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The Determinants of Electromagnetic Hypersensitivity

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The Determinants of Electromagnetic Hypersensitivity

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All scientific work is incomplete – whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action that it appears to demand at a given time.

- Sir Austin Bradford Hill, Professor Emeritus of Medical Statistics, University of London, 1965

Certification

I, Adam Verrender, declare that this thesis, submitted in fulfilment of the requirements for the conferral of the degree Doctor of Philosophy, from the University of Wollongong, is wholly my own work unless otherwise referenced or acknowledged. The document has not been submitted for qualifications at any other institution.

Adam Verrender

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Abstract

Electromagnetic Hypersensitivity (EHS), more formally known as Idiopathic Environmental Intolerance attributed to Electromagnetic Fields (IEI-EMF), is a controversial condition characterised by the experience of a broad range of non-specific symptoms which a person attributes to the electromagnetic fields (EMF) emitted by everyday electronic and wireless technologies. In contrast to growing anecdotal reports of sensitivity to EMF, much of the existing literature has not found evidence that exposure to EMF can result in the symptoms reported by IEI-EMF sufferers. Instead, the condition is thought to be the result of a nocebo effect, where conscious or subconscious symptom expectation leads to the development and detection of symptoms. Yet, despite decades of research, IEI-EMF sufferers and a minority of scientists argue that the symptoms are caused by exposure to EMF, via some as-yet unrecognised bioelectromagnetic mechanism.

In an effort to resolve the aetiological debate, this thesis aimed to clarify whether toxicogenic or psychogenic processes can explain the symptoms reported by IEI-EMF sufferers. Specifically, a number of methodological issues were addressed to more clearly determine whether individuals can be sensitive to EMF exposure, or whether psychogenic processes play a role in the presentation of symptoms attributed to EMF exposure.

First, as a means of establishing whether radiofrequency EMF (RF-EMF) exposure below the established safety guidelines could elicit adverse effects in humans and in an attempt to determine the most sensitive objective endpoints to test IEI-EMF participants, Study 1 investigated whether exposure to RF-EMF influences human cognitive performance in a dose-dependent manner. While the results showed that

exposure to RF-EMF can improve reaction time on a working memory task, given that methodological improvements employed in this study have not yet been replicated, and given that the effect was not found to be dose-dependent and that an improvement in performance does not reflect an adverse health effect, Study 1 did not provide convincing evidence that exposure to RF-EMF can adversely affect cognitive functioning. Furthermore, the study did not identify any sensitive cognitive performance measures with which to test IEI-EMF sufferers. This line of research was thus discontinued.

Study 2 aimed to determine whether the symptoms reported by IEI-EMF sufferers can be explained by toxicogenic or psychogenic processes, and incorporated several methodological improvements to overcome the limitations of previous research. The study was designed as a series of individual case studies to test whether exposure to RF-EMF results in an increase in IEI-EMF participants' self-nominated symptoms compared to sham, and , to determine whether IEI-EMF individuals could accurately detect the presence of RF-EMF emissions under double-blind conditions. Despite accounting for a number of potential limitations, the results of the case studies failed to demonstrate that the symptomatic response of self-reported IEI-EMF sufferers is affected by RF-EMF exposure, nor that IEI-EMF sufferers can detect the presence of RF-EMF emissions at greater than chance levels. While all participants displayed an increased symptom severity and were confident that they could detect the presence of RF-EMF in the open-label RF-ON but not RF-OFF trial, no significant differences in symptom severity or exposure detection were found between the double-blind RF-ON and RF-OFF (sham) conditions. Notably, a significant relationship between a participant's belief that they were being exposed (irrespective of the actual exposure

condition) and their symptomatic response was observed, giving a strong indication the symptoms experienced were due to a nocebo response, and therefore, that IEI-EMF can be explained by psychogenic processes.

Study 3 extended upon Study 2 by investigating whether a nocebo response is specific only to IEI-EMF sufferers, and by examining the potential role of psychological processes in the presentation of symptoms attributed to EMF exposure. Healthy participants were randomly assigned to watch either an alarmist video emphasising 'adverse effects of EMF exposure' or a control video completely unrelated to EMF and health, before completing a series of open-label and double-blind provocation trials. Consistent with Study 2, results showed that healthy participants reported higher symptoms in the open-label RF-ON compared to the RF-OFF trial. However, in the subsequent double-blind trials, no difference in either belief of exposure or symptoms was found between the RF-ON and sham conditions. Belief of exposure was also positively associated with higher symptom scores in the double-blind trials, further indicating that a nocebo effect, rather than EMF exposure itself, was responsible for the increase in symptoms. Additionally, participants who viewed the alarmist video reported higher symptom scores in the open-label trials, as well as a greater increase in state anxiety and risk perception from baseline, than those who viewed the control video. This indicates that viewing sensationalist media reports about perceived environmental hazards raises concerns and negative beliefs about EMF exposure, and may exacerbate a nocebo response, suggesting that the degree to which people experience symptoms that they believe are associated with EMF exposure may be influenced by media reporting.

Taken together, the findings from this thesis do not support the notion that toxicogenic processes can explain the symptoms attributed to EMF exposure. Instead the studies presented provide strong support for the view that psychological factors play an important role in triggering, maintaining, or exacerbating symptoms in response to perceived exposure to EMF.

Publications Constituting this Thesis

Verrender A., Loughran, S.P., Dalecki, A., McKenzie, R.J., & Croft, R.J. (2016). Pulse modulated radiofrequency exposure influences cognitive performance. *International Journal of Radiation Biology, 92,* 1- 8. (Chapter 2)

Verrender A., Loughran, S.P., Anderson, V., Hillert, L., Rubin, G.J., Oftedal, G., & Croft, R.J. (2018). IEI-EMF provocation case studies: A novel approach to testing sensitive individuals. *Bioelectromagentics, 39,* 132 – 143. (Chapter 3)

Verrender, A., Loughran, S.P., Dalecki, A., Freudenstein, F., & Croft, R.J. (2018). Can explicit suggestions about the harmfulness of EMF exposure exacerbate a nocebo response in healthy controls? *Environmental Research, 166,* 409 – 417. (Chapter 4)

Statement of Verification

This statement verifies that the greater part of the work in the above-named manuscripts is attributed to the candidate. Adam Verrender, under the guidance of his supervisors, took primary responsibility for the design of each study, data collection and analysis, prepared the first draft of each manuscript, and prepared the papers for submission to relevant journals. Co-authors, who included the supervisors of the candidate and relevant colleagues with expertise in the field, contributed to the thesis by providing guidance on the design, analysis and general structure of each study, and provided editorial suggestions for each paper.

Adam Verrender (PhD Candidate)

Professor Rodney Croft (Primary Supervisor)

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Verrender, A., Loughran, SP., Croft, RJ. Determining the aetiology of Idiopathic Environmental Intolerance attributed to Electromagnetic Fields: RF exposure or Nocebo effect? *The 2017 Australasian Radiation Protection Society Conference, Wollongong, Australia, August 06 – 09, 2017.*

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Verrender, A., Loughran, SP., Dalecki, A., Mackenzie, R., Croft, RJ. Pulse modulated radiofrequency exposure influences cognitive performance. *Joint Annual Meeting of the Bioelectromagnetics Society and the European Bioelectromagnetics Association, Ghent, Belgium, June 05 – 10, 2016.*

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Verrender, A., Loughran, SP., Anderson, V., Croft, RJ. IEI-EMF provocation case studies: A novel approach to testing sensitive individuals. *Joint Annual Meeting of the Bioelectromagnetics Society and the European Bioelectromagnetics Association, Asilomar, United States of America, June 14 - 19, 2015.*

Abbreviations

3G	3rd Generation
4G	4th Generation
5G	5th Generation
CBT	Cognitive Behavioural Therapy
CCK	Cholecystokinin
EEG	Electroencephalogram
EHS	Electromagnetic Hypersensitivity
ELF	Extremely Low Frequency
EMF	Electromagnetic Field(s)
EOG	Electrooculography
ES	Effect Size
GHz	Gigahertz
GP	General Practitioner
GSM	Global System for Mobile Communications
HPA	Hypothalamic-Pituitary-Adrenal Axis
Hz	Hertz
ICNIRP	International Commission on Non-Ionising Radiation Protection
IEI	Idiopathic Environmental Intolerance(s)
IEI-EMF	Idiopathic Environmental Intolerance 'attributed to' Electromagnetic Fields
IHS	Infrasound Hypersensitivity
ISM	Industrial, Scientific and Medical (Radioband)
kHz	Kilohertz
M	Mean

MCS	Multiple Chemical Sensitivity
MHz	Megahertz
MRI	Magnetic Resonance Imaging
MUS	Medically Unexplained Symptoms
NEO-FFI	Neo Five Factor Personality Index
PM-RF	Pulse Modulated Radiofrequency
RF	Radiofrequency
RPQ	Risk Perception Questionnaire
RT	Reaction Time
SAR	Specific Absorption Rate
SD	Standard Deviation
SESS	Symptom and Exposure Status Scale
STAI	State Trait Anxiety Index
UV	Ultra Violet
VAMS	Visual Analogue Mood Scale
VDU	Visual Display Unit
W	Watt
W/kg	Watts per Kilogram
W/m^2	Watts per Metre Squared
W-CDMA	Wideband Code Division Multiple Access
WHO	World Health Organisation
WHOQOL-BREF	World Health Organisation Quality of Life Brief Questionnaire
Wi-Fi	Wireless Fidelity Network
W-LAN	Wireless Local Area Network

1. CHAPTER 1: GENERAL INTRODUCTION

The research presented in this doctoral thesis was designed to investigate the determinants of Idiopathic Environmental Intolerance attributed to Electromagnetic Fields (IEI-EMF), a controversial condition more commonly referred to as Electromagnetic Hypersensitivity (EHS). While continuing technological advances have generally benefited greater society, the unprecedented rise in the number and diversity of electromagnetic field (EMF) sources has raised public concerns about potential adverse health risks posed by our increasing exposure to EMF. Amongst these concerns are the reports of a proportion of the population who claim to experience a variety of health problems which they attribute to exposure to EMF. As the associated symptoms are debilitating for many, it is crucial to understand the determinants of this condition. Two alternate theories have been proposed to explain the origin of the symptoms reported by individuals who suffer from IEI-EMF; namely the toxicogenic theory and the psychogenic theory. While much of the extant literature indicates that the reported symptoms are of a psychogenic origin, a number of methodological issues need to be overcome before this can be conclusively determined, and much remains to be clarified. In an effort to resolve the aetiological debate, the empirical studies presented in this thesis specifically sought to address a number of methodological issues to more clearly determine whether individuals can be sensitive to EMF exposure, and to examine the potential role of psychogenic processes in the presentation of symptoms attributed to EMF exposure. The following chapter outlines the general background and rationale for this research, the research aims, and the significance and originality of this research.

1.1 Background

The use of wireless technology has rapidly increased over the past two decades with the continuing development of mobile phone, laptop, tablet and smart devices. Globally, the United Nations estimates that there are approximately 7 billion active mobile phone subscriptions (International Telecommunications Union, 2017), a figure almost equivalent to the total human population. In Australia, one of the country's largest telecommunications companies has introduced over 2 million public wireless fidelity (Wi-Fi) hotspots since 2015 to cope with the increasing demand for access to high speed wireless internet (Telstra, 2014), while state utility companies across the nation are beginning to introduce smart metres, which communicate wirelessly, to track household electricity use (Energy Australia, 2014). By 2020, the roll out of 5G technology will further drive society globally into the 'internet of things', where various numbers of smart devices will communicate wirelessly with each other in an effort to improve the lives of the people who use them. There is probably no other technology that has been so quickly and widely adopted by the general public in recent times (Stewart, 2008).

Yet, despite the benefits associated with the introduction and widespread use of wireless technologies, concerns have been raised about whether there may be adverse health effects associated with our increasing exposure to the non-ionising EMF utilised by these devices. These concerns have been partially driven by reports from a proportion of the population who claim to have detected a clear association between their experience of distressing and sometimes debilitating symptoms and their exposure to the EMF emitted by various everyday technologies and infrastructure (as described in Vignette

1). These individuals suffer from a condition commonly described as Electromagnetic Hypersensitivity (EHS).

Vignette 1:

Steve (aged 45 years), is an IT professional who has been using computers his entire adult life. He has always considered himself an early adopter of new technologies. In 2001, after purchasing one of the most powerful Wi-Fi routers at the time, Steve began to experience symptoms including pressure in the chest, pressure in the head, mood changes, and tingling sensations in the hands and face within minutes using the Wi-Fi router. After turning off the Wi-Fi router, he experienced a headache that persisted for several hours. Steve soon believed that there was a consistent pattern between the symptoms that he was experiencing and the use of his Wi-Fi router. With the subsequent rollout and advancement of other wireless technologies including mobile phones, smart metres, digital cordless telephones and inflight Wi-Fi, Steve's health worsened to a point where he began to experience sleeping difficulties, constant headaches and extreme lethargy. These symptoms then resulted in a complete loss of motivation to do activities with his family and forced him to only use the rear parts of his home, which he shielded with metallic paint and "RF blocking" curtains. He can no longer drive through suburbs where smart metres have been installed without developing a serious headache that can last for several days. Steve is deeply concerned about the lack of support, care and understanding he has experienced from medical professionals, power utilities and various government departments and is adamant that his symptoms can be attributed to EMF exposure (Weller, 2014).

The aetiology of this condition, however, is extremely controversial. While some researchers and many of those who suffer from the condition believe that it is caused by exposure to EMF, to date, there has been insufficient scientific evidence to support this claim. Instead, much of the evidence suggests that that the condition is likely the result of a nocebo response (Röösli, Frei, Mohler, & Hug, 2010; Rubin, Das Munshi, & Wessely, 2005; Rubin, Nieto-Hernandez, & Wessely, 2010), where conscious or subconscious symptom *expectation* following a *perceived* exposure to EMF leads to the formation or detection of symptoms. Due to the lack of evidence for a relationship between the reported symptoms and exposure to EMF, the World Health Organisation (WHO) proposed the term 'Idiopathic Environmental Intolerance attributed to Electromagnetic Fields' (IEI-EMF) to be used in place of 'Electromagnetic Hypersensitivity' (EHS), in order to avoid implying a causal role of EMF in producing the reported symptoms (World Health Organisation, 2004)[1].

Idiopathic Environmental Intolerance (IEI) is an umbrella term used to describe a group of health conditions that are characterised by the experience of a wide range of somatic, non-specific symptoms which are claimed to arise in response to environmental triggers, but for which there is no established evidence of a relationship between the claimed environmental triggers and adverse symptoms (Van den Bergh, Brown, Petersen, & Witthöft, 2017). As well as being used to describe the symptoms which people attribute to EMF, the IEI term has also been used to describe a number of other conditions such as Multiple Chemical Sensitivity (MCS), which refers to the attribution of symptoms to a wide range of everyday chemical sources (which may include

[1] Consistent with this recommendation, the present thesis uses 'IEI-EMF' as a neutral term to refer to EHS.

cleaning products, air pollution and pesticides); and Infrasound Hypersensitivity (IHS), more commonly known as Wind Turbine Syndrome, which refers to the attribution of symptoms to the low frequency noise emitted by wind turbines. The aetiologies of the IEI conditions are controversial for a number of reasons. First, there is often no established relationship between the environmental exposure claimed to trigger symptoms and organ pathology or dysfunction. Second, the reported symptoms are extremely heterogeneous, and are often alleged to be triggered by environmental exposures at strengths well below the thresholds currently known to cause adverse health effects. In addition to this, there is generally no evidence to suggest that the symptoms claimed by IEI sufferers are associated with the claimed exposures. For instance, MCS and IEI-EMF sufferers who participate in well-designed laboratory studies which use double-blind protocols and utilise the particular environmental exposures purported to be responsible for symptoms generally fail to have their symptom claims verified in active compared to sham exposures, and instead behavioural and psychological processes have been consistently found to play a role in the presentation of symptoms (Das-Munshi, Rubin, & Wessely, 2006; Rubin et al., 2005; Rubin et al., 2010). Likewise, there is no evidence of a direct causal link between living in close proximity to wind turbines, the noise they emit and the physiological health effects claimed to be attributable to wind turbines by IHS sufferers (Knopper & Ollson, 2011). Finally, there is a large symptom overlap between IEI's and other somatoform disorders and functional syndromes such as chronic fatigue syndrome and fibromyalgia (Van den Bergh et al., 2017).

Like the other IEI conditions, the discrepancy between the scientific evidence and the reports of sensitivity to EMF has instigated a highly contentious debate concerning the

aetiology of IEI-EMF. On one side, advocates of the toxicogenic explanation argue that the adverse symptoms reported by IEI-EMF sufferers are the result of an intolerance or susceptibility to exposure to low levels of EMF, which affects the human body via some as-yet unrecognised toxicogenic or 'bioelectromagnetic' mechanism (Rubin et al., 2010; Staudenmayer, Binkley, Leznoff, & Phillips, 2003a). Alternatively, proponents of a psychogenic explanation contend that the condition can be explained by an individual's overvalued belief of toxicity, a belief which is shaped by a range of psychological, psychosocial and psychophysiological processes (Staudenmayer, Binkley, Leznoff, & Phillips, 2003b) and which ultimately culminates in the presentation of adverse symptoms via a nocebo response.

Given the debilitating nature of IEI-EMF (as described in Vignette 1), resolving the aetiological debate is extremely important, not least because the two opposing theories have very different implications in terms of identifying and developing the most appropriate treatments and support for those who experience the condition (Rubin, Das Munshi, & Wessely, 2006; Rubin et al., 2010). For instance, if EMF exposure is indeed responsible for the development of these symptoms, then exposure reduction and avoidance may be the most suitable strategy to alleviate symptoms. Conversely, if the symptoms are the result of a nocebo effect, then a psychologically complex scenario needs to be dealt with. Investigating the determinants of this condition forms the focus of this thesis.

1.2 Electromagnetic Fields and Health

Electromagnetic fields (EMF) are waves of energy which radiate through space. These waves are generated by both natural and man-made sources, and are comprised of two components, an electrical field (*E*-field) and a magnetic field (*H*-field) (Wood & Roy,

2017). *E*-fields are measured in volts per-metre (V/m) and are present whenever positive or negative electrical charges exert force on other charged particles in the field. *E*-fields are strongest closer to the device (for e.g. a mobile phone) and diminish rapidly with greater distance from the source (Wood & Roy, 2017; World Health Organisation, 2018). *H*-fields are measured in amperes per metre (A/m) and are produced by the physical movement of electrical charges. Like *E*-fields, *H*-fields are strongest closer to the source and diminish with greater distance from the source (Wood & Roy, 2017; World Health Organisation, 2018). EMFs are defined by the frequency or corresponding wavelength of the electromagnetic wave. Frequency is used to describe the number of oscillations or cycles per second (typically measured in Hertz (Hz)), while wavelength is used to describe the distance between one peak (or trough) of the electromagnetic wave and the next peak (or trough) (World Health Organisation, 2018). Frequency and wavelength have an inverse relationship, the higher the frequency, the shorter the wavelength.

