsløret slik at pasienten ikke er i stand til å skille tydelig mellom på- og avslått felt, eller reagere på selve feltendringen mellom på- og avslått. Dette gjelder spesielt når endringene skjer med korte mellomrom og når feltet bare varer kort tid. I slike tilfeller med kortvarige stimuli er det virkelig ikke rimelig å forvente en korrekt respons.

Nå har slett ikke alle provokasjonsstudier gitt negative resultater. Derfor er det forhastet om man utfra de tilsynelatende negative resultatene utelukker at EMF kan utløse virkninger. For ekstra lavfrekvente pulser (ELF) både i trådløs radiofrekvent kommunikasjon (RF TK) og i elektromagnetiske felt fra trådløs mikrobølget kommunikasjon (TK MB EMF) har faktisk vist seg i godt utformede provokasjonsstudier å utløse kliniske og biologiske helsevirkninger hos EHS-pasienter. Som angitt i Tabell 4, er det i slike enkelt- eller dobbeltblindede provokasjonsstudier påvist ulike kliniske og patofysiologiske endringer hos disse pasientene. Kliniske virkninger omfatter uregelmessig hjerterytme (hjerte-frekvensvariabilitet, HRV) og/eller endringer i blodtrykket (Havas et al., 2010; Havas, 2013; Koppel et al., 2018), endret pupillrefleks for lys (Rea et al., 1991), redusert synsoppfattelse (Trimmel og Schweiger, 1998), og unormale bevegelser under søvn (Mueller og Schierz, 2004). Alle disse er fastslått ved objektiv klinisk evaluering. I tillegg omfatter patofysiologiske virkninger endret elektro-encefalogram (EEG) under søvn (Arnetz et al., 2007; Lustenberger et al., 2013), endret elektromyogram (EMG) etter eksponering for trådløst lokalnettverk (WLAN) (Tuengler og von Klitzing, 2013; von Klitzing, 2021), endret kapillær blodstrøm i huden (SCBF) (Tuengler og von Klitzing, 2013; Loos et al., 2013), og endret elektrisk potensial (ESP) og ledningsevne i huden (Tuengler og von Klitzing, 2013) – også dette noe som kan registreres med objektive målinger.

I et dobbeltblindet eksperiment av ett enkelt EHS-tilfelle har for øvrig symptomer på EMF-tilknyttet følsomhet sammenlignet med liksomeksponering blitt rapportert å bli utløst av at feltet slås av og på, eller på selve feltendringen mellom på- og avslått, snarere enn av uavbrutt eksponering for EMF. Som forfatterne slår fast, betyr dette at «de statistisk pålitelige somatiske reaksjonene på EMF-eksponering som pasienten var uvitende om, ble oppnådd under forhold som rimeligvis utelukket noen årsaksmessig følgevirkning av noen som helst psykologisk prosess» (McCarty et al., 2011).

Slike positive virkninger som er registrert ved provokasjonstester er også påvist uavhengig i to ulike tidligere rapporter av EHS-tilfeller (Hocking og Westerman, 2002, 2003) og nylig i to studier som viser den objektivt påviselige virkningen fra EMF fra trådløs kommunikasjon (TK) på HRV hos EHS-pasienter i en dobbelt-blindet provokasjonsstudie (Havas et al., 2010), og mer generelt virkningen av RF/MB EMF på blodet, hjertet og det autonome nervesystemet (Havas, 2013). Uavhengig av disse er det også lagt fram provokasjonsstudier med lignende objektive endepunkter av de to tyske biofysikere Andreas Tuengler og Lebrecht von Klitzing, som vurderte HRV-, SCBF-, ESP- og EMG-registreringer til å være egnede ikke-invaderende metoder for å måle EHS hos EHS-pasienter (Tuengler og von Klitzing, 2013; von Klitzing, 2021). De samme forfatterne foreslår å kombinere kontinuerlige målinger av HRV, SCBP og ESP over tid via henholdsvis elektrokardiogram (EKG), Doppler-måler og elektrode-matrise-registreringer før, under og etter EMF-eksponering og sammenlikne med liksom-eksponering. Denne metoden gjør det kanskje mulig å skille EHS-pasienter fra individer som lider av andre patologiske tilstander (Tuengler og von Klitzing, 2013).

Som oppsummert i Tabell 4, omfatter de objektive unormale trekkene som er knyttet til EHS, akutte og reverserbare sympatiske og parasympatiske symptomer som HRV og pupillær lysrefleks, og andre akutte nevrologiske symptomer som oppmerksomhetstap/hukommelsestap og søvnforstyrrelser, og fremfor alt objektive biofysiske endringer i parametere som er knyttet til hjernen, og forbigående endringer i parametere knyttet til huden, men ikke alle symptomer er akutte eller reversible. Uten behandling og uten beskyttende tiltak kan kroniske symptomer (som tap av korttids- og langtidshukommelse, mental forvirring, søvnløshet, kronisk utmattethet, depressiv tendens, eventuelt med selvmordstanker) vedvare i lang tid og til og med bli irreversible, noe som i noen tilfeller fører til cerebral atrofi. En slik utvikling kan skje i tilfeller av kronisk vaskulær insuffisiens i hjernen, forårsaket av langvarig høy motstand i hjernens blodstrøm og lav pulsatilitet [pulserings-virksomhet] i hjernens mellomarterier [arteria cerebri media/ACM] (Belpomme og Irigaray, 2020).

Det ser i praksis ut til at det i tilfeller av ingen behandling og/eller ingen effektiv beskyttelse skjer en gradvis utvikling hos EHS-pasienter langs et kontinuum fra akutte til kroniske symptomer, og fra biologiske virkninger til helsevirkninger/sykdom. Vi antar to stadier av EMF-tilknyttet sykdomsutvikling: det første, der EMF-tilknyttede biologiske virkninger kan oppstå med minimale kliniske symptomer, og det andre, der patofysiologiske endringer og helse-symptomer dominerer og fører til kronisk lidelse. Mens det første stadiet kan være reversibelt, kan det andre være preget av det som ser ut til å være patologiske nevrologiske lesjoner, og som kan vedvare og være irreversible (se nedenfor).

Vi mener at når vi tar i betraktning alle tilgjengelige vitenskapelige data, peker den nåværende vitenskapelige kunnskapen sterkt i retning av at menneskeskapt EMF-eksponering faktisk kan være årsaksmessig involvert i å utløse plagsomme og uønskelige kliniske symptomer og skadelige patofysiologiske endringer hos EHS-pasienter, og at det følger derav at dagens belegg for EMF-tilknyttede somatiske multiorgan-påvirkninger innebærer at hypotesen om en psykopatologisk årsaksmekanisme som forklaring på forekomsten av symptomene som knyttes til EHS, må forkastes.

4. Jakten på el-overfølsomhetens kjennetegn

Forvirringen som alt har vart lenge, hersker fortsatt om EHS, som ble anerkjent av WHO (WHO, 2005) og IEI-EMF, som ble foreslått som betegnelse ett år tidligere under det WHO-sponsede Praha-møtet i 2004 (Mild et al., 2006). Som omtalt over, anses EHS for tiden av WHO som en funksjonshemmingstilstand som ikke er bevist å være årsaksmessig knyttet til EMF, og derfor som sådan ikke gjenstand for medisinsk diagnose, behandling og forebygging, mens IEI-EMF er definert som en idiopatisk miljøintoleranse-tilstand som muligens kan tilskrives EMF.