EMF can be classified into different ranges based on frequency and/or wavelength. When arranged on a continuum they form what is known as the electromagnetic spectrum. Based on frequency, the spectrum is divided into two distinct categories; ionising and non-ionising radiation. Figure 1.1 illustrates the distinction between ionising and non-ionising EMF and delineates the frequency bands typically used to refer to types of EMF, and the typical sources that utilise each frequency.

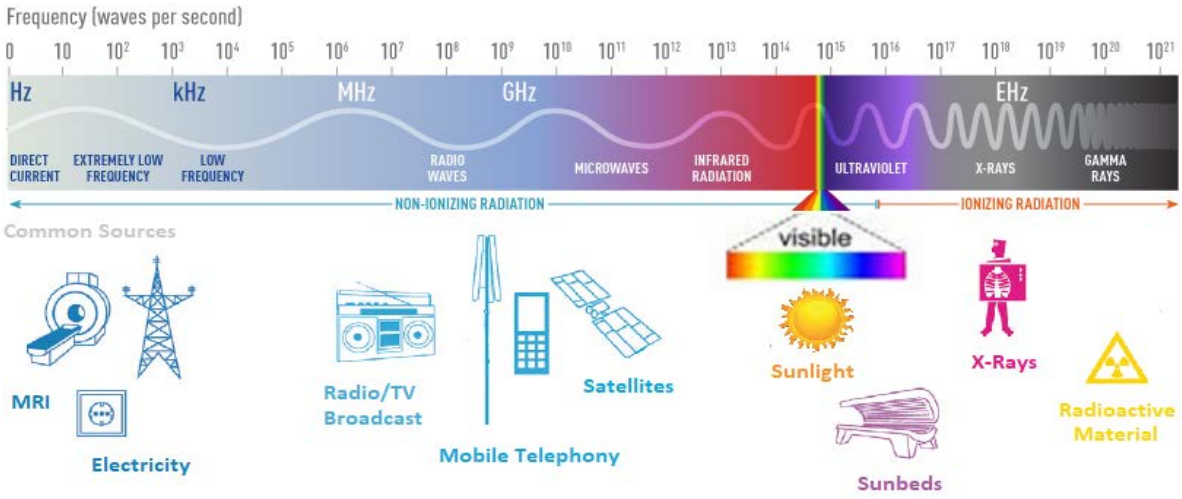

Figure 1.1: The electromagnetic spectrum and associated sources (adapted from the National Cancer Institute https://www.cancer.gov/PublishedContent/Images/images/infographics/electromagnetic-spectrum-enlarge.__v10067782.png)

Ionising radiation is at the high frequency (and thus high-energy) end of the spectrum and includes X-rays and gamma rays. Ionising radiation carries enough energy to free electrons from atoms or molecules, thereby creating free radicals, which are highly reactive. These free radicals can effectively break chemical bonds and damage biological tissue. While the destructive and detrimental effects of ionising radiation are well-known (for example when used in atomic weapons or during and following a nuclear reactor meltdown), ionising radiation is also used in a beneficial way in medicine, for example in radiography and cancer treatments. Non-ionising radiation, on the other hand, is at the lower-energy end of the spectrum and refers to the electromagnetic radiation that does not carry sufficient energy to remove electrons from atoms or molecules. Extremely Low Frequency (ELF) radiation, Radiofrequency (RF) radiation, infrared radiation, visible light, and Ultra Violet (UV) radiation are all forms of non-ionising EMF. Generally, electronic and wireless technologies utilise EMF in the ELF (from 3 to 30 Hz) and RF (from 3 kHz to 300 GHz) bands of the non-ionising

radiation domain (ARPANSA, 2012). A number of biological and health effects from exposures to high intensities of non-ionising EMF have been well documented. These effects generally relate to the localised heating or stimulation of excitable tissue that is associated with the amount of energy absorbed by the body (Repacholi, 1998). The rate and distribution of energy absorption in the body depends strongly on the frequency, strength and orientation of the incident EMF[2] as well as the body's size and its electrical properties (Health Canada, 2015).

The absorption of RF-EMF by the human body is commonly described in terms of the specific absorption rate (SAR), which is a measure of the rate of energy deposition per unit mass of body tissue and is usually expressed in units of watts per kilogram (W/kg). It is important to note that the high exposure levels known to adversely affect human health via thermal mechanisms do not exist in daily life, as the technologies which utilise RF-EMF are heavily regulated by governments and health agencies (for example ARPANSA, 2002) using internationally recognised safety guidelines (for example ICNIRP, 1998). For instance, the ICNIRP (1998) RF-EMF guidelines are based on SAR limits at many orders of magnitude lower than the thresholds known to cause temperature increases that would affect human health. For the general public, these guidelines recommend limiting the SAR to 2 W/kg; while for people undergoing occupational exposure, the guidelines recommend a limit of 10 W/kg (averaged over 10 grams of tissue) (ICNIRP, 1998). Likewise, internationally recognised safety guidelines have been developed to protect humans against the potential adverse effects of ELF-EMF exposures (ICNIRP, 2010). Exposure to high intensity ELF-EMF may cause well-defined biological responses, including perception and annoyance, alterations in some

[2] From herein, the present thesis refers to 'non-ionising EMF' as 'EMF' for readability

aspects of brain and nervous system function, and the induction of retinal phosphenes (the perception of faint flickering light in the absence of visual input). The physical quantity used to specify the exposure limits for ELF-EMF is the internal electric field strength (E_i), as it is the electric field that affects nerve cells and other electrically sensitive cells. However, as this value is often difficult to derive, reference levels based on measurements (or computations) of electric field strength, magnetic field strength, magnetic flux density and currents flowing through the limbs can be used to ensure compliance with the relevant exposure limit (ICNIRP, 2010). With the exception of some medical exposures to patients and some specialised occupational exposures, exposure to the high levels of ELF-EMF that are known to cause adverse health effects in humans are extremely rare, and are unlikely to occur in daily life.

Despite a number of authoritative reviews of the scientific literature generally concluding that there are no established health risks associated with exposure to EMF within the established guidelines, nor any established mechanism by which this could occur (Health Canada, 2015; Health Council of the Netherlands, 2009; SCENIHR (Scientific Committee on Emerging and Newly Identified Health Risks), 2009; World Health Organisation, 2014), the rapid development and increasing use of wireless technologies has generated considerable public concern about potential adverse health effects of exposure to EMF below the established exposure guidelines (World Health Organisation, 2018).

Evidence that EMF exposure below the established guidelines can have biological or physiological effects is often used by IEI-EMF sufferers and advocates as arguments to suggest that EMF exposure can adversely affect health (for example. BioInitiative Working Group, 2012). It is important in these instances, however, to distinguish

between biological/physiological effects and health effects. Biological/physiological effects are measurable responses to a stimulus or to a change in the environment, but they are not necessarily harmful for human health (World Health Organisation, 2018). These often occur in daily life, for example when eating food, playing sport or listening to music. Conversely, an adverse health effect is something which results in a detectable impairment in the health of the exposed individual (World Health Organisation, 2018). One of the most consistently reported effects of RF-EMF exposure, similar to that emitted by mobile phones, are alterations in the brain's electrical activity, specifically in the spontaneous resting alpha (8 – 12 Hz) (Croft et al., 2008; Croft et al., 2010; Curcio et al., 2005; Leung et al., 2011) and the sleep spindle frequency range (approximately 11 – 15 Hz) (Huber et al., 2000; Huber et al., 2002; Loughran et al., 2005; Regel et al., 2007; Schmid et al., 2012) of the electroencephalograph (EEG). These effects have been found to occur in a dose-dependent manner. Although dose dependency is a fundamental principle of toxicology, and refers to the change in effect caused by differing levels of exposure (Staudenmayer et al., 2003a), the change in EEG as a result of exposure to RF-EMF may not necessarily reflect an adverse health effect.

While the EEG is closely related to cognitive and mental processes and states (Andreassi, 2007), studies investigating the effect of RF-EMF on gross measures of cognitive performance, such as response times and accuracy, have produced contradictory, but mostly null results. For instance, one meta analyses reported that human attention and working memory is affected by exposure to EMF (Barth et al., 2008), while other meta analyses have found no influence of exposure on gross measures of performance (Barth, Ponocny, Gnambs, & Winker, 2012; Valentini, Ferrara, Presaghi, De Gennaro, & Curcio, 2010). The inconsistent results, however, may

be due to a number of methodological issues, including variation in methods between research groups (making it difficult to compare or verify previous results), poor exposure protocols and experimental designs, inadequate sample sizes and a lack of reliable cognitive performance measures (Regel & Achermann, 2011). In addition, individual differences in cognitive performance have not been adequately accounted for in previous studies, nor has the potential influence of thermal variability, which may be particularly important given that exposure to RF-EMF imparts a thermal load on the body (Adair & Black, 2003). Given these issues, the associated functional consequence(s) of the change in EEG, if any, remains to be clearly determined, and as such, there is some uncertainty as to whether exposure to RF-EMF can impair cognitive performance.

The World Health Organisation (WHO) defines health as "*a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity*" (World Health Organisation, 1946).While, to date, there is no evidence of any adverse health effects of EMF exposure below the established safety guidelines (Health Canada, 2015; Health Council of the Netherlands, 2009; SCENIHR (Scientific Committee on Emerging and Newly Identified Health Risks), 2009; World Health Organisation, 2014), the prevalence of adverse symptoms attributed to EMF exposure, while currently lacking evidence of a direct causal relationship (Röösli et al., 2010; Rubin et al., 2005; Rubin et al., 2010), may be viewed by many in the community as a major health effect, warranting the need for further investigation.

1.3 Perceived sensitivity to EMF

Anecdotal reports of sensitivity to devices which emit EMF began to emerge in the late 1970's, when reports of facial skin symptoms related to workers using visual display

units (VDU) began to arise in Great Britain and Norway, and were followed by similar reports in Sweden, the United States and Japan (Lindén & Rolfsen, 1981; Nilsen, 1982; World Health Organisation, 2004). As technology progressed, so too did the reports of sensitivity to devices which emit EMF, with emitters such as mobile phones and Wi-Fi now claimed to adversely affect individuals. While there is currently no established diagnostic criterion to identify individuals suffering from IEI-EMF (World Health Organisation, 2004), generally, the condition is characterised by a person experiencing a broad range of dermatological, neurasthenic, vegetative or mood symptoms (see table 1.1) which they attribute to the EMF emitted by various electronic and wireless devices or infrastructure.

The difficulty in establishing a clear aetiology for the symptoms reported by IEI-EMF sufferers is compounded by the heterogeneous nature of the condition. No consistent pattern in either the types of symptoms, the time it takes for symptoms to develop and subside or the sources of EMF that are claimed to trigger symptoms has been detected. For instance, cross-sectional survey studies and qualitative case studies have shown that IEI-EMF symptoms have been reported by sufferers in response to a wide range of EMF emitting devices and infrastructure including mobile and cordless phones, mobile phone base stations, personal computers, ELF-EMF sources (i.e., power lines, electrical appliances and railroads), Wireless Local Area Networks (W-LAN or 'Wi-Fi"), Magnetic Resonance Imaging (MRIs) and Ultra Violet (UV) radiation from sunlight. In addition, the sources that people report sensitivity to have been found to be either very specific (e.g., they report responding to one source only) or are more general (e.g., they report responding to a range of, or 'all' EMF sources) (Hagström, Auranen, & Ekman, 2013; Hocking, 1998, 2014; Kato & Johansson, 2012; Röösli, Moser, Baldinini, Meier,

& Braun-Fahrländer, 2004; Schüz et al., 2006). Furthermore, some sufferers report experiencing short lasting ‘acute’ symptoms which they allege are in relation to using or being in the vicinity of EMF-emitting devices, while others report experiencing more prolonged and debilitating symptoms which are claimed to be the result of a build-up of exposure from a variety of sources over time (Hocking, 1998, 2014; Röösli et al., 2004).

As well as experiencing considerable physical impairment, individuals suffering from IEI-EMF report a significant degree of social, mental, functional, and financial strain. This is often associated with increased levels of distress, increased health service use, the desire to move away from cities to areas perceived as ‘safer’ or ‘low-EMF’ and being either partly or completely unable to work due to their health issues (Hagström et al., 2013; Johansson, Nordin, Heiden, & Sandström, 2010; Kato & Johansson, 2012; Röösli et al., 2004; Rubin et al., 2010).

Table 1.1: Common symptoms reported by IEI-EMF sufferers

Neurasthenic	Vegetative/Mood	Dermatological
Headaches	Abnormal fatigue	Burning/warmth skin sensations
Dizziness	Depression	
Heavy/pressure sensations in the head	Mood changes	Stinging sensation of the skin
	Stress	
Concentration problems	General sensation of illness	Hot sensations of the head region
Memory problems	Nervousness/anxiety	
Increased sensitivity to noise		
Ear aches		
Tinnitus		
Arrhythmia		
Sore joints		
Back aches		
Nausea		
Sleeping disorders		
Muscle tension		
Muscle weakness		
Limb pain		

Table 1.2 displays the estimated prevalence of IEI-EMF in various countries around the world. These figures demonstrate that the prevalence of the condition is also relatively heterogeneous globally. It is important to note, however, that not all individuals attributing health complaints to EMF exposure identify themselves as 'being EHS' (Kato & Johansson, 2012; Schüz et al., 2006). It is also possible that the difference in prevalence rates are reflective of the differences in the type of questions asked and the time of the survey being administered, rather than an actual difference in prevalence rate. For instance, Hillert, Berglind, Arnetz, and Bellander (2002) asked surveyed

participants to mark factors for which they believed they were hypersensitive or allergic to from a range of 13 environmental stimuli (for example pollen, noise, electromagnetic fields) at a time when concern about EMF in Sweden was mainly focused on the EMF emitted by VDU and powerlines. Conversely, Blettner et al. (2009) conducted a survey which focused specifically on symptoms attributed to RF-EMF and asked participants whether they were worried about health effects of mobile phone base stations in general, and whether they believed that their health was adversely affected by mobile phone base stations. Clearly, these two methodologies differ substantially in terms of their focus and the time at which they were conducted. As such, the figures presented in Table 1.2 should be noted with caution, as they may represent an over- or under-estimate of the prevalence of the condition.

Table 1.2: Estimated prevalence of IEI-EMF

Country	Prevalence (%)	Source
Sweden	1.5	(Hillert et al., 2002)
California	3.2	(Levallois, Neutra, Lee, & Hristova, 2002)
Austria	3.5	(Schröttner & Leitgeb, 2008)
Netherlands	3.5	(Baliatsas et al., 2015)
United Kingdom	4.0	(Eltiti, Wallace, Zougkou, et al., 2007)
Switzerland	5.0	(Schreier, Huss, & Röösli, 2006)
Germany	~10	(Blettner et al., 2009)
Taiwan	13.3	(Tseng, Lin, & Cheng, 2011)

The distressing and debilitating symptoms experienced by those who suffer from IEI-EMF often lead to significant impairments in physical, mental and social wellbeing

(Johansson, Sandström, Heiden, & Nordin, 2010). Clearly, those who experience IEI-EMF are impaired according to the WHO definition of health, and further investigation is required to elucidate the aetiology of their symptoms. Although the characterisation of IEI-EMF through qualitative case studies and survey data generates considerable insight into how the condition affects people's lives, such studies cannot objectively comment on the existence of a causal relationship between EMF and the reported symptoms (Hocking, 1998). Generally, this is because these types of studies do not involve an empirical test of whether exposure to EMF can generate the symptoms, but instead rely heavily on retrospective self-report, which is known to suffer from recall bias (Baliatsas et al., 2015; Vrijheid et al., 2009). Anecdotal reports are also unable to account for the possibility that a psychological phenomenon, such as a nocebo effect, rather than the EMF exposure itself, is responsible for triggering the reported symptoms. As such, these studies cannot provide definitive support for the toxicogenic theory of IEI-EMF.

1.4 The Nocebo Effect

Medical practitioners have long been aware of a seemingly mysterious phenomenon known as a placebo effect. This effect is characterised by a genuine physiological or psychological response to a stimulus that has no inherent powers to produce the observed effect (Stewart-Williams, 2004; Stewart-Williams & Podd, 2004). The classic example of a placebo effect occurs when a patient is prescribed a sugar pill and the patient, unaware that the pill is merely a placebo (and is thus pharmacologically irrelevant to their complaint), makes a full recovery (Stewart-Williams, 2004). Of interest to this thesis, however, is the negative counterpart of the placebo effect, the nocebo effect. The term 'nocebo effect' was first introduced in the 1960's to describe

the genuine adverse physiological and psychological effects of stimuli which have no inherent powers to produce observed effects (Barsky, Saintfort, Rogers, & Borus, 2002; Hahn, 1997; Kennedy, 1961). Importantly, in both placebo and nocebo effects, the exposure or substance administered to a person is not a necessary or sufficient cause of the associated outcome (Hahn, 1997).

In comparison to the placebo effect, much less is known about the factors which contribute to a nocebo response. Generally, this is because inducing a nocebo response is a stressful and anxiety provoking procedure which may lead to a real deterioration in health, thus limiting its potential to be ethically investigated in humans (Enck, Benedetti, & Schedlowski, 2008). The extensive study of placebo effects has, however, revealed the complex interaction between psychological processes, such as expectation and anticipation, and particular neuronal systems that are capable of altering the course of a symptom or disease (Benedetti, Lanotte, Lopiano, & Colloca, 2007). This has led to the formation of a number of theories about the possible factors and neurobiological mechanisms that may contribute to a nocebo effect, though determining which mechanism sufficiently explains the nocebo effect has not yet been adequately clarified. So far, three theories have been proposed to explain the nocebo effect; namely misattribution, learning and expectation (Webster, Weinman, & Rubin, 2016).

The misattribution theory posits that pre-existing symptoms are misattributed to the effects of a new exposure (Webster et al., 2016). While it is common to experience symptoms in everyday life (Kroenke & Price, 1993; Petrie, Faasse, Crichton, & Grey, 2014; Reid, Wessely, Crayford, & Hotopf, 2001), when these symptoms are perceived to occur consistently with an environmental exposure, the potential to mistakenly attribute the symptoms to the exposure source are increased. In respect to IEI-EMF, one

recent qualitative study suggested that people who claim to suffer from IEI-EMF are actually individuals who have pre-existing medically unexplained symptoms (MUS) who are using the notion of sensitivity to EMF as a narrative to help explain their symptoms (and thereby misattribute their symptoms to EMF) in an effort to make their condition more practically and emotionally manageable (Dieudonné, 2016). Some researchers, however, argue that misattributing symptoms to an exposure does not technically constitute a nocebo effect, and that expectations and/or learning are the predominant factors underpinning a nocebo response (Barsky et al., 2002; Colloca & Franklin, 2011). This may be because misattribution can be viewed as one step in the nocebo process, rather than a nocebo effect itself. Indeed, Dieudonné (2016) did not consider the misattribution of MUS to EMF as a nocebo response *per se,* but rather suggested that the misattribution of symptoms occurs independently, and that a nocebo response based on negative expectations or learning may occur at a later stage to further reinforce the original symptom misattribution. In a recent systematic review, however, Webster et al. (2016) showed that a number of studies investigating the nocebo response had found that participants who suffered from conditions with symptoms similar to those being induced by an inert substance predicted increased symptom reporting, demonstrating that the misattribution mechanism of the nocebo effect may be plausible in some instances. While misattribution may be an underlying factor in the presentation of some IEI-EMF cases, it is a difficult factor to quantify through empirical research, as the original symptom misattribution could only be understood through qualitative interviews or epidemiological survey studies, which are often conducted retrospectively and may be influenced by recall bias. Yet, irrespective of the debate surrounding whether misattribution technically constitutes a nocebo effect, it is clear that the concept

of conscious or subconscious symptom misattribution is an important element in the remaining two nocebo effect theories.

The learning theory suggests that nocebo effects are elicited through classical conditioning (Barsky et al., 2002; Webster et al., 2016). For example, a person may experience side effects to a prescribed medication, but not because of a pharmacological reaction, but rather because they have experienced side effects to drugs with, for instance, a similar shape, colour, smell or taste, in the past (Barsky et al., 2002). In this way, the physical properties of the medication have acquired the capacity to elicit a physiological change as a result of classical conditioning, through either a conscious or subconscious mechanism (Webster et al., 2016). In regards to IEI-EMF, it is possible that the coincidental experience of a common symptom when a person comes into contact with a certain EMF emitting source on a number of occasions forms an association in the brain such that when that (or a similar) device is seen, it automatically triggers the associated response (i.e. a symptom). In this way, an individual will have become conditioned to experience a symptom when they come into contact with that specific or similar looking EMF emitting sources or if they come to believe that they are in an environment where they are being exposed to EMF. Misattribution may then play a role when a person attributes the cause of their symptoms (without any objective evidence) to the EMF emitting source as a way of labelling what the conditioning has demonstrated.

The expectation theory proposes that negative expectations of adverse symptoms or reactions and the associated emotional states associated with such expectations elicit the adverse symptoms or reactions in the expectant person (Hahn, 1997). The possible psychological mechanisms thought to underlie the formation of the negative

expectations that may drive nocebo responses are anticipation and information about negative outcomes, prior experience of negative therapeutic outcomes and the observation of other patient's negative outcomes (Colloca & Franklin, 2011). For instance, if a person is given information that an interaction with a (neutral) stimulus may have negative side effects (for example a headache), the negative expectation generated by this information may make an individual increasingly likely to notice or attend to either existing or new symptoms, and then attribute these symptoms to the stimulus (Barsky et al., 2002). In addition to this, the emotional states associated with negative expectations may also induce that particular negative emotional state. For example, the expectation of anxiety is in itself anxiety provoking, and therefore directly elicits the negative effect that was expected (Webster et al., 2016). In many instances, explicit suggestions about the effects of an exposure have been shown to be a key contributing factor in the generation of negative expectations that result in nocebo effects (Barsky et al., 2002; Benedetti et al., 2007; Webster et al., 2016). In terms of IEI-EMF, an example of this may be that the communication of information about potential adverse health effects, either through news media stories or through the precautionary information communicated by governments and health agencies, constitutes an explicit suggestion which could then be responsible for the formation of negative expectations and a subsequent nocebo effect. Yet while many studies have shown that explicit suggestions about the effects of EMF can negatively influence people's beliefs about EMF exposure (Barnett, Timotijevic, Shepherd, & Senior, 2007; Köteles, Tarján, & Berkes, 2016; Nielsen et al., 2010; Wiedemann, Boerner, & Repacholi, 2014; Wiedemann et al., 2013; Wiedemann & Schütz, 2005; Wiedemann, Thalmann, Grutsch, & Schütz, 2006; Witthöft et al., 2017), whether the negative beliefs

induced by such information can result in a symptomatic nocebo response following a perceived exposure to EMF has not been sufficiently established.

Recent studies investigating the potential factors and neurobiological mechanisms underlying placebo and nocebo effects mostly stem from the investigation of pain processing in healthy participants. Despite the ongoing debate about which theory can better explain placebo and nocebo effects, these studies have confirmed that mental processes, mediated by expectations and learning, have the ability to modify the experience of pain. Further, these changes in pain processing have been able to be objectively measured and associated with certain brain regions and psychopharmacological processes using neuroimaging methods (Colloca & Franklin, 2011). In particular, experimental studies have shown that negative verbal suggestions following the administration of inert substances can induce anticipatory anxiety about the impending pain increase which, in turn, triggers the activation of two different and independent biochemical pathways (Benedetti, Amanzio, Vighetti, & Asteggiano, 2006). One pathway involves the activation of cholecystokinin (CCK), a neuropeptide that has been found to play a crucial role in a number of psychological and physiological functions (Hebb, Poulin, Roach, Zacharko, & Drolet, 2005), including as a neuromodulator in the facilitation of the experience of pain (Benedetti et al., 2007). The other pathway involves the activation of the hypothalamic-pituitary-adrenal axis (HPA), a pathway implicated in the release of cortisol and the experience of anxiety (Benedetti et al., 2006; Benedetti et al., 2007). In addition to this, Landgrebe, Barta, et al. (2008) found that a sham EMF exposure administered to people with IEI-EMF resulted in symptom experiences that correlated with alterations in neural activity in the anterior cingulate cortex and the insula; brain regions that have been associated with

processing experimentally induced pain. The observation that sham exposures can elicit symptoms in IEI-EMF participants is often used to suggest that the symptoms experienced are the result of a nocebo effect (Rubin et al., 2010), and the findings of Landgrebe, Barta, et al. (2008) provides strong evidence of the neurobiological mechanism potentially underlying this response.

Although there appears to be relatively strong evidence for the ability of psychological and neurobiological processes to influence symptom perception via nocebo effects, whether this is the case for the symptoms experienced by IEI-EMF sufferer's remains highly contentious. Although Dieudonné (2016) concluded that IEI-EMF does not originate from a nocebo response, it is important to note that Dieudonné (2016) did not test the cause of the participant's symptoms, but rather, retrospectively asked participants about their beliefs regarding the cause of their symptoms using qualitative methods. Retrospective self-reports, especially in relation to the aetiology of symptoms associated with EMF exposure, are known to suffer from recall bias (Baliatsas et al., 2015; Vrijheid et al., 2009), and as such, the conclusions reached by Dieudonné (2016) cannot be used to comment on the possibility that IEI-EMF is associated with a nocebo effect. As such, only empirical studies, which test for a quantifiable relationship between symptoms and exposure, can be used to establish whether IEI-EMF can be better explained by a toxicogenic or psychogenic theory.

1.5 Empirical Studies Investigating Reported Sensitivity to EMF

Observational epidemiology studies and experimental laboratory studies are the two main scientific approaches that have been used to investigate whether humans are sensitive to EMF exposure within the established public safety guidelines (for e.g. ICNIRP, 1998). Epidemiological studies attempt to find an association between

symptom reports and exposure, by estimating the amount of exposure individuals (who may or may not believe they are sensitive to EMF exposure) are receiving in their daily lives in relation to the type, frequency and severity of non-specific symptoms that they experience. Laboratory studies, on the other hand, attempt to deliberately trigger symptom responses to specific types and strengths of EMF exposure in a controlled setting, and generally focus specifically on individuals who report experiencing IEI-EMF. Both of these approaches have a number of strengths and limitations, all of which must be taken into account when assessing our current understanding of IEI-EMF.

Although some researchers and IEI-EMF advocacy groups have suggested that studies utilising 'subjective' measures of symptoms are scientifically unreliable (Leszcynski, 2018), there is also no reliable evidence showing that IEI-EMF sufferers experience any consistent physiological responses as a result of exposure to EMF (Rubin, Hillert, Nieto-Hernandez, van Rongen, & Oftedal, 2011). As no consistent pattern of objectively measurable changes resulting from EMF exposure can be used to characterise or diagnose IEI-EMF, epidemiological and laboratory studies must rely on participant self-report. The use of self-report measures in IEI-EMF studies is, however, appropriate because IEI-EMF sufferers report subjective changes which they associate with perceived exposure to EMF, they do not rely on objective data of disease from situations where exposure is and is not present and then conclude that one causes the other.

While sensitivity to a whole range of devices which emit different types of EMF has been reported (as discussed in section 1.3), much of the recent literature has been focused on investigating the potential adverse effects of exposure to RF-EMF emitted by wireless communication technologies and infrastructure, as these have become

increasingly ubiquitous and have been the cause of most concern in recent times. In line with this, and in an effort to provide focus for the present doctoral research, the remainder of this thesis will focus on the possible relationship between RF-EMF exposure and IEI-EMF.

1.5.1 Epidemiological studies

Epidemiology is the study of the distribution and causes of human disease in free living populations; that is, the study of disease in populations that are not under the constraints of a laboratory environment. Epidemiological studies either measure the frequency of diseases or other health related characteristics in populations in order to observe whether such frequencies vary with other characteristics, or they assess whether associations exist between possible causative factors and health outcomes (Elwood, 2017). As epidemiological studies require large sample sizes to account for error variance, studies investigating the possible causal association between RF-EMF emissions and symptoms have mostly recruited participants from the general population (and may or may not have included IEI-EMF participants). These epidemiological studies typically examine whether there is an association between mobile phone base stations and symptoms. Although the lack of focus on IEI-EMF participants may be viewed as a major limitation for establishing whether symptoms attributed to EMF are associated with exposure, one of the major benefits of these studies (over laboratory studies) is that they allow for the investigation of longer exposure periods and symptom outcomes in large samples under normal living conditions, and thus may be useful for providing evidence (if any) of a potential association between EMF exposure and symptoms more generally.

Over the past two decades, a number of epidemiological studies have been conducted, with varying methods and results. Notably, two studies reported significant associations between symptoms and exposure to EMF. Hutter, Moshammer, Wallner, and Kundi (2006) investigated the relation between RF-EMF exposure and several measures of cognitive performance, well-being and sleep quality, in individuals living near 10 selected base stations. Spot measurements in the bedrooms of participants and a calculation of distance to the mobile phone base station were used to estimate exposure rates. Although it was found that exposure rates in the participant's homes were far below the recommended standards, the results showed a significant relationship between exposure level and headache score. Symptom ratings were also found to be higher in participants expressing concerns about health effects from the base station. Similarly, Abdel-Rassoul et al. (2007), using a cross-sectional study, reported a significantly higher prevalence of reported neuropsychiatric complaints, including headache, in people living or working near a base station than in matched controls. However, the measurement of RF-EMF exposure was made 4 years before the study was conducted and only in one area where some of the experimental participants were located, and no attempt was made to measure exposure in the control group. Therefore, the results of this study are unable to comment on the relationship between exposure and symptoms.

While there has been some limited evidence of an association between symptom reports and exposure, the majority of epidemiology studies assessing the potential association between symptom reports and exposure have failed to find such an association (Röösli et al., 2010). For instance, Thomas et al. (2008) did not find any significant association between exposure and chronic or acute symptoms in a sample of adults who were

required to wear personal dosimeters for a 24 hour period. In addition to this, in a large cross-sectional study which used dosimeters to measure RF-EMF in the bedrooms of participants, Berg-Beckhoff et al. (2009) found no association between RF-EMF exposure and a range of health outcomes including sleep quality, headache, psychosomatic complaints, mental and physical health or chronic stress. Moreover, Baliatsas et al. (2015) did not find an association between everyday life RF-EMF exposure, and non-specific symptoms or sleep quality.

Although epidemiological studies attempt to bridge the gap between the anecdotal reports of symptoms in response to EMF exposure and the controlled laboratory studies investigating the causal role of EMF exposure in producing symptoms, these studies face serious methodological limitations, especially in regards to exposure characterisation. Many studies rely on the historical reconstruction of exposure history or the individual's perceived distance to an exposure source to estimate how much exposure a person has received, but this is prone to recall bias and often does not take into account the variety of near and far field sources to which people are exposed (Baliatsas et al., 2015). In a systematic review of this field, Röösli et al. (2010) found that epidemiological studies with crude exposure assessments show health effects, while studies with more sophisticated exposure measurements rarely indicate an association. The more sophisticated methods of exposure characterisation include the use of spot measurements or personal exposure meters. But these again are limited, in that spot measurements can only provide information about exposure at specific locations and at specific times, while personal exposure meters can be influenced by a number of factors; including calibration, body shielding and bias associated with the alteration of behaviour if exposure levels become known to the participant (Baliatsas et al., 2015).

Consequently, while epidemiological studies vary substantially in both quality and outcomes, the limitations associated with such studies make it difficult to determine whether exposure to EMF is associated with non-specific symptoms or IEI-EMF itself. In support of this, the World Health Organisation has recommended that the aetiology of IEI conditions be determined via the results of double-blind, sham-controlled, provocation studies, where the ability to discriminate between active and sham environmental exposures suggests a toxicogenic mechanism, and inability to discriminate suggests a psychogenic mechanism (International Programme on Chemical Safety/World Health Organization (IPCS/WHO), 1996).

1.5.2 Laboratory studies

Human laboratory studies, often termed 'provocation studies', offer a powerful method for testing whether the presence of EMF is sufficient to trigger symptoms in humans. Provocation studies typically involve volunteers being exposed to active and sham EMF under controlled conditions, preferably in a double-blind testing protocol. Typically, these studies test whether people who report suffering from IEI-EMF are better at detecting EMF than people without the condition (or at greater than chance levels), and whether sufferers of IEI-EMF respond to the presence of EMF with increased symptoms compared to sham exposures. Over the past two decades, a number of provocation studies using varying methodologies simulating either mobile phone base station like exposures or mobile phone handset like exposure have been conducted. Provocation studies utilising mobile phone base station like exposures attempt to simulate the whole body exposures typically experienced in the vicinity of mobile phone base stations, whilst studies utilising mobile phone handset like exposures try to simulate the near field acute exposures typically experienced when using a mobile

phone handset in an active talking mode. Generally, provocation studies have failed to provide evidence to support the notion that IEI-EMF is a condition directly associated with the presence of EMF (Rubin et al., 2005; Rubin et al., 2010).

One of the first studies conducted investigating the effect of a base station-like exposure on well-being in a controlled laboratory setting reported that EMF exposure reduced well-being scores in both IEI-EMF and control participants (Zwamborn, Vossen, van Leersum, Ouwens, & Makel, 2003). However, in a follow up study using an improved methodology and a larger sample size, Regel et al. (2006) failed to find an effect of exposure on symptom scores or well-being in either IEI-EMF participants or healthy controls, and did not find evidence to suggest that participants could discriminate between the active and sham conditions. Interestingly, Eltiti, Wallace, Ridgewell, et al. (2007) found that IEI-EMF participants reported an increase in symptoms in an initial non-blind active trial, but that when the study was blinded, the increase in symptoms in the active trial was not present. The results did show a significant increase in reports of arousal during exposure compared to sham, however further analysis revealed that this was likely due to a lack of appropriate randomisation and counterbalancing. No other effect of exposure in either the IEI-EMF or healthy control group was found. Similarly, Wallace et al. (2012) found that IEI-EMF participants reported an increase in symptoms in an active base station exposure condition compared to sham in an initial non-blinded trial, but found no differences in well-being, symptom levels or discrimination in later double-blind trials. In addition to this, Furubayashi et al. (2009) did not find any effect of either long or intermittent exposures on measures of mood, discomfort or the ability to discriminate between active and sham conditions during a double-blind protocol, in either IEI-EMF participants or healthy controls.

Studies investigating mobile phone handset-like exposures and IEI-EMF have also generally failed to find any relationship. For example, Wilén, Johansson, Sandström, Kalezic, and Lyskov (2006) found no significant effect of RF-EMF exposure on a range of physiological or cognitive parameters in either participants who reported mobile phone exposure-related symptoms or matched healthy controls. Similarly, Rubin, Hahn, Everitt, Cleare, and Wessely (2006) found that while IEI-EMF participants reported greater symptom severity than matched controls, IEI-EMF individuals were unable to reliably discriminate between the exposure conditions, and the increase in symptoms was not dependent on the exposure condition, with the sham condition being sufficient to trigger symptoms. Corresponding to this, Oftedal, Straume, Johnsson and Stovner (2007) found that the sham condition also triggered symptoms in IEI-EMF participants, but did not find any evidence to suggest that either the IEI-EMF or matched control groups could accurately detect when they were being actively exposed. In addition, Nam et al. (2009) did not find any effect of exposure on subjective symptoms or a range of physiological parameters, and IEI-EMF participants were no better at detecting the presence of RF-EMF than controls. Moreover, after a subset of participants took part in a sleep component of a provocation study, no effect of exposure on self-reported sleepiness, fatigue or arousal following sleep was found (Lowden et al., 2011). Finally, in a recent study assessing whether IEI-EMF participants were able to correctly identify whether they were being exposed to an individually relevant signal (exposures which each individual participant reported reacting to) or sham condition, van Moorselaar et al. (2017) found that while participants were able verify that they were being exposed in an initial non-blinded exposure, subsequent double-blind testing revealed that they were not able to detect the active from sham exposure at better than chance levels.

Of the few studies that have found significant effects in the IEI-EMF group, methodological problems have often confounded the results, including the failure to account for multiple significance tests, inadequate counterbalancing or the possible de-blinding of participants or researchers (Rubin et al., 2010). These confounds appear to explain the failure to replicate such results in larger samples. For instance, although Hillert et al. (2008) found that neither the IEI-EMF or control group could detect RF-EMF exposure better than chance, a significant number of participants reported headache symptoms more commonly after RF-EMF exposure than sham. However, the effect was due to a rise in headache reporting in the control group and the statistical analyses were not corrected for multiple tests. In addition to this, one study reported that two IEI-EMF participants were able to accurately detect an active exposure condition at highly significant rates (Kwon, Koivisto, Laine, & Hamalainen, 2008), however on a subsequent retest six months later, the same two participants were unable to replicate their results, suggesting that their initial performance was not related to a bioelectromagnetic phenomenon (given that the exposures were the same in both situations). Moreover, Nieto-Hernandez et al. (2011) reported increased ratings of headache and difficulty in concentration in IEI-EMF participants and increased levels of headache and fatigue in non-IEI-EMF participants following exposure to a continuous wave signal. Concentration difficulties were again reported for the IEI-EMF participants after exposure to a pulsed signal. However, after appropriate adjustment for multiple comparisons, these results were not significant. Furthermore, McCarty et al. (2011) reported that an IEI-EMF participant's symptoms were associated with exposure (specifically, they were related to the field transitions created when switching from active to sham conditions), but this did not account for chance through statistics (and

thus could not be said to be less likely than chance), and the results were not replicated (Rubin, Cleare, & Wessely, 2011).