Tabell 4
Provokasjonstester utført på EHS-pasienter ved bruk av EMF-eksponering versus liksom-eksponering og/eller sammenligning med friske kontroller, og som resulterte i en positiv årsakssammenheng mellom EMF-eksponering og forekomst av symptomer og/eller patofysiologiske endringer.

Studie	Endepunkter	Kilde	Type studie	EHS-pasienter/Vurderte tilfeller	Resultater (virkning av EMF-eksponering)
Rea et al., (1991) (USA)	Pupillens reaksjon på lys	ELF (1–10 kHz)	Dobbelblindet provokasjonsstudie, EMF versus liksom-eksponering	25 EHS-pasienter versus 25 friske kontrollpersoner	16 av 25 EHS-pasienter rapporterte konsekvent om symptomer under aktive [påslått], men ikke under ikke-aktive betingelser, sammenliknet med 0 av 25 av de friske kontrollpersonene
Trimmel & Schweiger (1998) (Østerrike)	Oppmerksomhets-, persepsjons- og hukommelsestester	ELF (50 Hz)	Dobbelblindet provokasjonsstudie	36 EHS-pasienter versus 30 friske kontrollpersoner	Redusert yteevne mht visuell oppmerksomhet og oppfattelse når eksponering for et 50 Hz magnetfelt ble kombinert med akustisk støy, sammenliknet med virkningen av støy alene.
Mueller and Schierz (2004) (Sveits)	Søvnforstyrrelse	ELF (50 Hz)	Dobbelblindet cross-over provokasjonsstudie	54 EHS-tilfeller	Personene flyttet seg vekk fra området med høyest 50 Hz feltintensitet
Arnetz et al., (2007) (USA)	EEG under søvn	RF (884 MHz)	Dobbelblindet case-control provokasjons-studie sammenliknet med liksom-eksponering	38 IEI-EMF-tilfeller og 31 friske kontrollpersoner	Eksponering forårsaket lengre ledetid fra søvnen inntraff til dyp søvn og redusert mengde langsomme hjernebølger
Mc Carty et al. 2011 (USA)	Respons i form av [fysiske] symptomer og å kunne føle EMF-feltet	ELF (60 Hz)	Enkeltblindet provokasjonsstudie, EMF versus liksom-eksponering	Et enkelt EHS-tilfelle	I det første eksperimentet rapporterte EHS-personen somatiske reaksjoner mot en betydelig forskjell ved liksomekspomnering. I det andre rapporterte hun betydelig mer intense symptomer under eksponering for et pulset EMF i forhold til liksomeksponering. I det tredje var hun seg ikke bevisst om hun kunne merke noe EMF.
Havas et al., 2010 (Canada)	HRV, RBC klumping [i blod, dvs. «pengeruller»]	RF (2,4 GHz)	Enkeltblindet provokasjonsstudie, EMF versus liksom-eksponering	25 selvrapporterte EHS-pasienter	40% av EHS-pasientene opplevde noen endringer i sin HRV under eksponering for pulsede mikrobølger
Tuengler and von Klitzing, 2013 (Tyskland)	HRV, kapillær blodgjen-nomstrømning og SEP	RF (mobiltelefon)	Enkeltblindet provokasjonsstudie	En rekke typer EHS-pasienter	Endringer i biologiske parametere forårsaket av EMF eksponering
Koppel et al., 2018 (Estland)	HRV	ELF (50 Hz)	Enkeltblindet provokasjonsstudie	108 EHS-pasienter	HRV vesentlig lavere under EMF-eksponering enn under ikke-eksponering
Von Klitzing, 2021 (Tyskland)	ECG og EMG	RF (WiFi)	Enkeltblindet provokasjonsstudie	5 EHS-pasienter	Endring av EMG forårsaket av eksponering for WLAN.

Vi har foreslått at EHS defineres som en kroppslig opparbeidet patologisk overfølsomhetstilstand overfor menneskeskapte EMF hos genetisk eller epigenetisk disponerte EHS-personer, slik det er tilfelle med overfølsomhet for menneskeskapte kjemikalier hos MCS-pasienter (Belpomme et al., 2021). I motsetning til dette kan IEI-EMF defineres som en miljøintoleranse overfor menneske-skapte elektromagnetiske felt, kjemikalier eller andre stressfaktorer, uten at det nødvendigvis oppstår noen tilstand av overfølsomhet. Mer presist foreslo vi å definere EHS klinisk og biologisk som en reduksjon i den fysiologiske toleranseterskelen for EMF som er knyttet til sentralnervesystemet (CNS), noe som betyr at det hos EHS-pasienter kan oppstå manglende toleranse for svake, eller til og med svært svake, EMF-intensiteter, mens manglende toleranse for EMF også kan forekomme hos ikke-EHS-personer ved større EMF-intensiteter (Belpomme og Irigaray, 2021). Vi foreslår dermed at betegnelsen EHS begrenses til det som antas å være en patologisk kroppslig opparbeidet overfølsomhet for EMF, mens IEI-EMF defineres *i sin strenge betydning* som miljøintoleranse som antas å være knyttet til EMF. Det kan tenkes å gjelde en lignende patofysiologisk prosess for MCS, som i så fall omfatter at den kjemiske toleranseterskelen reduseres og er knyttet til sentralnervesystemet (CNS), og som på lignende måte kan føre til kjemisk intoleranse for svake eller til og med svært svake konsentrasjoner av en rekke ulike miljøkjemikalier. Legg merke til at en patofysiologisk definisjon som den foreslåtte, som ved å basere seg på en reduksjon i miljøtoleranseterskelen bedre kan definere EHS og MCS, ligner definisjonen for giftstoff-utløst tap av tåleevne som ble foreslått av Claudia S Miller (1999), og som introduserte dette nye konseptet for lidelser som er knyttet til ømfintlighet for miljøfaktorer.