Like epidemiological studies, provocation studies are challenged by several methodological limitations, some of which could potentially explain the inability of these studies to find an effect of exposure. One of these limitations relates to recruitment. Little is known about whether subsets of the condition exist and so it is conceivable that the samples tested may have included a combination of both individuals who are sensitive to EMF or to types of EMF not used in the testing protocol, and others who may suffer from unrelated conditions. This could result in a large amount of noise being added to the data, which would reduce statistical power and mask any real effects. Another issue is determining whether the environment that provocation studies are conducted in reflects the environment in which IEI-EMF individuals report symptoms. Often, studies are conducted in laboratories with specialised chambers which block out background electromagnetic fields. While it is generally thought that this should increase the chances of detecting an effect, it may potentially remove important synergistic elements present in the environment. Anxiety, which may be heightened due to participating in a laboratory experiment, may also mask any significant effects. Participants may have also encountered other EMF exposures on the way to the experimental session which inadvertently trigger symptoms (Rubin, Hahn, et al., 2006; Rubin et al., 2010). This again, would mask any potential significant effects from being discovered. Criticism has also been raised concerning the relevance of simulated RF-EMF signals used in many of these studies to participant's reported symptoms (Panagopoulos, Johansson, & Carlo, 2015). Yet, the evidence so far suggests that symptoms are triggered during provocation studies, regardless of

similarity of the exposure (and irrespective of the exposure status), which suggests that this is not an important confound. Likewise, although it has been suggested that symptom onset and offsets have not been matched in experimental studies, there is no evidence that this has confounded previous research either. Finally, while a number of studies have consistently demonstrated that the symptoms experienced by IEI-EMF sufferers are likely the result of a nocebo effect, few studies have investigated the underlying mechanisms which may contribute to such a response. It is important to note, however, that although these methodological issues are often used by IEI-EMF advocacy groups to argue that it is premature to rule out the role of EMF in the presentation of IEI-EMF, there is no evidence to demonstrate that these issues have influenced the results of previous studies.

1.6 Thesis Rationale and Aim

Clearly, IEI-EMF is a complex condition which is not adequately understood by current scientific models. In stark contrast to the anecdotal reports of sensitivity to EMF (see section 1.3), a considerable number of epidemiological and laboratory studies have failed to find evidence for a relationship between EMF exposure and IEI-EMF (see section 1.5 for review). While the majority of IEI-EMF sufferers believe that the condition is caused by exposure to EMF, much of the existing empirical literature indicates that the condition is more closely associated with a nocebo effect, where conscious or subconscious symptom expectation following a perceived exposure to EMF leads to the formation or the presentation of symptoms (Hillert et al., 2008; Landgrebe, Frick, et al., 2008; Oftedal et al., 2007; Rubin et al., 2010). While the discrepancy between the anecdotal reports of sensitivity to EMF and the scientific literature has instigated a highly contentious aetiological debate, sufferers have been left

to experience distressing and debilitating symptoms with no reliable, evidence-based treatment options or support. Considering that those who suffer from IEI-EMF could not be considered healthy according to the WHO definition of health (World Health Organisation, 1946), further research is required to clearly determine the aetiology of their symptoms.

The fundamental issue at the centre of the aetiological debate is whether the associated symptoms can be explained by a toxicogenic or psychogenic theory. The toxicogenic theory of IEI-EMF proposes that the presentation of adverse symptoms is the result of an intolerance or susceptibility to exposure to low levels of EMF, which then affect the body via an as-yet-unrecognised bioelectromagnetic pathway (Rubin et al., 2010; Staudenmayer et al., 2003a). Alternatively, the psychogenic theory of IEI-EMF suggests that the condition can be explained by an individual's overvalued belief of harm, a belief which is shaped by psychological, psychosocial and psychophysiological processes (Staudenmayer et al., 2003b) and which culminates in the presentation of adverse symptoms via a nocebo response. However, due to a number of potential methodological limitations, and a general lack of understanding of the possible mechanisms underlying toxicogenic and psychogenic explanations of IEI-EMF, it is difficult to conclusively determine the aetiology of IEI-EMF on the basis of the currently available evidence.

The overall aim of the present doctoral research is to clarify the determinants of IEI-EMF by investigating whether toxicogenic or psychogenic processes can explain the symptoms reported by IEI-EMF sufferers. Specifically, the research contained in this thesis encompasses three human provocation studies designed with methodological improvements to investigate the possible adverse health effects associated with

exposure to EMF and to examine the potential psychological mechanisms which may underlie IEI-EMF sufferers' perception that they are sensitive to EMF exposure.

This thesis is presented as a collection of manuscripts prepared for publication in accord with the requirements of the University of Wollongong (Thesis by Compilation). Each chapter represents a manuscript written for a specific journal with a defined audience. Each chapter begins with a Chapter Foreword which outlines the rationale, aims and significance of each manuscript, and establishes a link between each manuscript and the thesis aims more generally. The structure of the abstract and headings within each paper is consistent with the style used by the journal for which it is written. Chapter 2 has been published in the *International Journal for Radiation Biology*, Chapter 3 has been published in *Bioelectromagnetics* and Chapter 4 has been published in *Environmental Research*. While each journal requires a specific referencing style, for consistency all chapters in this thesis are referenced in the current style of the American Psychological Association (APA 6th edition).

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2. CHAPTER 2: PULSE MODULATED RADIOFREQUENCY EXPOSURE INFLUENCES COGNITIVE PERFORMANCE

2.1 Chapter Foreword

Despite decades of research, IEI-EMF provocation studies have generally failed to provide evidence that the symptoms reported by IEI-EMF sufferers are associated with EMF exposure. While many IEI-EMF sufferers and advocacy groups have raised concerns that subjective measures of belief of exposure and self-reported symptoms in provocation studies are 'scientifically unreliable', objective tests of sensitivity to EMF are currently lacking, as there have been no consistent reports of adverse effects of EMF exposure on endpoints that are relevant to health. Although a number of studies have consistently reported physiological effects of exposure to RF-EMF on the brain's electrical activity (see section 1.2 for review), these effects may not necessarily reflect an adverse health outcome, as many of these (and other) studies assessing various aspects of cognitive and behavioural functioning have found inconsistent, but mostly null results. Given the close relationship between the brain's electrical activity and cognition, and given that IEI-EMF sufferers often report memory and concentration difficulties as symptoms which they attribute to EMF exposure, this may offer a potentially useful avenue for research.

It is possible that a number of methodological issues have limited the ability of previous studies to detect effects of RF-EMF exposure on cognitive performance. These issues include the use of poor exposure protocols and experimental designs, inadequate sample sizes and cognitive performance measures which may not have adequately accounted for confounding variables. Given these methodological limitations, it remains unclear whether exposure to RF-EMF can affect cognition. Yet, if clear adverse effects of RF-

EMF exposure on cognitive performance endpoints can be established, then this may offer a starting point for determining the most sensitive objective endpoints to test IEI-EMF participants.

In an attempt to improve methodology and identify possible impairments on measures of cognitive performance, the study presented in this chapter aimed to determine whether exposure to mobile phone-like RF-EMF exposure could influence cognitive performance, and whether it does so in a dose-dependent manner. Although this does not directly test the claim that IEI-EMF symptoms are associated with EMF exposure, it is a useful first step for developing a potentially sensitive endpoint that may then be used to test IEI-EMF participants, while also overcoming criticisms that empirical studies investigating IEI-EMF need to utilise objective tests of sensitivity. This paper has been published in the *International Journal of Radiation Biology*.

Verrender, A., Loughran, S.P., Dalecki, A., McKenzie, R., & Croft, R.J (2016). Pulse modulated radiofrequency exposure influences cognitive performance. *International Journal of Radiation Biology. 92,* 603-610.

2.2 Abstract

Purpose: To investigate whether exposure to pulse modulated radiofrequency (PM RF) influences human cognitive performance, and whether it does so in a dose dependent manner.

Materials and Methods: Thirty six healthy adults participated in a randomised, double blind, counterbalanced provocation study. Cognitive performance was assessed using a visual discrimination task and a modified Sternberg working memory task, which were calibrated to individual performance levels in a preliminary testing session. An sXh920 planar exposure system was used to generate a 920 MHz GSM-like signal, providing three conditions (peak-spatial SAR averaged over 10g) of 0 W/kg (Sham), 1 W/kg (Low RF) and 2 W/kg (High RF).

Results: A significant decrease in reaction time (RT) in the Sternberg working memory task was found during exposure compared to Sham. This effect was not dose dependent.

Conclusions: PM RF exposure was shown to influence cognitive performance in a working memory task. While the majority of the literature has not found effects of PM RF exposure on cognitive performance, it is possible that the methodological improvements employed in the present study increased sensitivity, and thus the ability to detect potential effects.

2.3 Introduction

Over the past two decades, the increasingly widespread use of mobile phones has generated growing concern about potential adverse effects that the radiofrequency electromagnetic fields (RF-EMF) emitted by these devices could have on human health. While a number of independent reviews have concluded that there are no substantiated health effects associated with exposure to mobile phone RF-EMF (Health Canada, 2015; Health Council of the Netherlands, 2009; SCENIHR (Scientific Committee on Emerging and Newly Identified Health Risks), 2009; World Health Organisation, 2014), there is evidence which indicates that exposure can influence the brain's electrical activity. Specifically, it has been consistently shown that pulse modulated RF (PM RF) affects spontaneous resting alpha (8-12 Hz) (Croft et al., 2008; Croft et al., 2010; Curcio et al., 2005; Leung et al., 2011; Perentos, Croft, McKenzie, Cvetkovic, & Cosic, 2007; Regel, Gottselig, et al., 2007) and sleep spindle activity (approximately 11-15 Hz) in non-rapid eye movement sleep (Huber et al., 2000; Huber et al., 2002; Loughran et al., 2005; Regel, Tinguely, et al., 2007; Schmid et al., 2012).

However, the functional consequence of this change in EEG activity remains unclear, as studies assessing aspects of cognitive and behavioural functioning during and following exposure to PM RF have produced contradictory, but mostly null results (Barth, Ponocny, Gnambs, & Winker, 2012; Valentini, Ferrara, Presaghi, De Gennaro, & Curcio, 2010).

One measure of cognitive performance which has yielded inconsistent results is working memory performance, which has been primarily assessed using the N-back task. Three studies have reported decreases in reaction time (Koivisto, Krause, Revonsuo, Laine, & Hämäläinen, 2000; Regel, Gottselig, et al., 2007; Regel, Tinguely,

et al., 2007) and one study reported an improvement in accuracy (Regel, Tinguely, et al., 2007). The majority of studies using this task, however, have not found any effects of PM RF exposure on performance (Haarala et al., 2003; Haarala et al., 2004; Haarala et al., 2007; Krause, Pesonen, Bjornberg, & Hamalainen, 2007; Leung et al., 2011).

The difficulty in establishing a firm conclusion as to whether PM RF influences cognitive performance may be attributed to a number of methodological constraints. For example, the variation in methods between research groups makes it difficult to compare or verify previous results, while poor exposure protocols and experimental designs, inadequate sample sizes and a lack of reliable cognitive performance measures have limited the potential for finding an effect (Regel & Achermann, 2011). Several other issues may have also contributed to the mixed findings. For instance individual differences in cognitive performance have not been adequately accounted for in previous studies. It is also possible that the N-back task is not sensitive enough to adequately detect changes in cognitive performance as a result of PM RF exposure (Regel & Achermann, 2011) as it has been shown that this task can be significantly influenced by learning effects (Haarala et al., 2005; Haarala et al., 2004; Regel, Gottselig, et al., 2007; Regel, Tinguely, et al., 2007). Furthermore, as the only known interaction between RF-EMF exposure and the body is via heating (Adair & Black, 2003), thermally induced variability may have also influenced the results. In addition, the localised, intermittent exposure protocols utilised by some research groups may not have been sufficiently powerful to produce an effect (Boutry et al., 2008). These factors have the potential to introduce large amounts of error variance, which may have masked any potential real effects.

In order to overcome these issues, the present study has been designed with several methodological improvements to examine whether exposure to PM RF influences cognitive performance in a dose dependent manner. In particular, a visual discrimination task and a modified Sternberg working memory task (Sternberg, 1966) were employed, both of which were calibrated to individual performance levels in a preliminary testing session. The study also utilised a planar patch antenna system to ensure that there was a consistent, uniform exposure across the target hemisphere. Thermally induced variability was reduced by clamping skin temperature to a thermo-neutral level. In addition to these improvements, the cognitive performance data was treated using an index of the participants' response sensitivity and bias, as adapted from signal detection theory (Stanislaw & Todorov, 1999). This treatment takes into account how well a participant can discriminate between trials (sensitivity) and the participant's general tendency to respond with a 'yes' or 'no' button press (bias), giving a better indication of task accuracy and minimising Type I and Type II error. Further to the double-blind, counterbalanced, sham-controlled design; these improvements were implemented to increase sensitivity, and thus, the possibility of finding potential effects. The results of the present study are part of a larger study investigating the effect of PM RF exposure on the electroencephalogram and thermoregulatory processes.

2.4 Materials and Methods

2.4.1 Participants

Forty-three participants were recruited through advertisements and word of mouth. Seven participants failed to attend all testing sessions, leaving thirty-six participants (half male) aged 18 – 52 years (M = 24.44; SD = 6.27). To be included in the study, participants were required to be between 18 and 55 years of age, be right handed and report being of good health. Participants were excluded from the study if they reported having a current illness or medical condition, or having used illicit substances within the 7-day period prior to the study. Suitable participants were required to attend the Illawarra Health and Medical Research Institute, University of Wollongong, for four mutually convenient testing sessions, at the same time of day and separated by a period of at least 7 days. The study was approved by the Human Research Ethics Committee (University of Wollongong: HE13/146), and written, informed consent was obtained from all participants. All participants were instructed to abstain from alcohol for at least 8 hr before the commencement of a testing session, abstain from caffeine for at least 1 hour before a testing session, and to not use their mobile phone for at least 2 hr before the beginning of a testing session. All participants were compensated a total of $200 for their involvement in the study.

2.4.2 Radiofrequency exposure

An sXh920 planar exposure system (IT'IS Foundation, Zurich, Switzerland) was used to generate a 920 MHz GSM-like signal (as emitted by a mobile phone handset in active mode while transmitting voice). The signal included the basic GSM frequency components (8.33, 216.6, 1733 Hz, including corresponding harmonics; crest factor = 8.3). Two RF antennas placed on wooden pillars were positioned 42 mm vertically

above the ear canal at a distance of 115 mm from the head (Huber et al., 2002; Huber et al., 2005; Loughran et al., 2013). The RF exposure of the sXh920 system has been fully characterised and was calibrated to provide a peak-spatial SAR averaged over 10g of 0 W/kg, 1 W/kg and 2 W/kg, for the Sham, Low and High exposure conditions respectively (for full dosimetric data see Murbach, Christopoulou, Crespo-Valero, Achermann, and Kuster (2012)). These exposures were within the Australian general public RF exposure limits (ARPANSA RPS3). The system was controlled electronically using defined participant numbers. The fully randomised and counterbalanced exposure conditions were assigned to each participant and pre-programmed by a researcher not involved in the collection of data (RC) to ensure that double-blinding was maintained. An inbuilt failsafe mechanism ensured RF levels did not exceed RPS3 levels. Only the left hemisphere was exposed to RF, and brown noise was used to mask any sounds made by the exposure system in order to ensure that participants were not aware of the exposure condition. At the completion of each experimental testing session, participants were asked whether they were aware of the exposure status and the side of exposure via a pen and paper Likert scale (1 = 'left', 2 = 'right', 3 = 'both', 4 = 'no', 5 = 'don't know') and an open ended question asking for further details about their ability to perceive the exposure ('If yes, how did you perceive the field?').

2.4.3 Design

A double-blind, counterbalanced, cross-over design was employed. Following a preliminary calibration session, each participant's cognitive performance was tested under three conditions (Sham, Low and High RF) during exposure (block 1) and following exposure (block 2) over three separate sessions separated by at least seven days. RF exposure was emitted to the left hemisphere only, and participants were not

made aware of which antenna was emitting. Order of exposure was counterbalanced across participants and randomly assigned.

2.4.4 Cognitive Performance Tasks

Two tasks were used to assess cognitive performance. These tasks were calibrated to each participant's individual level of performance in a preliminary testing session and sufficiently long practice blocks were utilised prior to each experimental session to reduce learning, floor and ceiling effects. Behavioural outcomes were measured as the number of correct hits, correct rejections, false alarms and false rejections, as well as participant's reaction time (RT) to correct responses.

2.4.4.1 Visual discrimination task. Visual perception was assessed using a visual discrimination task. A series of crossed white lines were presented in the centre of a computer monitor with a black background for 200ms each. Between each stimulus presentation, a mask appeared for 500ms, followed by a blank screen for 300ms. Figure 2.1 presents an example of the stimuli and mask used in the visual discrimination task.

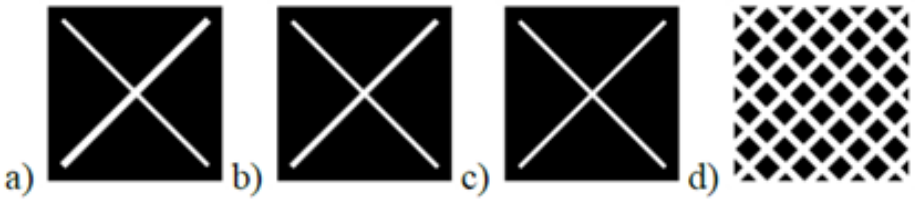

Figure 2.1: Examples of the stimuli used in the visual discrimination task. (a) Is an example of an easy target stimulus, (b) is an example of a difficult target stimulus (c) is a non-target stimulus and (d) is the mask used between stimulus presentations.

For each stimulus presentation, participants were asked to respond as quickly and accurately as possible via a 'yes' button press with the right thumb if they thought that the lines differed in thickness (target stimulus), or not to respond if they thought that the lines did not differ in thickness (non-target stimulus). A total of 180 stimuli were presented (half targets). The frequency and presentation of the target stimuli were pseudo-randomised across trials and balanced between blocks.

Task difficulty was manipulated by increasing the magnitude of thickness between the two lines in target stimuli. The greater the difference between the two lines, the easier the trial. In target stimuli, one line was kept at a constant thickness of 1.5 mm while the second line was manipulated in increments of 0.05 mm. In non-target stimuli, both lines were kept constant at 1.5 mm.

2.4.4.2 Sternberg working memory task. Working memory performance was assessed using a modified Sternberg working memory task (Sternberg, 1966). The task involved memorising a stimulus set, maintaining that stimulus set in memory during a 3 second retention period, and recalling whether a subsequent probe stimulus was in the original memory set. The memory sets ranged in difficulty level from 6 to 15 letters.