For at nåværende medisinskfaglige kunnskapsstatus skal kunne unngå enhver tolkning som trekker inn psykologi som årsak til forekomster av EHS så vel som symptomutvikling, gjenstår det et førsteordens uavklart spørsmål: Kan provokasjonstestene bevise [at det forekommer] overfølsomhet for menneskeskapte EMF, dvs. at EHS-pasienter er mer følsomme for menneskeskapte EMF enn friske personer uten EHS? Og vil disse pasientene kunne oppdage bedre enn andre personer om det fins ekstra lavfrekvente felt (ELF) eller felt fra trådløs radiofrekvent eller mikrobølget kommunikasjon (TK RF/MB EMF) i omgivelsene? Om disse to viktige spørsmålene antok man opprinnelig at bruk av provokasjonstester på friske mennesker ville vise mindre eller ingen respons under eksponering for EMF sammenliknet med EHS-pasienter (Wagner et al., 2000; Kleinlogel et al., 2008; Valentini et al., 2010; Baliatsas et al., 2015). Liknende resultater kan også forventes i case-kontroll-studier (Landgrebe et al., 2008) eller dobbeltblinde provokasjonsstudier (Lowden et al., 2011). Og man antok at EHS-pasienter ville framvise de typiske responsene i forhold til endepunktet man vurderte, under og/eller etter EMF-eksponering. Men slik er det ikke. Stikk i strid med de antagelsene man tidligere hadde gjort om at normale, friske individer ville framvise ingen eller færre virkninger av menneskeskapt EMF-eksponering, har mange provokasjonsstudier, for det meste ved bruk av ELF og RF ikke-termiske menneskeskapte EMF på friske frivillige, påvist biologiske virkninger. Samtidig var de fleste studier av EHS-pasienter negative av grunnene nevnt over. Hvilke typer EMF/EMS som har vært brukt i provokasjonsstudier av friske mennesker, er angitt i Tabell 5. Virkningene består av redusert konsentrasjon av β-sporprotein (prostaglandin D-syntase), i blodet i kroppens perifere deler (dette molekylet er et endogent søvnfremmende nevrohormon) (Hardell et al., 2010), endringer av søvn-EEG (Mann og Röschke, 1996; Schmid et al., 2012) og hvile-EEG (von Klitzing, 1995; Huber et al., 2002; Ghosn et al., 2015; Loughran et al. al., 2019), endring av elektriske potensialer som bygges opp (Carrubba og Marino, 2008), og endringer på EEG av alfarytmen (Croft et al., 2008; Vecchio et al., 2012) og på EEG av langsomme beta-, raske beta- og gammabånd (Roggeveen et al., 2015). Slik eksponering for ELF, og først og fremst for RF EMF (se Tabell 5), har hos friske personer også vist seg å endre hjerneresponsen under en hukommelsesoppgave (Krause et al., 2000), å påvirke søvnavhengig ytelsesbedring hos normale forsøkspersoner

Table 5
Dobbelt- og enkeltblindede provokasjonsstudier og observasjonsstudier som fant positive EMF-forbundede årsaksforbindelser i friske frivillige.

Forfattere, år, land	Endepunkter	Type studie	Tilfeller vurdert	Resultater (virkning av EMF-eksponering)
von Klitzing L. 1995 (Tyskland)	Endringer i EEG under hvile	Observasjonsstudie med lavfrekvent (217 Hz) eksponering	17 friske studenter	Endringer i omfanget av alfa-aktivitet under og etter eksponering i noen timer
Mann and Roschke, 1996 (Tyskland)	Endringer i EEG under søvn	Enkeltblindet studie med RFR (900 MHz) eksponering	24 friske mannlige frivillige	Tidsmønsteret for kortisol-utskillelse er forskjellig mellom placebo og nattlig eksponering
Braune et al., 1998 (Tyskland)	Blodtrykk (BP), pulsrate, gjennom-strømning i kapillærårer, og subjektiv velværefølelse	Enkeltblindet placebo-kontrollert studie med RFR (900 MHz) eksponering	7 friske frivillige	Endring i aktiviteten til blodtrykk-betinget baro-refleks
Freude et al., 1998 (Tyskland)	Langsomme hjernepotensialer (SBP)	Enkeltblindet studie med RFR (916.2 MHz) eksponering	16 friske unge folk	Vesentlig nedgang av SBP i sentrale og temporale-parietale-occipitale hjerneregionene
Crasson et al., 1999 (Belgium)	Endringer i hendelsestilknyttede potensialer (ERP) og EEG/psyko-fysiologisk og psykisk atferd	To dobbeltblindede eksperimentelle studier med 50 Hz eksponering og liksom-eksponering	21 friske mmannlige frivillige	Svak 50 Hz MF kan ha en svak påvirkning på ERP og på reaksjonstid under betingelser der det kreves vedvarende oppmerksomhet.
Krause C.M. 2000 (Finland)	Endringer i EEG (under en hukommelsesoppgave)	Enkeltblindet studie med RFR (902 MHz) eksponering	16 friske frivillige	RFR endrer hjernens respnser
Croft et al., 2002 (Autralia)	Virkninger av aktiv mobiltelefon (MP) på det nevrologiske system	Enkeltblindet cross-over studie med RFR (900 MHz) eksponering	24 friske frivillige	Eksponering for MP påvirker hjernefunksjoner
Huber et al., 2002 (Sveits)	Virkning på mennesker av EMF på regional cerebral blodstrøm (rCBF) ved våkenhet og på EEG i våken tilstand og under søvn.	Dobbeltblindet studie med to typer RFR (en 'basestation-liknende' og et 'mobiltelefon-liknende' signal) vs. liksom-eksponering av kontrollgruppe	16 friske unge, mannlige høyrehendte forsøkspersoner	Pulset EMF øker rCBF i våken tilstand og pulsmodulering av EMF er nødvendig for å påføre EEG-endringer ved våken tilstand og under søvn.
Curcio et al., 2005 (Italia)	Virkninger av GSM på det nevrologiske system:	RFR (902.4 MHz) eksponering	20 friske frivillige	EMF påvirker normale hjernefunksjoner
Carlsson et al., (2005) (Sverige)	Ubehag knyttet til elektriske og kjemiske faktorer i en vanlig svensk befolkning.	Tverrsnittstudie med ulikt elektrisk utstyr.	13 604 forsøkspersoner, representative for befolkningen i Scania, Sverige	Sammenhenger mellom plager fra miljøet, velvære og funksjonsevne
Huber al., 2005 (Sveits)	Virkninger av EMF på regional cerebral blodstrøm (rCBF)	Dobbeltblindet studie med to typer RFR (en 'basestation-liknende' og et 'mobiltelefon-liknende' signal) vs. liksom-eksponering av kontrollgruppe	12 friske unge, mannlige forsøkspersoner	Bare 'mobiltelefon-liknende' RFR-eksponering påvirket rCBF
Aalto et al., 2006 (Finland)	Virkninger av en aktiv mobiltelefon på rCBF	Dobbeltblindet, motbalansert studieutforming med forsøkspersoner som utførte en datamaskinkontrollert verbal hukommelsesoppgave	12 friske frivillige	EMF som sendes ut fra en vanlig mobiltelefon påvirker rCBF hos mennesker
Croft et al., 2008 (Australia)	Virkninger av MP opå det nevrologiske system under hvile	Dobbeltblindet cross-over-studie. RFR (895 MHz) eksponering versus liksom-eksponering.	120 friske frivillige	Styrking av alfa-energi under MP-eksponering
Carrubba and Marino, 2008 (USA)	Framkalte elketriske potensialer i hjernen, EEG hos normale mennesker, og pasienter med epilepsi	Gjennomgang av ulike studier av normale mennesker	ulike studier av normale personer	Endringer i hjerneaktivitet
Curcio et al., 2009 (Italia)	Oksygennivå i fremre hjernebark ved hjelp av funksjonell nær-IR-spektroskopi (fNIRS)	Dobbeltblindet case-control studie av GSM signal (902.4 MHz) sammenliknet med liksom-eksponerning	31 friske studenter	En viss påvirkning i fremre hjernebark (cortex)
Moussavy et al., 2009 (Iran)	Structure and function of Hemoglobinets struktur og funksjon	Eksperimentell studie med eksponering for RFR (910 MHz og 940 MHz)	Hemoglobin fra voksne personer, preparert fra menneskelig RBC fra friske donorer	Elektromagnetiske felt fra MP svekker oksygen- affinitet og endrer hemoglobinets tertiærstruktur avhengig av feltintensitet og tidspunkt for eksponeringen.
Hardell et al., 2010 (Sverige)	Virkning av MP og/eller snorløs telefon på konsentrasjonen i blodet av β-trace-protein (enzymet lipocalin prostaglandin D2 synthase (L-PGDS))	Observasjonsstudie med RFR (MP og snorløs fasttelefon)	62 friske frivillige	Langtids bruk av trådløs telefon svekker [mengden av] β-trace protein
Carrubba et al., 2010 (USA)	Virkninger av MP (217 Hz) på det nevrologiske system	Dobbeltblindet studie	20 friske frivillige	MP utløser elektriske potensialforskjeller [målt som EP/evoced potentials] ved frekvensen 217 Hz under vanlig MP-bruk
Lowden et al., 2011 (Sverige)	EEG under søvn ved eksponering for RFR (884 MHz) versus liksomeksponering	Dobbeltblindet studie	48 friske frivillige	RFR eksponering øker aøfa-området i EEG under søvn
Volkow et al., 2011 (USA)	Glukosestoffskiftet i hjernen (PET-skanner)	Enkeltblindet studie med 50 min eksponering for mobiltelefon (837,8 MHz)	47 friske deltakere	Økt glukosestoffskifte i hjernen i området nærmest antenna
Tao og Huang 2011 (USA)	Blodets viskositet	Eksperimentell studie med 1,3 T magnetisk puls og et lite antall blodprøver	Menneskeblod fra friske donorer	Etter 1 min eksponering er blodets viskositet redusert med 33%
Vecchio et al., 2012 (Italia)	Endringer i desynkronisering (ERD) på EEG under hvile knyttet til endringer i GSM-signalering	Placebo-kontrollert dobbeltblindet studie med eksponering for RFR (902,4 MHz)	11 friske frivillige	Toppen av amplituden til α-ERD og reaksjonstida ved start-stimuli ble modulert av virkningen av GSM-strålingen på aktiviteten i hjernebarken
Schmid et al., 2012 (Sveits)	EEG under hvile og polysomnografi kognitive/atferdsmessige endepunkter	Dobbeltblindet cross-over-studie med eksponering for RFR (900 MHz)	30 unge friske menn	puls-modulert RFR endrer hjernens funksjonsevnectionning
Muehsam et al., 2013 (USA)	Hemoglobinets struktur og funksjon	Eksperimentell studie med en puls-modulert RFR (27,12 MHz) eller eksponering for et statisk magnetfelt.	Hemoglobin fra voksne personer, preparert fra menneskelig RBC fra friske donorer	Eksponering i 10–30 min for enten puls-modulert radiofrekvent eller statisk magnetisk felt økte deoksyge-neringsraten til hemoglobin, som skjedde flere minutter til flere timer etter endt EMF-eksponering

(*fortsetter neste side*)

(Lustenberger et al., 2013), å forandre menneskelig ytelse og psykofysiologiske parametere ved påvirkning fra 50 Hz-eksponering (Crasson et al., 1999), å utløse irritasjon og endret velvære (Zheng et al., 2015; Miller et al., 2019), å endre lukter (Carlsson et al., 2005), og å påvirke kognitiv ytelse (Verrender et al., 2016).
I tillegg er det rapportert at EMF-eksponering fra trådløs kommunikasjon med mobiltelefon svekker langsomme hjernepotensialer i den sentrale hjerneregionen og i den temporale-parietale-occipitale delen [som dekker isse, tinning og bakhode] (Freude et al., 1998), øker hjernens glukose-stoffskifte-aktivitet (Volkow et al., 2011) og oksygenforbruket ved den fremre hjernebarken (Curcio et al., 2009), at ikke-termisk radiofrekvent stråling (RFR) utløser hemo-

Tabell 5 (*fortsettelse*)

Forfattere, år, land	Endepunkter	Type studie	Tilfeller vurdert	Resultater (virkning av EMF-eksponering)
Lustenberger et al., 2013 (Sveits)	Hjerneaktivitet (EEG) under søvn	Dobbeltblindet cross-over-studie med eksponering for RFR (900 MHz)	16 friske mannlige personer	RFR påvirker pågående hjerneaktivitet under søvn
Ghosn et al., 2015 (Frankrike)	Endringer i EEG under hvile som virkninger av GSM på nervesystemet	Dobbeltblindet case-control-studie med eksponering for RFR (900 MHz) sammenliknet med liksom-eksponering	26 friske frivillige	Med lukkede øyne er styrken på alfa-båndet vesentlig redusert under og etter eksponering sammenliknet med under og etter liksom-eksponering.
Roggeveen et al., 2015 (Storbritannia)	Endringer i EEG under hvile	Enkeltblindet cross-over-studie med eksponering for RFR (1.9291–1.9397 GHz) [3G mobiltelefon]	31 unge kvinner	All hjernebølger unntatt delta endres vesentlig som følge av eksponering mot øret, sammenliknet med ved liksom-eksponering, med sterkeste virkninger på samme side som eksponeringen.
Burgess A.P. et al., 2016 (Storbritannia)	EEG og ECG (HRV) under hvile	Blindet randomisert provokasjonsstudie med et standardisert TETRA-signal versus liksom-eksponering	164 polititjenestemenn og 60 frivillige	Stimulering av vagusnerven sammen med utslag på ECG og EEG
Verrender et al., 2016 (Australia)	Oppgaver knyttet til å se forskjeller og modifisert Sternberg-test av korttidshukommelse	Dobbeltblindet cross-over-studie med eksponering for pulsmodulert RFR (PMRF) (920 MHz)	36 friske frivillige	Ved en oppgave med korttidshukommelse er den kognitive ytelsen raskere under PMRF-eksponering enn under liksom-eksponering
Bères et aL 2018 (Ungarn)	Hjerterytmeasymmetri- (HRA-) and HRV-parametere med gjentatte målinger	Dobbeltblindet cross-over-studie med eksponering for RFR (1800 MHz)	20 friske frivillige	Økt HRV under 1:1 pusting og eksponering for RFR
Loughran et al., 2019 (Australia)	Endringer i EEG under hvile	Dobbeltblindet cross-over-studie med eksponering for RFR (920 MHz) versus liksom-eksponering	36 friske frivillige	Alpha-aktivitet øker under høy eksponering sammenliknet med liksom-eksponering

globin-deoksygenering i cellefrie preparater (Mousavy et al., 2009; Muehsam et al., 2013), påvirker elektriske egenskaper i menneske-blod målt med impedansspektroskopi (Sosa et al., 2005), øker blodviskositeten (Tao og Huang, 2011), modifiserer hjernevaskula-risering (Huber et al., 2002; Aalto et al., 2006), endrer virksom-heten til baro-refleksen som er knyttet til regulering av blodtrykket (Braune et al., 1998), og utløser stimulering av vagusnerven målt med EKG og EEG (Burgess et al., 2016). I tillegg har det vist seg at mobiltelefon-utløst HRV er avhengig av pusten, dvs. av innpust-/utpust-forholdet (Béres et al., 2018). De fleste av disse eksperi-mentelle studiene på friske mennesker er oppsummert i Tabell 5, som spesifiserer hvilken type EMF/EMS-eksponering som ble brukt.