The memory sets were constructed from a pool of 21 consonants (B, C, D, F, G, H, J, K, L, M, N, P, Q, R, S, T, V, W, X, Y and Z) presented simultaneously in a horizontal arrangement over two lines. The letters were 66pt, capitalised, black and in bold Calibri font and presented in a centred 6.24 x 25.40 cm grey box on a black background. Each letter was spaced 2 cm apart.

Figure 2.2 outlines the Sternberg working memory task design and progression for each trial. For each trial, participants were asked to respond as quickly and accurately as

possible with a button press using the right thumb if the probe consonant appeared in the preceding memory set (target), or to not respond if the probe consonant was not presented in the memory set (non-target). Maximum response time was set at 2 seconds. 40 memory sets were presented (half targets). The frequency and presentation of the probe stimuli were pseudo-randomised across trials and balanced between blocks. The position of the target probe stimuli in the memory set was also pseudo-randomised across trials.

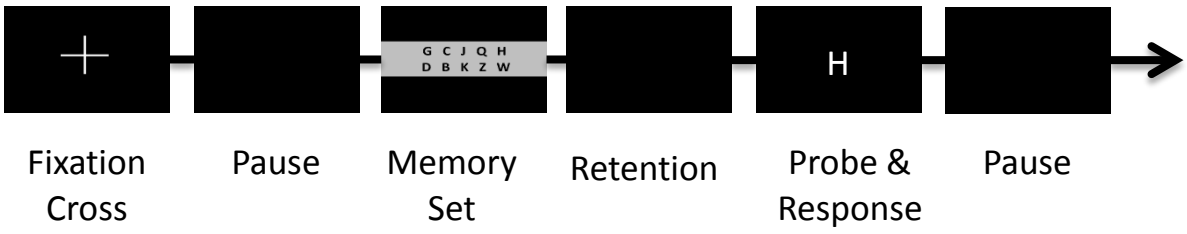

Figure 2.2: The modified Sternberg working memory task design and progression. A fixation cross (800 ms) was followed by a 1000 ms pause. The memory set was then presented (4000 ms) and was followed by a blank screen retention period (3000 ms). Following this, a probe stimulus appeared (2000 ms) during which time participants had to respond with a button press if the probe was present in the preceding memory set, or not respond if the probe was not present in the preceding memory set. A blank screen pause (2000 ms) concluded the trial before the onset of the next trial.

2.4.4.3 Cognitive Task Calibration. For each participant, the cognitive tasks were calibrated during a preliminary testing session, to ensure that the tasks were at a level which was difficult, yet still achievable. In the visual discrimination task, the level of difficulty was defined as the two most difficult line manipulations that resulted in a response sensitivity score of 0.8 and 0.6, corresponding to the easy and difficult versions of the task respectively. In the Sternberg working memory task, this level was defined as the most difficult stimulus set that resulted in a response sensitivity value of 0.8. To attain this value, participants completed four blocks. A five minute break separated each of the calibration blocks. In all blocks, the visual discrimination task preceded the Sternberg working memory task. During the calibration session, participants were not exposed to RF and no physiological data was recorded.

2.4.5 EEG and thermo-physiological apparatus

A water perfusion garment (Grant Instruments Ltd., Cambridge, U.K.) was used to clamp body skin temperature to 34 °C to produce a thermo-neutral environment. The water perfusion suit was made of cotton and enclosed a series of pipes which distributed water across the skin at a rate of 2.5L/min at a temperature of 34 °C. The garment covered the torso, arms, waist and legs. Water temperature was controlled using a digital thermostat (Type: GD120, Grant Instruments Ltd., Cambridge, U.K.) which could heat or cool water accordingly to 34 °C ± .02°C. A range of physiological measures, including core body temperature, skin temperature, blood pressure, resting EEG and cutaneous blood flow to the left hand were also acquired. These measures are beyond the scope of this paper and will not be discussed.

2.4.6 Procedure

Participants arrived at the laboratory at either 09:00 or 13:00, with the start time for all experimental sessions kept constant within-subjects to minimise circadian effects. Participants then completed a 16-item visual analogue mood scale (VAMS) and a series of short questionnaires asking about sleep, caffeine and alcohol consumption and mobile phone usage before being fitted with the water perfusion suit, EEG and physiological recording apparatus.

Participants were then seated inside a Faraday cage in front of a Dell U2311H LCD monitor between the two RF antennas and the water perfusion suit was switched on. Participants were positioned such that their eyes were approximately 90 cm from and at the same height as the centre of the computer screen. The plane of the monitor was perpendicular to both the floor and the sagittal plane of the participants. Participants then completed a practice version of the visual discrimination and modified Sternberg working memory tasks (2.5 min each).

Once setup was complete, participants completed a 16 min 'Baseline' block, during which they were not exposed to RF. During this block participants completed an EOG correction task (Croft, Chandler, Barry, Cooper, & Clarke, 2005) and resting EEG and physiological data were also recorded.

The Baseline block was followed by two 30 min experimental blocks, the first block being 'RF-ON' (Sham, Low or High RF depending on counterbalancing) and the second 'RF-OFF'(post-exposure), with a 1 min break between each block. At the beginning of the experimental blocks, resting EEG and physiological measures were recorded. Subsequent to this, participants completed the cognitive battery; consisting of a 6 min visual discrimination task (easy and difficult consecutively) and a 9 min

Sternberg working memory task, with a 1 min break between tasks. Following the cognitive tasks, EEG and physiological data were again recorded.

At the completion of testing, all monitoring equipment was disconnected and the participant completed a 16 item VAMS and an exposure condition questionnaire. This procedure was repeated for the remaining testing sessions.

2.4.7 Data analyses

Behavioural measures were defined as mean reaction time to correctly identified target probes ('RT') recorded in ms, as well as the number of correct hits, correct rejections, false alarms and false rejections 100-900 ms post stimulus in the visual discrimination task and 100-2000 ms post probe stimulus in the Sternberg working memory task. Response sensitivity (Grier's A') and response bias (Grier's B") were used to assess task accuracy. All data points were converted to *z* scores for analysis.

Where a participant performed at below 55% accuracy in the visual discrimination task or 50% accuracy in the Sternberg working memory task, data points were interpolated in order to preserve counterbalancing. This criterion affected 1 participant (3 blocks) in the difficult version of the visual discrimination task, and 6 participants (12 blocks) in the Sternberg working memory task.

Data points were missing for a further 2 participants (2 blocks for 1 participant, 1 block for 1 participant) in the visual discrimination task due to the incorrect difficulty level being administered. The missing data points were interpolated in order to preserve counterbalancing.

Statistical analyses were performed with SPSS statistical package 21.0. Paired samples *t* tests were performed to assess the effect of exposure on RT, response sensitivity and

response bias overall (comparing the 0 W/kg condition against the average of the 1 W/kg and 2 W/kg conditions) and whether these effects depended on the dose of exposure (comparing the 1 W/kg condition against the 2 W/kg condition). Exploratory paired samples *t* tests were also conducted on non-interpolated data. To correct normality, square root transformations were computed for the response sensitivity data in the easy version of the visual discrimination task.

2.5 Results

2.5.1 RF Status

Overall, there was a greater tendency for participants to rate the exposure as being off (50.93%) or unsure as to whether it was on or off (37.05%) than on (12.03%). The participants were unable to detect the RF status better than chance with 18.52% correctly identified trials. No participant was able to correctly identify all 3 exposure conditions.

2.5.2 Cognitive Tasks

The mean RTs and standard deviations for each cognitive task and the significance of each paired samples *t* test are presented in Table 2.1. As shown in Figure 2.3, there was significant decrease in RT in the Sternberg working memory task during the exposure conditions compared to Sham, t (35) = 2.070, p = .046, r^2 = .109. This effect, however, was not significant using non-interpolated data (p = .052). No other RT effects were detected in either the visual discrimination tasks or the Sternberg working memory task when comparing Sham against the exposure conditions or the Low against the High RF condition, either during or following exposure.

The mean response sensitivity and standard deviations for each cognitive task and the significance of each paired samples *t* test are presented in Table 2.2. There were no significant differences in response sensitivity when comparing Sham against the exposure conditions or the Low against the High RF condition, either during or following exposure.

The mean response bias and standard deviations for each cognitive task and the significance of each paired samples *t* test are presented in Table 2.3. There were no significant differences in response bias when comparing Sham against the exposure conditions or the Low against the High RF condition, either during or following exposure.

Table 2.1: Mean reaction times (ms) and standard deviations for each cognitive task in each exposure condition with *p* values for Sham vs Exposure and Low vs High comparisons (values significant at $p < .05$ are in bold).

Condition	Perceptual Easy		Perceptual Difficult		Sternberg	
	BL1 M (SD)	BL2 M (SD)	BL1 M (SD)	BL2 M (SD)	BL1 M (SD)	BL2 M (SD)
Sham	358 (42)	359 (45)	368 (39)	362 (63)	877 (136)	367 (148)
Low RF	360 (42)	350 (37)	377 (63)	364 (45)	833 (138)	872 (140)
High RF	359 (42)	356 (37)	374 (42)	367 (36)	833 (130)	873 (138)
p values						
Sham vs Exposure	*.762*	*.207*	*.192*	*.330*	***.045***	*.845*
Low vs High	*.850*	*.162*	*.870*	*.436*	*.989*	*.983*

Table 2.2: Mean response sensitivity (Grier's A') and standard deviations for each cognitive task in each exposure condition with *p* values for Sham vs Exposure and Low vs High comparisons.

Condition	Perceptual Easy		Perceptual Difficult		Sternberg	
	BL1 M (SD)	BL2 M (SD)	BL1 M (SD)	BL2 M (SD)	BL1 M (SD)	BL2 M (SD)
Sham	.94 (.04)	.94 (.05)	.91 (.09)	.90 (.08)	.85 (.10)	.86 (.07)
Low RF	.94 (.06)	.94 (.06)	.91 (.08)	.91 (.08)	.85 (.08)	.85 (.09)
High RF	.93 (.06)	.94 (.05)	.92 (.07)	.91 (.08)	.86 (.07)	.86 (.07)
p values						
Sham vs Exposure	*.454*	*.741*	*.528*	*.078*	*.758*	*.515*
Low vs High	*.391*	*.920*	*.297*	*.905*	*.512*	*.228*

Table 2.3: Mean response bias (Grier's B'') and standard deviations for each cognitive task in each exposure condition with *p* values for Sham vs Exposure and Low vs High comparisons.

Condition	Perceptual Easy		Perceptual Difficult		Sternberg	
	BL1 M (SD)	BL2 M (SD)	BL1 M (SD)	BL2 M (SD)	BL1 M (SD)	BL2 M (SD)
Sham	.10 (50)	.08 (.52)	.25 (.45)	.23 (.46)	.28 (.36)	.29 (.28)
Low RF	.14 (.48)	.07 (.52)	.30 (.41)	.28 (.45)	.25 (.33)	.25 (.29)
High RF	.11 (.50)	.08 (.41)	.22 (.32)	.21 (.43)	.28 (.32)	.30 (.30)
p values						
Sham vs Exposure	*.583*	*.964*	*.882*	*.842*	*.863*	*.636*
Low vs High	*.607*	*.907*	*.197*	*.134*	*.512*	*.228*

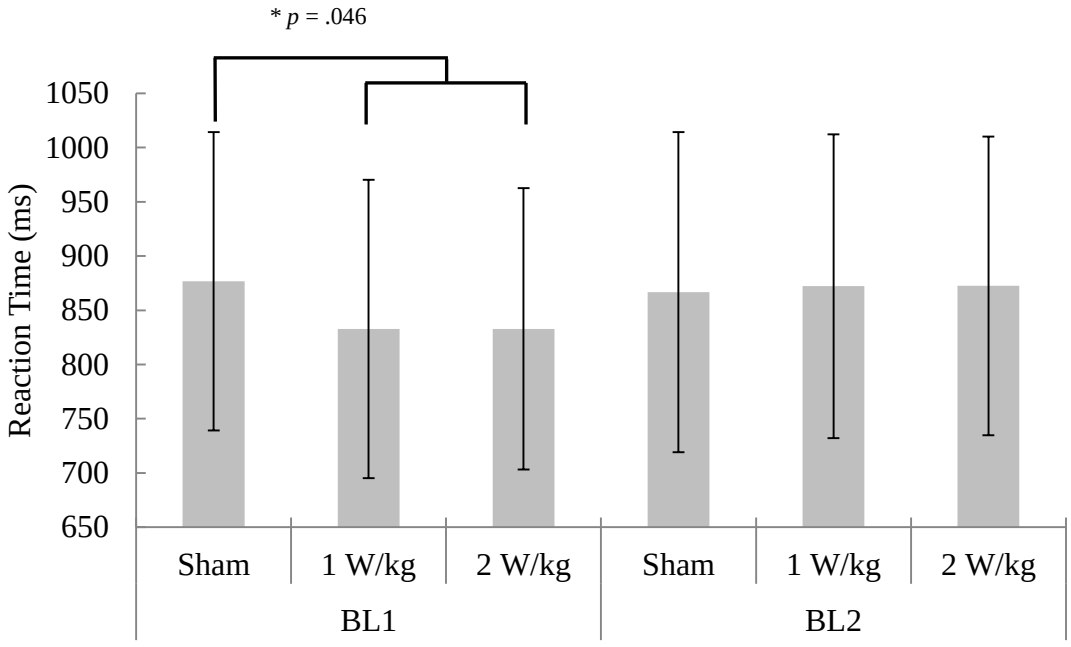

Figure 2.3: Mean reaction times (ms) in the Sternberg working memory task as a function of condition and block (BL1: during exposure, BL2: post exposure). Paired samples *t* tests showed a significant decrease in reaction time during exposure when compared to sham. Error bars represent standard deviation.

2.6 Discussion

The results of the present study indicate that exposure to PM RF influences cognitive performance in the Sternberg working memory task. Specifically, a significant decrease in RT was revealed during the active exposure conditions compared to Sham. This suggests that exposure to PM RF may have a positive influence on cognitive performance. This effect, however, was not found to be dose dependent. While the exploratory analysis revealed that this effect was not significant using the non-interpolated data, the interpolation was used to preserve the sample size and counterbalancing. Therefore, because the non-interpolated data set contained fewer participants, the significance of this effect was expected to be reduced in the non-interpolated dataset.

While the present study's findings are consistent with early reports that exposure to PM RF affects working memory performance (Koivisto et al., 2000; Regel, Gottselig, et al., 2007; Regel, Tinguely, et al., 2007), the majority of the literature has not found such an influence (Haarala et al., 2003; Haarala et al., 2004; Haarala et al., 2007; Krause et al., 2007; Leung et al., 2011). Although we cannot be conclusive, a number of reasons related to the methodological improvements employed by this study may explain the results.

The working memory task used in the present study differs markedly from the N-back task, which is the task that has been typically utilised in provocation studies assessing the effect of PM RF exposure on working memory performance. It seems that the N-back task was used primarily because of its perceived face validity and (in latter studies) as a means to replicate previously reported effects. The problem, however, is that the N-back task is limited in its ability to control for individual differences in working

memory performance, as well as learning, floor and ceiling effects. These confounds have the potential to add large amounts of noise to the data, thereby masking potentially real effects. The Sternberg working memory task overcomes these confounds by utilising a greater number of difficulty levels to calibrate the task to each individual's cognitive ability. This calibration, alongside sufficiently long practice blocks prior to each experimental session, increases sensitivity and thus the ability to detect any potential effect of PM RF exposure on working memory performance.

To date, only one other study has accounted for individual differences in working memory performance by tailoring cognitive task difficulty to participants' individual ability. After calibrating the N-back task to a level which was 'difficult, but achievable for each participant,' Leung et al. (2011) did not find any difference in accuracy or RT during PM RF exposure compared to Sham. However, in a 3G (W-CDMA) condition, Leung et al. (2011) found a significant decrease in accuracy in an adolescent group compared to Sham. The present study was able to control for a greater amount of variation in individual differences in performance, and thus remove the noise which may have masked any effects.

As the only known mechanism of interaction between RF and the human body is thermal (Adair & Black, 2003), it is possible that whole body thermoregulatory processes play an important role in mediating the changes in the brain's electrical activity and any potentially associated functional effects resulting from exposure to PM RF. To reduce thermally induced variability, the present study clamped skin temperature to a thermo-neutral state. As this is the first study to attempt to reduce the influence of this potential confound, this should be further explored in future studies.

Variation in exposure setups and SAR profiles and a lack of detailed dosimetric data has made it difficult to compare and replicate previously reported effects (Boutry et al., 2008; Regel & Achermann, 2011). While it is possible that certain brain regions need to be adequately exposed to PM RF to produce an effect, it should be noted that because the SAR distribution produced by the present planar exposure system is more homogenous than mobile phone hand-set exposure, this present exposure differs significantly to the more localised exposure produced by a typical mobile phone. Therefore, the results of this study can only reflect whether RF related bioeffects can occur at the maximum exposure level anywhere within the exposed hemisphere (Loughran, McKenzie, Anderson, McIntosh, & Croft, 2008). Thus, the present study cannot definitively comment on whether cognitive performance is influenced by PM RF emitted by *mobile phones*, nor can it comment on the effect of long term exposure or the effect of exposure on different age groups within the population. Indeed, the World Health Organisation has identified research investigating acute effects of PM RF exposure on cognition and EEG with children as a priority. While some studies have not found effects of PM RF exposure on cognitive performance in children and adolescents (Haarala et al., 2005; Leung et al., 2011; Preece et al., 2005), the methodology used in the present study may prove to be more sensitive, and should be considered in future research with children.

A number of factors may have limited this study. First, while the statistical analysis controlled for comparison-wise error by restricting the planned contrasts to degrees of freedom error (without multiple comparison adjustment) (Tabachnick & Fidell, 2013), this method does not control for experiment-wise error. Second, it is possible that the exposure levels were affected by the EEG electrodes. However, the potential for this to

occur has been explored in great detail and is unlikely to have influenced the results. Generally, EEG electrode leads have been found to produce a shielding effect, reducing the SAR in head regions close to the antenna and also where the maximum value is obtained. The reduction in SAR tends to be less than 20% (Hamblin et al., 2007; Murbach, Neufeld, Christopoulou, Achermann, & Kuster, 2014). These findings indicate that electrode configurations do not act like an antenna, and therefore do not enhance SAR. Furthermore, as the reductions have been found to be less than 20%, electrode configurations are also not thought to greatly attenuate SAR to the point where results are influenced (Hamblin et al., 2007; Murbach et al., 2014).

Whether small variations in performance on cognitive tasks as a result of exposure to PM RF constitute any meaningful effects in real life situations remains a valid question. In a critical review of this field, Regel and Achermann (2011) hypothesise that if such elementary motor reactions are influenced by exposure to PM RF, effects on higher cognitive functions may be even stronger. It is also possible, however, that the significant cognitive performance effects found in some RF-EMF provocation studies have occurred unpredictably and independent of task type (Regel & Achermann, 2011). This may explain why the significant differences found in the present study only occurred in one cognitive task on one variable. But while the results of the present study indicate that there is a slight change in performance, it is important that the methodology implemented in this study is replicated before determining whether such an effect is meaningful.