Hypotesen om at EHS-pasienter faktisk er mer følsomme for menneskeskapt EMF enn friske mennesker, og at de kan oppdage om det er EMF i omgibelsene bedre enn friske mennesker, utfordres av biologiske studier (Markovà et al., 2005), samt av epidemiologiske studier (Röösli, 2008) og av provokasjonsstudier (Rubin et al., 2011). Disse studiene framviser ingen bevis for at kortvarig eksponering for EMF fra trådløs kommunikasjon (TK EMF) hos EHS-pasienter vil forårsake symptomene de selv rapporter, og heller ikke at disse pasientene vil være i stand til å oppdage ekstra lave frekvenser (ELF), radiofrekvenser (RF) eller mikrobølgede elektromagnetiske (MB EMF) bedre enn friske forsøkspersoner.

Tatt i betraktning de positive funnene angitt ovenfor av virkninger forårsaket av EMF hos friske mennesker, vil det være ekstremt vanskelig å påvise vitenskapelig ved bruk av sammenlikningsmetoder at EHS-pasienter kan ha noen særegen overfølsomhet knyttet til EMF, altså at deres følsomhet for EMF slår ut ved lavere intensitet. Dersom man bruker en slik klinisk tilnærming, kan derfor forskningen på overfølsomhet for EMF hos EHS-pasienter bli stående med uavklarte spørsmål i lang tid. Selv om man i ulike dyre- og menneskestudier har tillagt EMF rollen som det toksiske, patofysiologisk elementet, er denne rollen fortsatt ikke blitt studert spesifikt i forbindelse med EHS.

5. Jakten på den opprinnelige årsaken – etiologien

De usikre resultatene i mange provokasjonstester som har vært utført på pasienter som selv rapporterer at de er EHS, og de feil-aktige tolkningene i dem har ført til at man har postulert [dvs. forutsatt uten begrunnelse eller bevis] at årsakene må ligge i en eller annen slags nocebo-virkninger. Dette er grunnen til den store forvirringen som for tiden hersker mellom forskere innen det viten-skapelige og medisinske miljøet og følgelig innenfor de internasjo-nale og nasjonale medisinske, folkehelse- og samfunnsmessige institusjonene. Det er et stort feilgrep at de manglende funnene som er kommet ut av disse provokasjonsstudiene, ikke er blitt tolket som å skyldes feil ved deres arbeidsmetode (Blackman, 2009; Schmiedchen et al., 2019; Belpomme et al., 2021), men snarere å skyldes en eller annen nocebo-virkning. Dette har ført til at man har vurdert EHS som en psykisk lidelse (Rubin et al. 2010, 2011). Den såkalte nocebo-virkningen er i beste fall en hypotese som må bekreftes gjennom dertil passende eksperimentelle studier (Belpomme et al., 2021; Belpomme og Irigaray, 2021). Men noen slike studier har slett ikke forekommet. Derimot har det, på grunnlag av et begrenset antall intervjuer av EHS-pasienter, vært framsatt mistanke om at den psykososiale atferden knyttet til EHS hos disse pasientene er sekundær til sykdomsforekomsten og lidelsen, det vil altså si en konsekvens av, og ikke en årsak til, EHS (Dieudonne, 2016). Dessuten viser de molekylære (Belpomme et al., 2015; Irigaray et al., 2018a; Belpomme og Irigaray, 2020) og radiologiske uvanlige forekomster (Heuser og Heuser, 2017; Irigaray et al., 2018b; Greco, 2020) som er blitt oppdaget hos EHS-pasienter, at EHS er en nevrologisk somatisk lidelse, og ikke en psykisk lidelse. På samme vis har MCS ikke bare vist seg å være forbundet med økt følsomhet for en rekke kjemikalier, men også å være forårsaket av enkelte innledende akutte eller nærmest akutte toksiske hendelser som har vært utløst av miljøkjemikalier – for det meste syntetiske – hos genetisk mottakelige verter (Bartha et al., 1999). Det følger derfor at verken MCS eller EHS kan anses å ha psykisk opphav. Dermed kan også EHS kjennetegnes ikke bare som en særegen tilstand av manglende toleranse for lavintensitets EMF, men også som forårsaket av forutgående for sterk EMF-eksponering. Denne banebrytende tolkningen ble opprinnelig gitt av David Carpenter ved å analysere mikrobølgesyndromet (Carpenter, 2014, 2015). Konseptet ble nylig videreutviklet i en gjennomgang av Y. Stein og I.G. Udasin (2020) som analyserte de underliggende mekanismene knyttet til EMF-eksponering som ligger bak EHS. I Tabell 6 er forekomsten av EHS-personer i den totale befolkningen uttrykt som prosentandeler, og beregnet til å variere fra 0,7 % til 13,3 %, og i hovedtrekk til å ramme i gjennom-snitt 3 %–5 % av befolkningen i mange ulike områder og land verden over, noe som betyr at millioner av mennesker faktisk kan være rammet av følsomhet for menneskeskapt EMF, og i mange av disse tilfellene av EHS. Lignende tall for hele verden kan se ut til å gjelde for MCS (Genuis, 2010). Fra analysen av våre egne data og de vi finner i den vitenskapelige litteraturen, anser vi at det nå

Tabell 6
Anslått prevalens av folk som selv-rapportert intoleranse for EMF og/eller EHS i ulike land.

Forfatter, år, land	Resultater forelå år	Utvalgs-størrelse	Svarprosent (%)**	Anslått % av folk med EHS
Hillert et al. (2002), Sverige	1997	15 000 (19–80)[a]	73	1,5
Palmquist et al. (2014), Sverige	2010	3406	40	2,7
Schreier et al. (2006), Sveits	2004	2048 (>14)[a]	55,1	5
Röösli et al., 2010a, Sveits	2008	1122 (30–60)[a]	37	8,6
Röösli et al., 2010b, Sveits	2009	1122 (30–60)[a]	37	7,7
Blettner et al. (2009), Tyskland	2004	30 047	58,6	10,3
Kowall et al. (2012), Tyskland	2004	30 047	58,4	8,7
Kowall et al. (2012), Tyskland	2006	30 047	58,4	7,2
Levallois et al. (2002), USA	1998	2072	58,3	3,2
Korpinen and P¨a¨akkönen, 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), Østerrike	2008	460	88	3,5
Furubayashi et al. (2009), Japan	2007	2472	62,3	1,2
Baliatsas et al. (2014), Nederland	2011	5789	39,6	3,5
van Dongen et al., 2014, Nederland	Before 2013	1009	60	7

[a] Når alder til pasienter som er angitt, er den tatt med i parentes.
**Svarprosenten er prosentandelen av folk som har svart positivt på spørreundersøkelsen.

foreligger en rekke sterke og overbevisende argumenter som beviser at EHS er forårsaket av ikke-termisk menneskeskapt EMF-eksponering:

1. På grunn av funnene som viser forbindelsen til unormale somatiske tilstander, så som lavgradig betennelse, oksidativt stress og påfølgende forstyrrelse/åpning av blod-hjerne-barrieren [BBB], samt i noen tilfeller autoimmun respons mot myelin PO (Belpomme et al., 2015; Belpomme og Irigaray, 2021), kan ikke EHS anses å ha sitt opphav i noen nocebo-virkning, dvs. å være en psykiatrisk lidelse. EHS bør derfor betraktes som en somatisk lidelse. I tillegg har vi vist at EHS i omtrent 25 % av tilfellene er forbundet med MCS, som allerede anses som en somatisk lidelse (Belpomme og Irigaray, 2021). Dessuten er EHS en økende, verdensomspennende farsott, så også derfor kan det ikke være rimelig å anta at EHS er en nocebo-lidelse.
2. Forekomster av EHS har dukket opp etter kunstig elektromagnetisk miljøforurensning, tilsynelatende med gradvis økende utbredelse etter at trådløse kommunikasjonsteknologier ble tatt i bruk (Bandara og Carpenter, 2018).
3. Som angitt i Tabell 6, er manglende toleranse for EMF-eksponering, herunder forekomster av EHS, ikke begrenset til enkelte regionale områder eller land, men er en verdensomspennende farsott med pandemisk utbredelse, noe som også er tilfelle for den verdensomspennende utbredelsen av teknologier som sender ut EMF (Hallberg og Oberfeld, 2006; Bandara og Carpenter, 2018).
4. Det foreligger mange uavhengige provokasjonsstudier som beviser at ELF/RF/MB EMF kan påføre organismen biologisk skade og at disse er skadelige virkestoffer for friske mennesker (se Tabell 5). Men siden man har brukt feil metodikk (se Tabell 4) ved undersøkelser av pasienter som lider av EHS, fins det bare et begrenset antall studier som viser patofysiologiske endringer og påføring av symptomer. Derfor kan de negative provokasjonsstudiene som foreligger, definitivt ikke utelukke at EMF har en rolle som årsak hos EHS-pasienter.
5. Flere EHS-tilknyttede hovedsymptomer, så som søvnforstyrrelser (Davis, 1997), depressive tendenser (Poole et al., 1993; Verkasalo et al., 1997) og selvmordsrisiko (Perry et al., 1981; Johnston, 2008), er blitt påvist i uavhengige epidemiologiske studier å være doseavhengige resultater av EMF-eksponering, noe som innebærer at for høy EMF-eksponering er årsaken til disse karakteristiske EHS-tilknyttede symptomene (Perry et al., 1981; Poole et al., 1993; Davis, 1997; Verkasalo et al., 1997; Johnston, 2008).
6. Som tidligere rapportert, er mange EHS-pasienter preget av mulig lavgradig betennelse, nitrosativt-oksidativt stress [OS], BBB-forstyrrelser/-åpning og av forandringer i hjernens nevrotransmittere (Belpomme et al. 2015, 2018; Irigaray et al., 2018a; Belpomme og Irigaray, 2020). Alt dette er i ulike uavhengige studier på laboratoriedyr påvist å være forårsaket av menneskeskapt EMF-eksponering (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).
7. De fleste EHS-pasienter har sterkt forhøyet eksponering for radiofrekvente felt fra trådløs kommunikasjon eller mikrobølgede elektromagnetiske felt (TK RF/MB EMF) og/eller ekstra lavfrekvente (ELF) felt i sin tidligere medisinske historie, noe som bekrefter at eksponering for menneskeskapt EMF kan være en hovedsakelig og plausibel årsaksfaktor for at EHS har blitt påført pasienten (Belpomme og Irigaray, 2020).
8. Mange uavhengige *in vitro* og *in vivo* studier viser at menneskeskapte elektromagnetiske felt kan samvirke med endogene fysiologiske elektriske felt som kontrollerer biologiske funksjoner på cellenivå i normale organismer (Weisenseel, 1983; Nuccitelli, 1988, 2000; Borgens, 1988; Blanchard og Blackman, 1994; Shi og Borgens, 1995; McCaig og Zhao, 1997; McCaig et al., 2005; Yao et al., 2009; Del Giudice et al., 2011; Funk, 2015). Når hele den menneskelige organismen utsettes for menneskeskapte elektromagnetiske felt, forvrenges de fysiologiske endogene elektromagnetiske feltene. Dermed forvrenges også de tilknyttede cellefunksjonene, noe som resulterer i uheldige biologiske/helsevirkninger via EMF/vevsinteraksjon på molekylært nivå (Blank, 2005; Vander Vorst et al., 2006). Dette er spesielt tilfelle for den menneskelig hjerne, for hjertet og for muskler, som alle er involvert biologisk og symptommessig i EHS, noe som er et funn som bekrefter at menneskeskapt EMF-eksponering har en rolle som en årsak som samtidig er rettet mot mange ulike mål (Frey, 1993; Vander Vorst et al., 2006).
9. Det er blitt påvist at menneskeskapte EMF og deres tilsvarende EMS er fullstendig polariserte og koherente, og at de dermed skiller seg fysisk fra naturlige EMF/EMS, som er ikke-polariserte. Denne nøkkelforskjellen kan forklare deres skadelige og toksiske virkninger på biomolekyler, celler og vev, i motsetning til naturlige elektromagnetiske felt, som er nødvendige for alt liv (Panagopoulos et al., 2015a; Panagopoulos, 2017, 2019, 2021).
10. Den patofysiologiske mekanismen som gjør at polariserte og koherente (menneskeskapte) EMF kan forårsake nevrotoksiske virkninger, er nå påvist. Mange *in vitro* og *in vivo* studier med dyr (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 og Seyhan, 2016), og med mennesker (Gandhi et al., 1996; Cardis et al. al., 2008; Dasdag et al., 2012; Belpomme et al., 2018) beviser empirisk de nevrologiske og i hovedsak hjerneskadelige virkningene av menneskeskapte ikke-termiske eller mikrotermiske elektromagnetiske felt.
11. På molekylært nivå er det påvist at ikke-termisk eller mikrotermisk lavintensitets/langvarig EMF-eksponering virker direkte på DNA, ikke bare ved å utvirke trådbrudd og fragmentering av DNA-et (Lai og Singh, 1995, 2004; Phillips et al.,

2009; Panagopoulos, 2019; Lai, 2021), men også ved å føre til kromosomendringer (Sekeroglu et al., 2012, 2013) og modifikasjon av kromatin (Belyaev og Kravchenko, 1994; Belyaev, 2005). I tillegg kan EMF-eksponering etter genetisk skade (Lai, 2021 vedlegg 1 og 2) og/eller epigenetiske endringer (Blank og Goodman, 1999; Belyaev, 2005: Belyaev et al., 2006; Leone et al., 2014; Dasdag et al., 2015a; Dasdag et al., 2015b), utvirke endringer i genregulering (Lai, 2021 vedlegg 3) og proteinfeilfolding (Millenbaugh et al., 2008). I kjølvannet av ekstern eksponering for EMF på hele organismen – som for det meste vil være tale om RFR og MB EMF – bør man faktisk rette oppmerksomheten mot en hel rekke mål på cellenivå i ulike slags vev, inkludert hjernen. Det er fortsatt uklart om de ulike genetiske og/eller epigenetiske mekanismene er involvert når EHS oppstår, men som det er påvist i mange studier, kan cellers produksjon av frie radikaler etter eksponering for ELF eller RF EMF (Lai 2019) medvirke til slike endringer. Vi har påvist at i 80 % av EHS-pasienttilfellene er EHS forbundet med produksjon av frie radikaler av typen reaktive oksygenarter (ROS) og/eller reaktive nitrogenarter (RNS), noe som tyder på at EMF kan være indirekte involvert når EHS oppstår (Irigaray et al., 2018a).