In conclusion, the present study has shown that PM RF exposure influences cognitive performance in a working memory task. While the majority of the literature has not found an effect of PM RF exposure on cognitive performance, it is possible that the

methodological improvements employed in the present study increased sensitivity, and thus the ability to detect potential effects. However, as this is the first PM RF provocation study to implement these improvements, replication is required in order to determine whether these effects represent more than chance findings.

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3. CHAPTER 3: IEI-EMF PROVOCATION CASE STUDIES: A NOVEL APPROACH TO TESTING SENSITIVE INDIVIDUALS

3.1 Chapter Foreword

Despite accounting for a number of methodological limitations, as no clear evidence of adverse effects was demonstrated in Study 1, the study failed to identify any sensitive health-relevant cognitive performance endpoints with which to test IEI-EMF participants. In light of this outcome, a change of focus for investigating the determinants of IEI-EMF was required.

While provocation studies offer one of the most powerful methods for testing the claims of IEI-EMF sufferers, these studies have been heavily criticised by many IEI-EMF advocacy groups and a minority of researchers. These criticisms are often related to questions about whether laboratory testing environments adequately reflect the conditions in which symptoms normally develop, and a failure of previous studies to account for the heterogeneous nature of the condition. While there is currently no empirical evidence to suggest that such issues have affected the outcomes of previous studies, as these criticisms are often used to suggest that provocation studies are inappropriate for investigating the symptom claims of IEI-EMF sufferers, it is clear that an improved provocation study design is needed in order to adequately determine the aetiology of the condition.

The study presented in this chapter (Study 2) incorporated a number of methodological improvements designed to specifically address the potential methodological issues that are claimed to render provocation studies inappropriate for assessing the determinants

of IEI-EMF. The study aimed to determine whether IEI-EMF symptoms are more closely related to EMF exposure or a nocebo effect. This chapter has been published in the journal *Bioelectromagnetics.*

Verrender, A., Loughran, S.P, Anderson, V., Hillert, L., Rubin, G.J, Oftedal, G., & Croft, R.J (2018). IEI-EMF provocation case studies: A novel approach to testing sensitive individuals. *Bioelectromagnetics, 39,*132 – 143

3.2 Abstract

The aetiology of Idiopathic Environmental Intolerance attributed to Electromagnetic Fields (IEI-EMF) is controversial. While the majority of studies have indicated that there is no relationship between EMF exposure and the symptoms reported by IEI-EMF sufferers, concerns about methodological issues have been raised. Addressing these concerns, the present experiment was designed as a series of individual case studies to determine whether there is a relationship between RF-EMF exposure and an IEI-EMF individual's self-reported symptoms. Three participants aged 44-64 were tested during a series of sham and active exposure trials (2 open-label trials; 12 randomised, double-blind, counterbalanced trials), where symptom severity and exposure detection were scored using 100mm visual analogue scales. The RF-EMF exposure was a 902-928 MHz spread spectrum digitally modulated signal with an average radiated power output of 1 W (incident power density at the participant 0.3 W/m^2). In the double-blind trials, no significant difference in symptom severity or exposure detection was found for any of the participants between the two conditions. Belief of exposure strongly predicted symptom severity score for all participants. Despite accounting for several possible limitations, the present experiment failed to show a relationship between RF-EMF exposure and an IEI-EMF individual's symptoms.

3.3 Introduction

A proportion of the population report experiencing a wide range of non-specific symptoms which they attribute to the electromagnetic fields (EMF) emitted by various electronic and wireless technologies. Commonly referred to as Electromagnetic Hypersensitivity (EHS), the condition is characterised by a variety of dermatological, neurasthenic and/or vegetative symptoms, with headaches, nausea, skin irritations, fatigue and concentration difficulties amongst the most commonly reported symptoms (Hagström, Auranen, & Ekman, 2013; Hillert, Berglind, Arnetz, & Bellander, 2002; Kato & Johansson, 2012; Röösli, Moser, Baldinini, Meier, & Braun-Fahrländer, 2004). Generally, the reported symptoms are claimed to be triggered by technologies which emit EMF in the radiofrequency (RF-EMF) and extremely low frequency (ELF-EMF) domains of the non-ionizing radiation spectrum, at levels well below the thresholds known to cause adverse health effects in humans (ICNIRP, 1998, 2010). These devices include mobile phones and their base-stations, Wi-Fi, electricity transmission and distribution systems, and 'smart' meters. The condition can have major implications for an individual's quality of life and is often associated with decrements in general health status, increased levels of distress, increased levels of health service use and serious impairments in occupational and social functioning (Johansson, Sandström, Heiden, & Nordin, 2010).

Yet, despite the considerable prevalence of the condition globally (estimated to be between 1.5 – 13.5%) (Baliatsas et al., 2015; Blettner et al., 2009; Eltiti, Wallace, Zougkou, et al., 2007; Hillert et al., 2002; Levallois, Neutra, Lee, & Hristova, 2002; Schreier, Huss, & Röösli, 2006; Schröttner & Leitgeb, 2008; Tseng, Lin, & Cheng, 2011), recent reviews of the scientific literature have concluded that there is no

relationship between exposure to EMF and the non-specific symptoms reported by EHS individuals (Health Canada, 2015; Health Council of the Netherlands, 2009; Röösli, Frei, Mohler, & Hug, 2010; Rubin, Das Munshi, & Wessely, 2005; Rubin, Nieto-Hernandez, & Wessely, 2010; Scientific Committee on Emerging and Newly Idenified Health Risks, 2015). This discrepancy between the scientific consensus and the subjective reports of sensitivity to EMF not only limits the treatment options and support for those who experience EHS, but also leaves some members of the public feeling uncertain and anxious about potential adverse health effects of EMF exposure. Due to the lack of evidence for an association between exposure to EMF and EHS, the World Health Organization recommended that the term Idiopathic Environmental Intolerance attributed to Electromagnetic Fields (IEI-EMF) be used in place of EHS to avoid implying a causal role of EMF in producing the reported symptoms (World Health Organisation, 2004).

Experimental provocation studies have been predominately used as a means of investigating IEI-EMF. In these studies, a participant is exposed to both active and sham EMF under controlled, preferably double-blinded protocols, while their symptomatic response to each condition is monitored. Over the past decade, a number of provocation studies using a range of EMF and varying methodologies have failed to provide sufficient evidence to support the view that IEI-EMF is directly associated with exposure to EMF (Rubin et al., 2010; Scientific Committee on Emerging and Newly Idenified Health Risks, 2015; World Health Organisation, 2014). Indeed, sham exposures alone have been found to be sufficient to trigger symptoms in IEI-EMF participants (Nam et al., 2009; Oftedal, Straume, Johnsson, & Stovner, 2007; Wilén, Johansson, Sandström, Kalezic, & Lyskov, 2006). Two studies have also reported an

increase in symptoms in an initial non-blinded active exposure condition, compared to sham, but have then found no significant differences between active and sham conditions in subsequent double-blind trials (Eltiti, Wallace, Ridgewell, et al., 2007; Wallace et al., 2012). Similarly, a recent study reported that IEI-EMF participants were unable to correctly identify when they were being exposed under double-blind conditions, despite an initial verification that they could detect active from sham conditions in an open-label trial (van Moorselaar et al., 2017). These findings have led many to suggest that IEI-EMF may be the result of a nocebo response, where conscious or subconscious symptom *expectation* following a *perceived* exposure to EMF leads to the formation or detection of symptoms (Hillert et al., 2008; Landgrebe et al., 2008; Oftedal et al., 2007; Rubin et al., 2010). Recent findings from a qualitative study, however, suggest that instead of the condition originating from a nocebo response, IEI-EMF individuals may be using the notion of sensitivity to EMF to provide a narrative to explain their medically unexplained symptoms (MUS) in an effort to make their condition more practically and emotionally manageable (Dieudonné, 2016).

Although the reviews cited above have failed to support the view that EMF exposure was related to symptoms in self-diagnosed IEI-EMF participants, it is important to note that there are a number of studies that have claimed to identify such relations, and which are often used in support of the claim that there is a causal relation. However, such studies do not provide the claimed support, but are more easily explicable in terms of methodological considerations. For example, McCarty et al. (2011) claimed an effect of on-off electric field transitions, but as the study was later criticised for lacking clear methodology, and given that it has not been replicated, this cannot be taken as evidence for a relation (Rubin, Cleare, & Wessely, 2011, 2012). Similarly, Kwon et al.(2008)

reported that two healthy participants were able to detect EMF at greater than chance levels, but they could not replicate their results in the same individuals a month later, which suggests that whatever caused the initial significant results, it is unlikely that it was due to a bioelectromagnetic phenomenon.

In line with the focus on methodology, both advocacy groups and some researchers have argued that the null results are due to methodological limitations, such as a failure to account for the heterogeneous nature of the condition and the way in which participants have been selected and tested. For example, it is possible that the samples tested have included a combination of both individuals who are sensitive to EMF and others who may suffer from unrelated conditions (Rubin et al., 2010). This is problematic, as the majority of studies have taken a nomothetic approach to testing IEI-EMF, and have therefore relied on group means which may have had potentially reduced statistical power due to the noise added to the analysis from non-responders. In addition, few studies have tested whether the exposure signal used was relevant in eliciting symptoms for each individual in the sample, which again may have potentially made the RF-EMF exposure irrelevant for many of the participants. Furthermore, while the experience of IEI-EMF is known to vary considerably between individuals in terms of the type and severity of symptoms experienced and the amount of time required for symptoms to develop and subside following exposure (Hocking, 1998; Röösli et al., 2004), the majority of provocation studies have not taken this heterogeneity into account. Instead, studies have generally used relatively standard exposure and wash-out periods across all participants, which, without verification of an open-label effect using the particular study protocol, may again make the protocol irrelevant to the reported EMF-symptom relation and make interpretation problematic.

Concerns have also been raised about whether the testing environments of provocation studies adequately reflect the conditions in which IEI-EMF individuals report symptoms. It is possible, for instance, that the laboratory setting has caused some participants to experience anxiety, which may have then affected their symptom response. It is also possible, on the assumption that there is a relation between EMF and symptoms, that participants have encountered other EMF exposures on the way to an experimental session which have then inadvertently triggered symptoms (Rubin et al., 2010). If symptoms had been triggered by external factors prior to the experimental manipulation, this would also increase the error variance and potentially mask any real effects. However, although it is logically possible that these limitations have masked real effects of EMF on symptoms, it is important to point out that there is no substantiated evidence that this is the case; such issues need to be determined empirically.

In light of this, the present experiment was designed as a series of individual case studies to determine whether there is a relationship between RF-EMF and an IEI-EMF individual's self-reported symptoms, employing several important methodological improvements in order to overcome potential limitations of previous studies. First, the study utilised a portable exposure device which enabled double-blind testing to take place in environments where participants generally felt safe and asymptomatic, such as in their own home. This was implemented in order to reduce the stress and anxiety which may be experienced by a participant in a laboratory setting, while also reducing potential confounding effects associated with inadvertent exposures to environmental EMF emissions on the way to an institutional testing location. Second, the methodology incorporated a consideration of each participant's IEI-EMF symptom history. This

included using a similar RF-EMF exposure to the one which the participant claimed triggers symptoms, and both the exposure source and reported symptoms were individually verified in an initial open-label, non-blinded trial. This limits potential 'non-responder' data from statistically confounding 'responder' data. Further to this, the study included a consideration of the reported symptom onset and recovery periods, such that the testing regime, if necessary, could be modified to incorporate these. A sufficient number of sham and exposure conditions were also used to determine statistically, within the individual, whether any symptom/exposure relation was significant. Finally, the design incorporated a fully counter-balanced protocol in order to reduce time of day and time on task effects. The aim of the study was first to test whether exposure to RF-EMF from the portable exposure device resulted in an increase in an IEI-EMF participant's nominated symptom compared to sham, and second, to determine whether IEI-EMF participants could detect the active RF-EMF signal at greater than chance levels, under double-blind conditions.

3.4 Materials and Methods

3.4.1 Participants

In total, twenty-five potential participants contacted the research centre during the recruitment period. Of these, three participants aged 44-64 (two male) completed the study. Six participants were excluded from the study in an initial phone screen due to not meeting the eligibility criteria. The remaining sixteen participants either expressed that they did not want to continue participation in the study (after receiving a participant information sheet and speaking with the researchers via telephone) or could not be re-contacted by the researchers.

Participants were recruited through advertisements on the research centre website and via a press release in the local newspaper and television network. All participants were first screened via a telephone interview to confirm eligibility for the study. To be included in the study, participants must have reported one or more *acute* symptoms which they attributed to the use of or to their personal proximity to mobile phone or Wi-Fi devices. Acute symptoms were defined as any symptom with an onset time of less than 30 min and which took less than 2 hr to subside following exposure, and that could be self-managed without the need of a health professional. Participants must have also self-diagnosed or labelled themselves as having EHS or IEI-EMF for greater than 1 year. Participants were excluded from the study if they reported any serious medical or psychological illnesses, or indicated that they used recreational illicit drugs.

A mutually convenient testing time was arranged with suitable participants. The study was approved by the Human Research Ethics Committee (University of Wollongong: HE15/160), and informed written consent was obtained from all participants.

3.4.2 Radiofrequency Exposure

RF exposure was generated using a portable, self-contained, battery-operated device (Two Fields Consulting, St Kilda, Australia). The RF device was placed 30 cm from the participant (either on the side or to the front depending on what was comfortable for the participant) on a hard surface. The main exposure from the device was a spread spectrum RF signal in the 902-928 MHz ISM band which was digitally modulated in a similar manner to signals from Wi-Fi and 3G and 4G mobile phones. The RF signal was generated by a commercial RF modem which emitted a frequency hopping spread spectrum signal with an average radiated power output of 1 W for 30 min, or was completely EMF silent (RF-OFF, sham trials). The incident RF exposure level from the

side of the device facing the participant was measured using a calibrated broadband instrument with an uncertainty of ±2.4 dB for a two-sided coverage interval and a coverage factor of 2 (Narda EMR 300 meter and Type 9 E-field probe, Narda Safety Test Solutions, Hauppauge, NY), and was found to be 0.3 W/m². This RF exposure level is below the power density reference level limit of 4.6 W/m² specified for the Australian general public (ARPANSA RPS3) and by the ICNIRP (1998). It is important to note that the maximum localised specific absorption rate (SAR) from the exposure device used in the present study is less than that which typically results from personal mobile phone use (held against the ear in the active taking mode) due to the greater separation distance. Conversely, the whole body averaged SAR and localised SAR of the device used in the present study is greater than that which normally results from Wi-Fi and mobile phone base station signals. The device was fully enclosed in a thermally insulated case and coded inputs were used to maintain double-blinding. The device contained an independent RF monitor to check the status of the RF transmitter and each use of the device was logged using internal memory. The fields emanating during the RF-ON exposure and sham conditions were demonstrated to each participant in the open-label trial using a Nardalert S3 broadband monitor (Narda Safety Test Solutions, Hauppauge, NY). This monitor was then removed for the subsequent double-blind testing.

3.4.3 Questionnaires

3.4.3.1 Demographic and health questionnaire

Demographic and health screening questionnaires were used to capture data on the age, handedness, education level, gender, general medical condition, and caffeine, tobacco, alcohol, illicit and medically prescribed substance use of each participant.

3.4.3.2 Symptom history questionnaire

Two open ended questions were used to assess each participant's symptom history. These were "What are the two most immediate EMF symptoms you experience?" and "Do you suffer from any debilitating EMF symptoms?" Participants were asked to include information on the source perceived to be responsible for triggering the symptoms, the symptom severity, the time of onset and the time needed for the symptom to subside, the first time the symptom was experienced and any treatment methods used to relieve the symptom. Participants were also asked to indicate any other EMF symptoms which they regularly experienced on a checkbox list of 11 common IEI-EMF symptoms (Rubin, Hahn, Everitt, Cleare, & Wessely, 2006).

3.4.3.3 WHOQOL-BREF

The WHOQOL-BREF (World Health Organisation, 1998) assesses how disease impairs the subjective well-being of a person across a range of domains. The questionnaire comprises 26 items, which measure quality of life in the following broad domains: physical health, psychological health, social relationships, and environment.

3.4.4 Symptom and Exposure Status Scale (SESS)

During the provocation trials, participants were asked to indicate symptom severity and exposure status via pen and paper 100mm visual analogue scales. Participants were asked "how sure are you of the current exposure status *right now*?" anchored with the terms 'Definitely OFF' and 'Definitely ON', and "how strong/unpleasant is your nominated symptom *right now*?" anchored with the terms 'Barely Detectable' and 'Maximum Severity'. While a full symptom history was obtained from each participant prior to testing, the symptom tested in the double-blind trials was defined as the most immediate symptom triggered during the initial open-label RF-ON trial.

3.4.5 Design

Each participant's symptom severity and exposure detection ability was tested under a series of 14 sham and active provocation trials. On the first day of testing, two open-label trials (1 RF-OFF, 1 RF-ON) were conducted, where both the participant and the researcher were aware of the exposure status. This was used to determine whether the exposure device could trigger individually-relevant symptoms in each participant. If a participant did not report symptoms or was unable to detect the exposure in the RF-ON condition in this initial test, their participation in the experiment ceased at this point. The initial open-label trials were followed by a series of 12 double-blind, randomised, counterbalanced trials, consisting of 6 sham and 6 RF-ON exposure conditions. This was achieved using the Excel randomization command, such that a sham and RF-ON condition were treated as a pair; the conditions of each pair were randomly allocated before assigning the next pair; where more than two sequential pairs had the same order the third pair was replaced with the alternate pair order; and no more than three of the same pair-order were permitted. In total, each trial ran for 105 min (except for the RF-OFF open-label trial, where there was no post-trial assessment or rest interval as there had been no exposure). For each participant, the 14 trials took 24 hr to complete, spread over a period of 3 consecutive days (the number of RF-ON and sham trials were matched within each day).

3.4.6 Testing Location

Testing was conducted in a safe, asymptomatic environment (determined by the participant) in order to reduce stress and to reduce any confounding effects due to environmental RF emissions. In all three cases, participants chose (and were tested in) their own home.