Videre er det påvist at EMF kan samvirke direkte med DNA i et bestemt område i promotoren HSP70 som påvirkes av magnetfelt, og at dette utvirker rask syntese av varmesjokkproteiner. Dette er et funn som kan forklare den antiinflammatoriske responsen som er rapportert å forekomme hos friske mennesker (Lin et al. 1999, 2001; Blank og Goodman, 1999, 2011; Blank, 2005), et resultat som også vi har påvist forekommer hos EHS-pasienter (Belpomme et al., 2015).

12. Alle disse ulike funnene taler klart for at EMF har en rolle som årsak til å påføre EHS direkte eller indirekte via ROS og/eller RNS. Selv om EMF-eksponering synes å være hovedårsaken til EHS og er i stand til å forklare både den patofysiologiske endringen og de symptomene som opptrer, er det fortsatt bare antatt hva som kan være den særegne mekanismen for EHS-genese, altså hvordan det oppstår en senkning av terskelen for intoleranse for EMF (se nedenfor). I tillegg kan MCS i noen EHS-tilfeller oppstå forut for at EHS oppstår. Derfor har vi for et begrenset antall tilfeller (11 %) antatt at også kjemikalier kan være involvert som forårsakende virkestoffer i EHS-genesen (Belpomme og Irigaray, 2020). I tillegg til de samvirkende rollene som EMF og/eller kjemikalier spiller som årsaker, vil dessuten selvstendige risikofaktorer kunne bidra til at EHS oppstår, som f.eks. en allerede eksisterende depresjon eller en psykiatrisk lidelse (Meg Tseng et al., 2011), et tidligere hjernetraume, en opportunistisk infeksjon som kan oppstå ved svikt i immunsystemet, eller en medfødt misdannelse. Slikt og annet vil kunne fremme at EHS oppstår knyttet til EMF- og/eller kjemikalie-eksponering hos genetisk og/eller epigenetisk disponerte individer. Fremtidig forskning må fokusere på disse ulike risikofaktorene med dertil passende epidemiologiske studier og egnede biokliniske metoder.

6. Hypotetiske biofysiske mekanismer som er spesielt involvert i EHS-genese

I tillegg fins det enkelte indikasjoner som støtter hypotesen om en særegen biofysisk mekanisme som kan være ansvarlig for at EMF har en rolle som årsak til å utløse overfølsomhet:

(a) Fordi mennesker, på samme måte som bakterier og mange dyr, har elektromagnetiske reseptorer, er alle mennesker følsomme for EMF, men normalt ikke overfølsomme. Slike reseptorer har blitt identifisert som «kryptokromer» i netthinnen hos dyr (Gegear et al., 2010; Grehl et al., 2016) og som «magnetosomer» i den menneskelige hjernen (spesielt i hippocampus) og i hjernehinnene (Kirschvink et al., 1992a; Dunn et al., 1995; Maher et al., 2016). Magnetosomene befinner seg hovedsakelig i de samme områder der de observerte EHS-tilknyttede patofysiologiske avvikene og kliniske symptomene hos EHS-pasienter antas å være (hippocampus og hjernehinner). Disse sistnevnte reseptorene har vist seg å inneholde jernholdig magnetitt (graigitt) og maghemittkrystaller (Kirschvink et al., 1992a) som man har antatt kan merke EMF. Dessuten har biogen magnetitt vist seg å være forbundet med ferromagnetisk resonans og å absorbere elektromagnetiske felt, og kan derfor utgjøre en mekanisme som er i stand til å produsere en viss biologisk respons ved påvirkning fra EMF (Kirschvink et al., 1992b; Johnsen og Lohmann, 2005). Siden disse reseptorene i utgangspunktet består av mineraler, tenker man seg at de kan føle ikke bare naturlig ELF, dvs. jordens magnetfelt, men også menneskeskapt polarisert statisk ELF EMF og menneskeskapt radiofrekvent stråling (RFR), der ELF [med nødvendighet] inngår. Det er altså meget mulig at mennesker har et nevrologisk system som er følsomt for geomagnetisme, slik mange andre dyr har. Men de fleste av dyrene har ikke noen bevisst oppfattelse av Jordklodens magnetfelt slik vi møter det til hverdags (Wang et al., 2019). Muligens kan de ha mistet dette felles magnetiske sansesystemet fordi det har utviklet seg noen beskyttelsessystemer [mot EMF]. Om så dette antatte anti-EMF-tilpassede nevrologiske systemet endres (eller ødelegges) ved for mye eksponering for menneskeskapt EMF (se under), kan det forklare at det oppstår overfølsomhet for EMF ved at den gjenværende opprinnelige sansningsvirkningen til magnetosomene gjenopprettes. For i tillegg å forklare den spesielle tilstanden EHS er, kan det tenkes at også andre hypotetiske EMF-følsomme reseptorer kan være involvert og settes i funksjon igjen.

(b) På molekylært nivå er det utviklet teorier om at de spenningsstyrte ionekanalene (VGICs) i cellemembraner kan være et mulig mål for polariserte og koherente (menneskeskapte) EMF (Bawin og Adey, 1976; Liburdy, 1992; Walleczek, 1992; Balcavage et al., 1996; Panagopoulos et al., 2002, 2015b, 2021). Det er blitt foreslått at biogen magnetitt, under påvirkning av EMF, kan åpne slike VGICs (Kirschvink et al., 1992b; Johnsen og Lohmann, 2005). Men VGIC'enes fysisk-kjemiske prosess, som hovedsakelig involverer kalsiumioner (Bawin og Adey 1976; Liburdy, 1992; Walleczek, 1992; Pall, 2013), har man antatt blir brukt av alle celler i organismen. Derfor kan ikke denne teorien forklare den unike sansemekanismen/virkningen som er knyttet til EHS og de spesielle EHS-tilknyttede patofysiologiske endringene som observeres i CNS, spesielt i hippocampus og hjernehinnene. Andre mekanismer/virkninger som skyldes EMF kan derfor være involvert.

(c) Det er påvist i laboratoriedyr at elektromagnetiske felt og/eller kjemikalier særlig kan skade nevroner (Frey, 1993; Redmayne og Johansson, 2014; Megha et al. 2015a, 2015b), og at de kan endre proteinkonsentrasjoner knyttet til nevrotransmittere og synapser, særlig i hippocampus (Bas et al., 2009; Leone et al., 2014; Teimori et al., 2016; Tan et al., 2019). Dessuten er nevroner mer sårbare for å utsettes for EMF-påført programmert celledød enn andre celler i organismen (Salford et al., 2003; Joubert et al., 2008; Sonmez et al., 2010; Zuo et al., 2014; Odaci et al., 2003. , 2016; Eghlidospour et al., 2017). Siden EHS, som tidligere definert, klinisk sett synes å være en erverver og vedvarende tilstand, er vår hypotese at hos EHS-pasienter har menneskeskapte EMF og/eller kjemikalier som er i handelen, permanent endret eller ødelagt nevroner i det adaptive beskyttelsessystemet og i nevronkretser i hjernen, muligens i hippocampus (Belpomme og Irigaray, 2020). Dette er en vei å følge for videre biofysisk og patofysiologisk forskningsinnsats for bedre å finne fram til hva som kjennetegner (over)følsomheten knyttet til EHS og/eller MCS, for så å vurdere vår fremsatte hypotese via mer spesifikke nevrologiske undersøkelser av CNS.