3.4.7 Procedure

Upon arrival at the participant's home, the researchers set up the exposure device in a comfortable area and ensured that all known electronic and RF emitting devices were switched off. All participants were then given a verbal description of the ensuing session before completing demographic and health screening questionnaires. To begin the provocation trials, participants were asked to sit comfortably in a chair with the exposure device placed approximately 30 cm from them (either to the side or in front of them, depending on what was comfortable for the participant). The progression of each provocation trial is shown in Figure 3.1. The first day of testing began with two open-label trials. The first open-label trial was an RF-OFF (sham) trial, which began with a 15 min baseline interval (no exposure; status known to participant and researcher) to assess the participant's symptom severity pre-trial. The SESS was completed at the 1- and 14-min mark (B1 and B2) of the trial. This was followed by a 30 min exposure interval, where the exposure device was switched to an RF-OFF (sham) setting (exposure status known to the participant and researcher) and the SESS was again completed at the 16-, 30- and 44-min mark of the trial (E1, E2 and E3). The RF-OFF open-label trial was immediately followed by the RF-ON (active) open-label trial. Again, a 15 min baseline interval (no exposure; status known to participant and researcher) was used to assess the participant's symptom severity pre-trial. The SESS was completed at the 1- and 14-min mark (B1 and B2) of the trial. This was followed by a 30 min exposure interval where the exposure device was switched to an RF-ON setting (exposure status known to the participant and researcher) and the SESS was completed at 16-, 30- and 44-min mark (E1, E2 and E3) of the trial. The exposure interval was then followed by a 30-min post-exposure assessment (no exposure; status known to the participant and researcher), where the SESS were again completed at the

46-, 60- and 74-min mark (PE1, PE2 and PE3) of the trial. The post-exposure interval was followed by a 30 min rest interval, where the participant was free to move around, rest and consume food and water before the onset of the next trial. The subsequent 12 double-blind trials followed the same progression as the open-label RF-ON trial, except that during the exposure interval, the exposure device was set to either sham or RF-ON (status unknown to the participant and researcher) depending on randomization and counterbalancing.

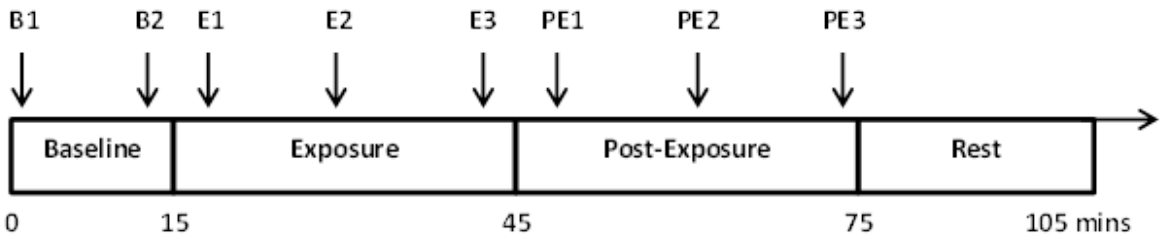

Figure 3.1: Provocation trial design. Each trial begins with a 15 min Baseline (B) interval, followed by a 30 min Exposure (E) interval, a 30 min Post-Exposure (PE) interval and a 30 min Rest interval, with a total trial time of 105 min. Arrows represent the time points where the SESS was administered.

3.4.8 Statistical Analysis

Statistical analyses were performed with SPSS Statistics for Windows 21.0 (IBM, Armonk, New York). For each individual, a Mann-Whitney U test was used to assess the difference in symptom severity and exposure detection ability, comparing the 6 sham to the 6 RF-ON double-blind exposure conditions (which are treated as independent). This provides power (0.80) to detect effect sizes of > 1.6 with an alpha = 0.05, which is consistent with the (anecdotal) reports of effect sizes from IEI-EMF sufferers (who claim to be able to reliably detect and/or suffer symptoms from EMF). It is important to note that there are currently no effect sizes related to actual effects of exposure, which is why one based on anecdotal reports of IEI-EMF has been used. The primary dependent variable was the difference between the baseline score at 14 min of the trial (B2) and the exposure score at 44 min of the trial (E3), for both symptom severity and exposure detection. A difference score was used to minimise the influence of baseline variability and potential carry-over effects. In order to determine the magnitude of the effect induced by the open-label exposure for each participant, an effect size was calculated, based on the difference in symptom severity for the RF-ON and RF-OFF condition. However, because there is no measure of variability in the open-label trial, the experimental double-blind data was used to calculate a standard deviation. To achieve this, the effect of belief of exposure first needed to be removed. To do this, a simple linear regression was conducted to predict symptom score based on how confident each participant was that the exposure was on or off in the double-blind trials (belief of exposure), and unstandardised residuals were calculated. The unstandardised residuals were then used to calculate the standard deviation, which could then be used in the effect size calculation of the open-label trials. These linear regressions also provided important information regarding the potential relation

between belief and symptom severity for each participant via the resultant r-squared values.

3.5 Results

3.5.1 General health status

The participants did not report any severe medical or psychological conditions. One participant reported suffering from tinnitus and one participant was on thyroid hormone replacement therapy but was clinically euthyroid at the time of the tests.

3.5.2 Effect size in the open-label trials

Confirming that the open-label manipulation had worked in each case, all of the calculated effect sizes in the open-label trials were extremely large (P01 = 5.97, P02 = 3.66, P03 = 6.98), and much larger than the traditionally used nomenclature of Cohen (1988), which treats the largest category of effect size as >.5.

3.5.3 Participant 1 (P01):

3.5.3.1 Symptom history

The two most common immediate symptoms the participant reported experiencing in response to EMF were headache (severity 8/10) and dizziness (severity 8/10), with an onset time of 10 min and taking up to 2 h to subside. The two most common debilitating symptoms reported by the participant were Vertigo (with an onset time of 12 to 24 hr following exposure and taking up to 2 days to subside), and confusing thoughts (onset time and time needed to subside not known). The participant also reported experiencing nausea, fatigue, eye pain, skin itching, sensation of burning on the skin, memory loss, insomnia and immune system deficiency. These symptoms were attributed to mobile phone base stations, Wi-Fi, mobile phones and wireless phones. The symptoms

developed 5 years prior to testing. Although the participant reported a number of symptoms, headache was reported as the immediate symptom in the RF-ON open-label trial and used as the symptom assessed in the double-blind trials.

3.5.3.2 Exposure Detection and Symptom Provocation

Open-label trial: The results of the open-label trial are shown in Figure 3.2a. In the RF-ON condition, the participant was confident that the exposure device was emitting RF, and experienced an increase in symptom severity from baseline throughout the trial. As shown in Figure 3.2a, the severity of these symptoms gradually decreased during the post-exposure interval. These results indicate that the participant developed symptoms and reported detecting the active RF signal. A gradual decrease in symptom severity post-exposure was also observed. The participant did not detect the presence of RF or exhibit an increase in symptom severity in the RF-OFF condition.

Double-blind trials: The results of the double-blind trials are shown in Figure 3.2b. Symptom severity (*Median* =14.00 versus 34.00, $U = 15.00$, $z = -.481$, $p = .699$, $r = .139$) and detection ability (*Median* = 54.50 versus 86.50, $U = 17.50$, $z = -.087$, $p = .930$, $r = .025$) did not differ significantly between the RF-ON and sham trials respectively. The regression analysis showed that 'belief of exposure' significantly predicts symptom severity ($F(1, 10) = 48.799$, $p < .001$; $R^2 = .830$).

3.5.3.3 WHOQOL-BREF

As shown in Table 3.1, the participant's overall quality of life, physical health, psychological health and overall health scores are below the mean population norm (but within one standard deviation). The social relationships and environment scores are above the population norms.

3.5.4 Participant 2 (P02):

3.5.4.1 Symptom history

The two most common immediate symptoms the participant reported experiencing in response to EMF were feelings of 'induced hangover' with an onset time of 30 s to 5 min (severity 5/10) and a burning sensation in the throat (severity 5/10) with an onset time of 4 to 5 min. The participant reported that the time symptoms take to subside can vary substantially depending on the exposure, but estimated a range of between 30 min to 4 hr. No debilitating symptoms were reported by the participant. The participant also reported experiencing eye pain and spots on the face. The reported symptoms were attributed to mobile phones and developed 16 years prior to testing. Although the participant reported a number of symptoms, a burning sensation in the throat was reported as the immediate symptom in the RF-ON open-label trial and was therefore used as the symptom assessed in the double-blind trials.

3.5.4.2 Exposure Detection and Symptom Provocation

Open-label trial: The results of the open-label trial are shown in Figure 3.2c. In the RF-ON condition, the participant was confident that the exposure device was emitting RF, and experienced an increase in symptom severity from baseline throughout the trial. The severity of this symptom fluctuated during the post-exposure interval. These results indicate that the participant developed an individually relevant symptom and reported the presence of the active RF exposure. In the RF-OFF trial, the participant did not report the presence of RF but a slight increase in symptom severity was also observed.

Double-blind trials: The results of the double-blind trials are shown in Figure 3.2d. Symptom severity (*Median* = 6.50 versus 2.50, $U = 14.00$, $z = -.656$, $p = .512$, $r = .189$) and detection ability (*Median* = 49.00 versus 15.50, $U = 8.00$, $z = -1.601$, $p = .109$, $r =$

.462) did not differ significantly between the RF-ON and sham trials respectively. The regression analysis showed that 'belief of exposure' significantly predicts symptom severity ($F(1, 10) = 79.290$, $p < .001$; $R^2 = .888$).

3.5.4.3 WHOQOL-BREF

As shown in Table 3.1, the participant's overall quality of life score is below the population norm, overall health score above the population norm, and the remaining domains are within the population norms.

3.5.5 Participant 3 (P03):

3.5.5.1 Symptom history

The two most common immediate symptoms the participant reported experiencing in response to EMF were feelings of pain and strain in the head and ears with an onset time of 1 to 5 min (severity 5/10), which they attributed to Wi-Fi. The participant reported that these symptoms subside within 5 to 15 min. The participant also indicated that they experience headache, mild dizziness, fatigue, tinnitus, and "sensations which self-highlight in the knees, elbows, tendons and lower arms" which they attributed to EMF exposure. The participant also reported experiencing a heavy head and eyelids, memory loss, pain and strain, and a tingling sensation attributed to EMF from television, however, the symptomatic response to EMF from television was unable to be tested in the current protocol. The reported symptoms developed at least 12 years prior to testing. Although the participant reported a number of symptoms, a feeling of pain and strain in the head and ears was reported as the immediate symptom in the RF-ON open-label trial and was therefore used as the symptom assessed in the double-blind trials.

3.5.5.2 Exposure Detection and Symptom Provocation

Open-label trial: The results of the open-label trial are shown in Figure 3.2e. In the RF-ON trial, the participant was confident that the exposure device was emitting RF and they experienced an increase in symptom severity during the trial. The severity of this symptom decreased during the post-exposure interval. In the RF-OFF trial, the participant did not report the presence of RF but there was a decrease in symptom severity from baseline. These results indicate that the participant developed symptoms and reported detecting the presence of RF during the RF-ON exposure trial.

Double-blind trials: The results of the double-blind trials are shown in Figure 3.2f. Symptom severity (*Median* = 0.50 versus 1, $U = 17.50$, $z = -.082$, $p = .935$, $r = .024$) and detection ability (*Median* = 50.50 versus 47.00, $U = 12.00$, $z = -.966$, $p = .334$, $r = .288$) did not differ significantly between the RF-ON and sham conditions respectively. The regression analysis showed that 'belief of exposure' significantly predicts symptom severity ($F(1, 10) = 34.093$, $p < .001$; $R^2 = .773$).

3.5.5.3 WHOQOL-BREF

As shown in Table 3.1, the participant's overall quality of life is below the population norm, their psychological health, social relationships, physical health and environment scores are well below the population norms, and their overall health score is below the population norm (but within 1 SD).

Table 3.1: WHOQOL-BREF participant domain scores and mean population norms. Mean population scores were derived from (World Health Organisation, 1998).

Domain	P01 Domain Score	P02 Domain Score	P03 Domain Score	Population Norms (*SD*)
Overall Quality of Life	4	3	3	4.3 (*0.8*)
Overall Health	2	5	3	3.6 (*0.9*)
Physical Health	63	81	88	80.0 (*17.1*)
Psychological Health	69	63	38	72.6 (*14.2*)
Social Relationships	81	56	0	72.2 (*18.5*)
Environment	75	69	94	74.8 (*13.7*)

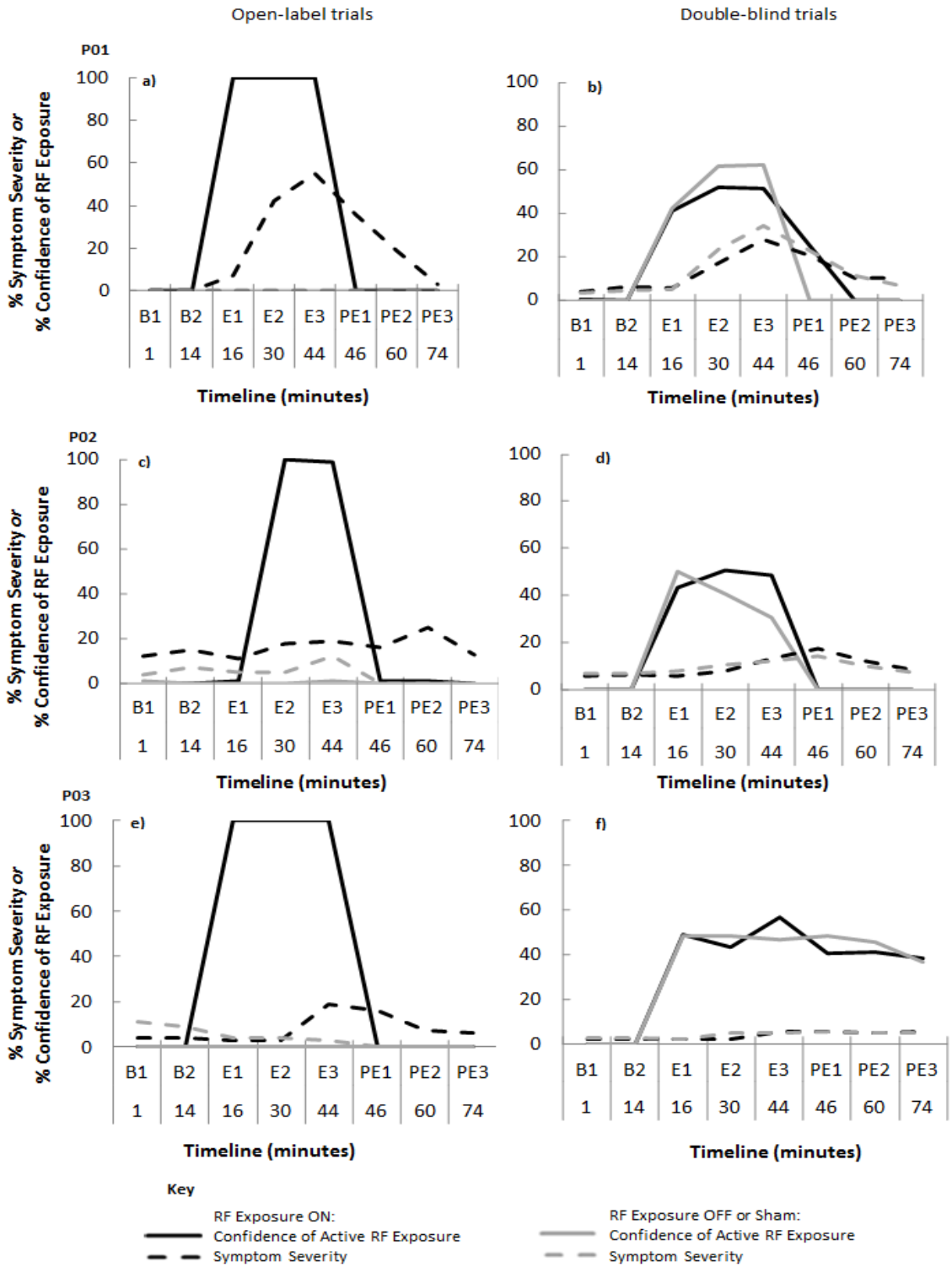

Figure 3.2: Mean exposure detection and symptom severity scores across the Baseline (B1 – B2), Exposure (E1 – E3) and Post Exposure (PE1 – PE3) intervals for P01, P02 and P03 are shown, for the open-label (RF-ON and OFF) [left column; a, c, e] and double-blind provocation trials (RF-ON and sham) [right column;b, d, f] separately.

3.6 Discussion and Conclusions

A number of methodological issues have been raised by both IEI-EMF advocacy groups and researchers as possible explanations for why provocation studies have generally failed to provide evidence of a relationship between EMF exposure and IEI-EMF symptoms. The present study was designed as a series of individual case studies which incorporated several methodological improvements to overcome limitations of previous studies. In order to determine whether these methodological improvements were adequate in providing the necessary conditions to test IEI-EMF participants, an initial open-label trial was conducted in each case.

Crucially, the results of these open-label trials show that the limitations of previous studies were sufficiently dealt with. Specifically, the testing environment and the type of exposure used were shown to be sufficient to produce the individually relevant symptoms which each participant self-nominated as being due to exposure to EMF and for each participant to report that RF exposure was indeed active in the RF-ON trial. This is important, as it confirms that the environment, RF-EMF exposure device and emitting EMF strength used in the study was relevant for eliciting symptoms for these particular IEI-EMF individuals. In addition, the observed increase in symptoms over the 30 min open-label active exposure interval (on average) shows that the exposure interval was sufficient to evoke relevant symptoms in each participant, while the reduction in symptoms in the post-exposure interval demonstrates that the time course of each trial was sufficient to allow symptoms to subside prior to the next trial. The effect sizes observed in the open-label trials in each case were also extremely large (greater than 3.6), and much larger than the traditionally used nomenclature of Cohen (1988), which treats the largest category of effect size as greater than .5. These factors

verify that the protocol used in the present study was appropriate for testing the sample of IEI-EMF individuals.

While all three participants displayed an increased symptom severity and were confident that they could detect the presence of RF-EMF in the RF-ON exposure condition compared to RF-OFF in the initial open-label trial, no significant differences in symptom severity or exposure detection between the RF-ON and sham conditions were found in the double-blind trials. These findings correspond to those reported by Eltiti, Wallace, Ridgewell, et al. (2007) and Wallace et al. (2012), who found that IEI-EMF participants had a greater symptomatic response in an initial open-label active trial compared to sham, but no difference in subsequent double-blind trials. Likewise, in a study similar to the present investigation, van Moorselaar et al. (2017) reported that IEI-EMF participants were unable to correctly identify when they were being exposed during double-blind testing, despite participants reacting to the exposure in an initial un-blinded test. Generally, the results of the present experiment agree with the majority of previous studies, which have not found any relationship between IEI-EMF symptoms and EMF exposure in double-blind provocation paradigms (Rubin et al., 2005; Rubin et al., 2010).

Interestingly, belief of exposure was found to significantly predict symptom severity, with belief accounting for 83, 89 and 77 percent of the variance for Participants 1, 2 and 3 respectively. This may explain why a sham exposure is sufficient to trigger symptoms, as has been reported previously (Nam et al., 2009; Oftedal et al., 2007; Wilén et al., 2006). The strength of belief was particularly noteworthy in Participant 3, who reported that the experiment was designed with a deception element. As a result the participant reported detecting RF exposure in the post-exposure interval of the double-blind trials,

despite specific instruction from both the participant information sheet and the researchers throughout the trial that the RF exposure was switched off during the post-exposure interval.