7. Drøfting

Ved å bruke en rekke biomarkører i det perifere blodet [dvs. det blodet som flyter rundt i kroppen] og urinen, og egnede hjerneavbildningsteknikker (Irigaray et al., 2018b; Belpomme og

Irigaray, 2021), har vi tidligere ført belegg for at EHS er en hjernepatologisk lidelse som kan diagnostiseres objektivt og behandles. Dessuten er det blitt påvist at selv om EHS og MCS er forskjellige i sin etiologi og patogenese, deler de både en lignende klinisk og biologisk signatur, slik at de må betraktes medisinsk som deler av et eget unikt nevrologisk miljøintoleranse-syndrom (Belpomme et al., 2015). Dette er noe som mange forskere nylig ble enige om i en vitenskapelig konsensusrapport som fremhever det vendepunktet som rollen til biomarkører og bildebehandling har skapt for studiet av EHS (Belpomme et al., 2021). Vårt funn om EHS, som hovedsakelig er basert på bruk av biomarkører og egnede bildeteknikker, må imidlertid bekreftes gjennom andre studier, men vi viser her at den fremgangen i forskningen som alt foreligger, medfører at EHS erkjennes å være en ekte patologisk lidelse som er forårsaket av EMF-eksponering. Selvsagt bør det gjøres ytterligere forskningsinnsats for å bevise definitivt den rollen EMF spiller som årsak i å utløse EHS-symptomer og i EHS-genesen som sådan. Imidlertid oppfyller de forskjellige og uavhengige dataene vi har lagt fram, de årsakskriteriene som WHO har foreslått (WHO, 2006) ettersom a) de omfatter en dose-responsvirkning for de viktigste EHS-tilknyttede symptomene i epidemiologiske studier, b) de bevitner at biologiske endringer av *in vitro* og *in vivo* laboratoriedyr som er utsatt for menneskeskapte EMF, ligner på det som observeres hos EHS-pasienter, c) de gir også empirisk belegg for en EHS-tilknyttet ikke-termisk eller mikrotermisk patofysiologisk mekanisme som kan forklare forekomstene av symptomer, og fremfor alt d) de følger fullt ut de generelle vitenskapelige prinsippene som brukes av ulike uavhengige forskerteam. Dataene støtter derfor påstanden om at menneskeskapte EMF har en rolle som årsak til EHS. I tillegg er det tydelig demonstrert i ulike uavhengige studier gjennom bruk av provokasjonstester at EMF er skadelig for friske mennesker. Følgelig foreligger det tilstrekkelig av etablerte fakta til sterkt å anbefale beskyttelsestiltak mot dagens menneskeskapte elektromagnetiske forurensning, og til å bruke føre-var-prinsippet for å beskytte spesielt gravide kvinner, spedbarn, barn, tenåringer og unge voksne i alle land over hele verden. Gitt de syv milliarder menneskene det fins i verden – hvorav de fleste bruker trådløse telefoner og/eller mobiltelefoner, Wi-Fi og andre trådløse enheter – og gitt dagens og den fremtidige utviklingen av 5G (Hardell og Nyberg, 2020; Hardell og Carlberg, 2020; Pall, 2021), forventes det at forekomsten av intoleranse for EMF og av EHS vil øke betydelig over hele verden i løpet av de neste årene. Men fordi tallene som er angitt i Tabell 6 er estimater og ikke er basert på objektive kriterier for å identifisere EHS (Hallberg og Oberfeld, 2006), mener vi at disse dataene må bekreftes ved hjelp av mer objektive vurderinger. Selv om de rapporterte tallene for EHS-prevalens kun er estimater, forventes det at intoleranse for EMF og prevalensen av EHS vil fortsette å vokse, i takt med at produsentene av trådløse teknologier og kjemisk industri vil fortsette utviklingen av sine produkter.

Som rapportert i denne oversikten, har det siden WHOs offisielle publikasjoner fra 2005 og 2014 blitt gjort store fremskritt i identifisering og forståelse av EHS (og MCS) som patologiske lidelser og av de biokliniske helsevirkningene som menneskeskapte EMF og/eller kjemikalier har på organismer. Men EHS og MCS er fortsatt ikke blitt anerkjent i rimelig grad av WHO. De ikke-termiske og/eller mikrotermiske helsevirkningene fra menneskeskapt EMF-eksponering, som er påvist på dyr så vel som på mennesker, og deres fysisk-kjemiske virkningsmekanismer (Pall, 2013; Yakymenko et al., 2016; Belpomme og Irigaray, 2020; Panagopoulos et al., 2021) bør vurderes av WHO. I motsetning til de urealistiske påstandene fra Den internasjonale kommisjonen for ikke-ioniserende strålevern (ICNIRP), som fortsatt benekter eksistensen av ikke- og/eller mikrotermiske biologiske og toksiske helsevirkninger fra menneskeskapte elektromagnetiske felt (ICNIRP 1998, 2010, 2020), understreker vi igjen at det i det siste er gjort avgjørende fremskritt i forskningen, noe som gjør at ikke- og mikrotermiske virkninger fra EMF i dag er alminnelig erkjent blant forskere og av folk i sivilsamfunnet, noe som bevitnes av de mange internasjonale vitenskapelige appeller som krever en rimelig begrensning av den elektromagnetiske forurensningen og avvikling eller et *moratorium* i utviklingen av 5G (Hardell og Nyberg, 2020; Hardell og Carlberg, 2020; Pall, 2021). Det ser ut til å være et faktum at de ikke-termiske eller mikrotermiske toksiske helsevirkningene fra EMF (i tillegg til de MCS-forbundne miljøvirkningene) er årsaken til patogenesen og etiologien bak EHS, og at helsevirkningene fra EMF også er en mulig årsak til kreft (Hardell et al., 1995; IARC, 2002; Belpomme et al., 2007; IARC, 2013; Hardell et al., 2013).

Videre bør behovene for helsehjelp til mennesker med miljøømfintlighet, så som EHS og MCS, defineres og utvikles for dagens sosioøkonomiske miljø og som medisinske utfordringer (Gibson et al., 2015).

Dagens vitenskapelige kunnskapsnivå legger et stort etisk ansvar på forskere og myndigheter og nasjonale og internasjonale helseorganer for å avdekke de negative helsevirkningene av den økende menneskeskapte EMF-eksponeringen, og for å advare mot de nye og verdensomspennende EHS- og MCS-farsottene som nå øker på overalt i verden. Dette betyr at passende tiltak innen folkehelsen snarest må iverksettes for å anerkjenne EHS og MCS som nye patologier og for å redusere EMF-eksponeringen.

Vi ber derfor WHO på det sterkeste om, på det grunnlag at EHS og MCS er klinisk og patofysiologisk identifisert, å legge disse lidelsene til i fremtidige versjoner av WHOs internasjonale klassifikasjon av sykdommer, akkurat slik man allerede har gjort for andre sykdommer og lidelser som er erkjent.

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

Dominique Belpomme [a,b,*], Philippe Irigaray [b]

[a] Medical Oncology Department, Paris University, Paris, France
[b] European Cancer and Environment Research Institute (ECERI), Brussels, Belgium

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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).

* Corresponding author. ARTAC, 57/59 rue de la Convention, 75015, Paris, France.
E-mail address: contact.belpomme@gmail.com (D. Belpomme).

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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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