Although varied, each participant also scored lower than the general population in terms of overall quality of life and other measures of health on the WHO-QOL BREF questionnaire. This is consistent with the conclusions of many cross-sectional survey studies (Hagström et al., 2013; Johansson et al., 2010; Kato & Johansson, 2012), and highlights that, in addition to physical impairment, IEI-EMF can significantly impact daily functioning and quality of life. This emphasises the importance of developing appropriate treatments and support for these individuals, but given the strong belief within the IEI-EMF community that EMF is a cause of their symptoms, this will remain challenging.

The results of the present study are limited by a number of factors. First, the results of the study cannot be generalised across the entire IEI-EMF population due the relatively small sample size. Despite intending to recruit a larger sample, it seems that scepticism of the scientific process and of the results of previous studies, as well as warnings about the present study from IEI-EMF advocacy groups (Stop Smart Meters Australia, 2015), may have led to many IEI-EMF sufferers being persuaded not to participate. Nevertheless, the idiographic nature of the study protocol and the 6 RF-ON and 6 RF-OFF comparisons were designed to enable the detection of partial IEI-EMF responses within each individual case separately. Second, the exposure device used a simulated RF signal in the 902-928 MHz ISM band which, although digitally modulated like Wi-Fi and 3G and 4G signals, would not be typically reported as being the associated trigger of symptoms by individuals who experience IEI-EMF as it is a signal band

reserved for industrial, scientific and medical use. The use of simulated signals in provocation studies has been criticised (Panagopoulos, Johansson, & Carlo, 2015), however, as all 3 participants responded to the active signal in the initial non-blind trial, this does not seem to be an issue. Finally, the present study is unable to comment on individuals who report more chronic forms of IEI-EMF, as it was unable to assess individuals who report more-prolonged symptoms that some IEI-EMF individuals report to result from the build-up of exposure from a variety of EMF sources over time [Hocking, 1998; Röösli et al., 2004].

Despite accounting for a number of possible limitations of IEI-EMF provocation studies to date, the results of the case studies presented here fail to demonstrate that the symptomatic response of self-reported IEI-EMF participants is affected by EMF exposure, nor that they can detect the presence of RF-EMF emissions at greater than chance levels. As in other studies, our results also support an alternative hypothesis for the aetiology of IEI-EMF; that the symptoms experienced are the result of a nocebo response. Indeed the size of resultant r-squared values shows that symptoms are more closely related to belief than EMF itself. Given the increasing prevalence of distressing and debilitating IEI-EMF symptoms in the general public, there is a great need to better understand the triggers for eliciting a harmful EMF nocebo response. Public messaging on the EMF topic by scientists and health administrators are no doubt significant influences (Wiedemann, Boerner, & Repacholi, 2014; Wiedemann et al., 2013). A nocebo aetiology hypothesis also provides useful direction in developing effective treatments for people who experience IEI-EMF, whose only current solutions for minimizing symptoms involve exposure reduction strategies or the complete avoidance of all perceived exposures of EMF. Often these remedies not only involve considerable

financial cost, but they can also have major implications for social and occupational functioning. Unfortunately, the ongoing debate over the aetiology of IEI-EMF continues to limit the development of appropriate treatments and support of people who experience IEI-EMF.

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4. CHAPTER 4: CAN EXPLICIT SUGGESTIONS ABOUT THE HARMFULNESS OF EMF EXPOSURE EXACERBATE A NOCEBO RESPONSE IN HEALTHY CONTROLS?

4.1 Chapter Foreword

Despite accounting for a number of potential methodological limitations, the results of the case studies presented in Study 2 (Chapter 3) failed to demonstrate that the symptomatic response of self-reported IEI-EMF sufferers is affected by EMF exposure, nor that IEI-EMF sufferers could detect the presence of RF-EMF emissions at greater than chance levels. In line with previous research, the results also showed that the symptoms reported by IEI-EMF sufferers are more closely related to a nocebo response, as a significant relationship between a participant's belief that they were being exposed (irrespective of the actual exposure condition) and their symptomatic response was observed in each case. As many of the methodological concerns raised by some researchers and IEI-EMF sufferers were adequately accounted for in Study 2, these results also suggest that the overall lack of evidence for a relationship between EMF exposure and symptoms in the extant literature was not a result of potential methodological issues.

While the results of Study 2 provide further support for the psychogenic theory of IEI-EMF, whether the nocebo response exhibited by IEI-EMF sufferers is specific only to IEI-EMF sufferers has not yet been determined. If healthy people were found to exhibit a similar response to those who suffer from IEI-EMF during a perceived threatening exposure to EMF, this would suggest that the nocebo response exhibited by IEI-EMF sufferers may be a normal human response. In addition to this, the factors which contribute to such a response have not been adequately clarified. Although there has

been some suggestion that alarmist media reports and precautionary information can negatively influence people's beliefs about EMF exposure, whether the negative beliefs induced by such information can result in a symptomatic nocebo response following a perceived exposure to EMF has not been sufficiently determined.

The study presented in this chapter (Study 3) investigates whether symptomatic nocebo effects can occur in healthy participants, and whether explicit suggestions about the adverse effects of EMF can exacerbate a nocebo response. This research will not only clarify the role of the nocebo effect in the development of symptoms attributed to EMF, but will also examine the factors which may underlie such a response, and will determine whether alarmist media reports may contribute to such a response. The findings from this study may have important implications for the development of effective treatments for IEI-EMF sufferers, as it may help to remove the stigma attached to the notion that the condition has a psychogenic origin, which may currently be a barrier to seeking treatment. In addition to this, the findings of the current study may also help to identify some of the factors which contribute to nocebo responses. This chapter has been published in the journal *Environmental Research.*

Verrender, A., Loughran, S.P., Dalecki, A., Freudenstein, F., & Croft, R.J. (2018). Can explicit suggestions about the harmfulness of EMF exposure exacerbate a nocebo response in healthy controls? *Environmental Research, 166,* 409 – 417

4.2 Abstract

While there has been consistent evidence that symptoms reported by individuals who suffer from Idiopathic Environmental Intolerance attributed to Electromagnetic Fields (IEI-EMF) are not caused by EMF and are more closely associated with a nocebo effect, whether this response is specific to IEI-EMF sufferers and what triggers it, remains unclear. The present experiment tested whether perceived EMF exposure could elicit symptoms in healthy participants, and whether viewing an 'alarmist' video could exacerbate a nocebo response. Participants were randomly assigned to watch either an alarmist (N = 22) or control video (N = 22) before completing a series of sham and active radiofrequency (RF) EMF exposure provocation trials (2 open-label, followed by 12 randomised, double-blind, counterbalanced trials). Pre- and post-video state anxiety and risk perception, as well as belief of exposure and symptom ratings during the open-label and double-blind provocation trials, were assessed. Symptoms were higher in the open-label RF-ON than RF-OFF trial (p < .001). No difference in either symptoms (p = .183) or belief of exposure (p = .144) was observed in the double-blind trials. Participants who viewed the alarmist video had a significant increase in symptoms (p = .041), state anxiety (p <.01) and risk perception (p < .001) relative to the control group. These results reveal the crucial role of awareness and belief in the presentation of symptoms during perceived exposure to EMF, showing that healthy participants exhibit a nocebo response, and that alarmist media reports emphasizing adverse effects of EMF also contribute to a nocebo response.

4.3 Introduction

The public's perception of the potential health implications associated with the use of modern technologies has been steadily changing in recent years (Petrie et al., 2001; Petrie & Wessely, 2002). This is often reflected in the mainstream media, where news reports consistently suggest that there are dangers of various aspects of modern life while often neglecting more mundane causes of illness (Frost, Frank, & Maibach, 1997; Petrie & Wessely, 2002). Generally, these stories do not reflect the current state of science (Claassen, Smid, Woudenberg, & Timmermans, 2012; Eldridge-Thomas & Rubin, 2013), but instead focus on reports of members of the community who claim to experience conditions characterised by a variety of adverse symptoms which they ascribe to their use of, or proximity to, various environmental stimuli, including vaccinations, genetically modified food, infrasound from wind turbines and electromagnetic fields (EMF) emitted by mobile phone and wireless technologies (Petrie & Wessely, 2002).

One particularly prominent condition is Idiopathic Environmental Intolerance attributed to Electromagnetic Fields (IEI-EMF). People who suffer from this condition typically report experiencing a diverse range of non-specific symptoms which they attribute to their exposure to the EMF emitted by everyday electrical and wireless technologies and infrastructure (Baliatsas, Van Kamp, Lebret, & Rubin, 2012; Röösli, Moser, Baldinini, Meier, & Braun-Fahrländer, 2004). Yet, while a considerable proportion of the population report experiencing IEI-EMF (estimated to be between 1.5 – 13.5% (Baliatsas et al., 2015; Blettner et al., 2009; Eltiti, Wallace, Zougkou, et al., 2007; Hillert, Berglind, Arnetz, & Bellander, 2002; Levallois, Neutra, Lee, & Hristova, 2002; Schreier, Huss, & Röösli, 2006; Schröttner & Leitgeb, 2008; Tseng, Lin, & Cheng,

2011)), there has been no robust evidence to implicate a bioelectromagnetic mechanism in producing the reported symptoms (Health Canada, 2015; Health Council of the Netherlands, 2009; Röösli, Frei, Mohler, & Hug, 2010; Rubin, Das Munshi, & Wessely, 2005; Rubin, Nieto-Hernandez, & Wessely, 2010; Scientific Committee on Emerging and Newly Idenified Health Risks, 2015; Staudenmayer, Binkley, Leznoff, & Phillips, 2003). For instance, when tested under double-blind protocols, IEI-EMF participants do not report an increase in symptoms to EMF and are unable to perceive the difference between active and sham exposures (Röösli et al., 2010; Rubin et al., 2010). Instead, the evidence suggests that the condition is more closely associated with a nocebo response, as awareness of the exposure and a belief of being exposed have been shown to play an important role in the presentation of the condition. For example, a number of studies have found that participants experience an increase in symptoms when they are aware of the active exposure condition in an initial non-blinded trial compared to sham, but do not exhibit more symptoms in active than sham exposures in subsequent double-blind trials (Eltiti, Wallace, Ridgewell, et al., 2007; van Moorselaar et al., 2017; Verrender et al., 2018). Furthermore, sham exposures (i.e. with no EMF) have been shown to be sufficient to trigger symptoms in IEI-EMF participants (Nam et al., 2009; Oftedal, Straume, Johnsson, & Stovner, 2007; Verrender et al., 2018; Wilén, Johansson, Sandström, Kalezic, & Lyskov, 2006). The exact role of the nocebo response in the development of IEI-EMF, however, is not fully understood. For instance, recent findings from a qualitative study suggest that instead of the condition originating from a nocebo response, IEI-EMF individuals may be using the notion of sensitivity to EMF to provide a narrative to explain their pre-existing medically unexplained symptoms, in an effort to make their condition more practically and emotionally manageable Dieudonné (2016). Yet, it is important to note that Dieudonné (2016) did not test the cause of the

participant's symptoms, but rather, retrospectively asked participants about their beliefs regarding the cause of their symptoms. As retrospective self-reports are known to suffer from recall bias (Baliatsas et al., 2015; Vrijheid et al., 2009), these methods are not able to determine symptom aetiology.

Given the prevalence of distressing and debilitating IEI-EMF symptoms, and in light of the evidence suggesting that such symptoms may be the result of a nocebo response, there is a great need to better understand the triggers that elicit such responses. Generally, a nocebo response occurs when conscious or subconscious negative expectations trigger or exacerbate adverse symptoms in response to an exposure that is not known to cause those effects (Bräscher, Kleinböhl, Hölzl, & Becker, 2017; Hahn, 1997). These expectations may be induced by explicit suggestions about the potential effects of an exposure (Benedetti, Lanotte, Lopiano, & Colloca, 2007; Webster, Weinman, & Rubin, 2016) or by learning through classical conditioning (Bräscher, Kleinböhl, et al., 2017).

The communication of information about potential adverse health effects associated with EMF exposure constitutes an explicit suggestion which may be responsible for the formation of negative expectations and consequent nocebo response seen in IEI-EMF individuals (Webster et al., 2016). For example, there has been consistent evidence that precautionary information can negatively influence beliefs about EMF exposure, despite this information originally being intended to reassure the public (Barnett, Timotijevic, Shepherd, & Senior, 2007; Nielsen et al., 2010; Wiedemann, Boerner, & Repacholi, 2014; Wiedemann et al., 2013; Wiedemann & Schütz, 2005; Wiedemann, Thalmann, Grutsch, & Schütz, 2006). Similarly, viewing mainstream media reports which either promote the view that EMF exposure is hazardous, or focus on individuals with IEI-

EMF, have been shown to increase worries about EMF exposure (Witthöft et al., 2017), while viewing an advertisement claiming to protect against the 'harmful effects of everyday EMF exposure' has been shown to increase both heart rate and concern about EMF (Köteles, Tarján, & Berkes, 2016). Further, recent content analyses have shown that mainstream media reports about EMF exposure often misrepresent the current state of scientific evidence by focusing on an electromagnetic cause for IEI-EMF, or suggesting a relationship between EMF exposure and ill-health (Claassen et al., 2012; Eldridge-Thomas & Rubin, 2013). If such misinformation is being distributed on a wide scale and is negatively influencing people's beliefs about EMF exposure, it is possible that this may be a contributing factor to the prevalence of IEI-EMF.

Yet, it remains unclear whether the negative beliefs induced by such communications can result in greater symptom formation following a perceived exposure to EMF. Although Szemerszky, Köteles, Lihi, and Bárdos (2010) demonstrated that suggestions about the strength of EMF exposure can lead to increased symptom scores and an increase in the belief that a sham magnetic field was active, that study did not assess the effect of explicit suggestions of risk from EMF exposure (which may induce negative expectations) and was limited by a lack of counterbalancing. Furthermore, while Witthöft and Rubin (2013) reported that viewing a sensationalist media report about the adverse effects of Wi-Fi can increase the likelihood of a person experiencing symptoms following a sham exposure and developing an apparent sensitivity to EMF, the effect was only found for those with high pre-existing levels of state anxiety. This may be because the study lacked a verified non-exposure condition, potentially resulting in insufficient statistical power to detect effects in non-anxious individuals. In support of this notion, a similar study which included a cued non-exposure condition found that

those who watched a film focusing on 'adverse effects of Wi-Fi' perceived tactile electrical stimuli as more intense during a cued Wi-Fi exposure (which was actually a sham exposure) compared to a cued no Wi-Fi condition, and that the effect was not mediated by anxiety (Bräscher, Raymaekers, Van den Bergh, & Witthöft, 2017). This suggests that manipulating a participant's belief of exposure via cues may be important for influencing symptom perception irrespective of pre-existing state anxiety levels. The latter study, however, assessed somatosensory perception rather than symptom perception, and so it remains uncertain as to whether negative beliefs induced by information about EMF exposure can result in greater symptom formation or belief regarding exposure status following a perceived exposure to EMF.

To address these limitations, the present study was designed to determine whether perceived EMF exposure could elicit symptoms in a healthy population, and additionally, whether messages emphasizing 'adverse health effects of EMF exposure' can exacerbate a nocebo response. The study was also designed to explore, within-subjects, whether there is a relationship between a person's belief of exposure and symptoms, and whether there is a difference in symptom response between participants with low, medium and high pre-existing levels of state anxiety. To this end, an initial non-blinded open-label trial was employed, where the status of exposures emanating from the device (during an active and sham condition) were visually demonstrated to each participant using an EMF meter.

4.4 Materials and Methods

4.4.1 Participants

Forty-four participants aged 18 – 30 years (M = 21.92, SD = 4.88; 50% male) were recruited through advertisements placed online and around the University of

Wollongong campus. A power calculation conducted in G*Power 3.0 (Faul, Erdfelder, & Buchner, 2007) for an independent samples *t* test based on an effect size of 0.8, an alpha level of .05 and a power of 0.80 recommended a total sample size of 42.

All participants were first screened via a telephone interview to confirm eligibility for the study. To be included in the study, participants were required to be over the age of 18 and report being of good health. Participants were excluded from the study if they reported having a current illness or medical condition, or having used illicit substances within the 7-day period prior to the study. Suitable participants were required to attend the Illawarra Health and Medical Research Institute for one mutually convenient testing session. The study was approved by the Human Research Ethics Committee (HE: 2016/981). All participants were instructed to abstain from alcohol for at least 8 hr, caffeine for at least 1 hr, and mobile phone use for at least 2 hr before the beginning of the testing session. Participants were compensated with a monetary gift card for their involvement in the study.

4.4.2 Radiofrequency exposure

RF exposure was generated using a portable, self-contained, battery-operated device (Two Fields Consulting, St Kilda, Australia). The RF device was placed 30 cm to the left side of the participant (at approximately shoulder to head height) on a hard surface. The main exposure from the device was a spread spectrum RF signal in the 902-928 MHz ISM band which was digitally modulated in a similar manner to signals from Wi-Fi and 3G/4G mobile phones. The RF signal was generated by a commercial RF modem which emitted a frequency hopping spread spectrum signal with an average radiated power output of 1 W for 10 min (RF-ON), or was completely EMF silent (RF-OFF, sham trials). The incident RF exposure level from the side of the device facing the

participant was measured using a calibrated broadband instrument with an uncertainty of ± 2.4 dB for a two-sided coverage interval and a coverage factor of 2 (Narda EMR 300 meter and Type 9 E-field probe, Narda Safety Test Solutions, Hauppauge, NY), and was found to be 0.3 W/m². This RF exposure level is below the power density reference level limit of 4.6 W/m² specified for the Australian general public (ARPANSA RPS3) and by the International Commission on Non-Ionizing Radiation Protection (ICNIRP, 1998). It is important to note that the maximum localised specific absorption rate (SAR) from the exposure device used in the present study is less than that induced from personal mobile phone use (held against the ear in the active talk mode) due to the greater separation distance. Conversely, the whole body averaged SAR and localised SAR of the device are greater than those normally produced by Wi-Fi and mobile phone base station signals. The device was fully enclosed in a thermally insulated case and coded inputs were used to maintain double-blinding. The device contained an independent RF monitor to check the status of the RF transmitter and each use of the device was logged using internal memory.

4.4.3 Questionnaires

4.4.3.1 Symptoms and exposure status scale (SESS)

During the provocation trials, participants were asked to indicate whether they believed the exposure was on or off, and to rate whether they were experiencing any symptoms via pen and paper 100 mm visual analogue scales. To assess belief of exposure, participants were asked "how sure are you of the current exposure status *right now*?" anchored with the terms 'Definitely OFF' and 'Definitely ON'. To assess symptom experience, a modified state version of the 34 item Checklist for Symptoms in Daily Life (Wientjes & Grossman, 1994; Witthöft & Rubin, 2013) was used. Participants were

asked “how strong/unpleasant are the following symptoms *right now?”* anchored with the terms ‘Barely Detectable’ and ‘Maximum Severity’. These response categories differed from the original questionnaire (Wientjes & Grossman, 1994) and were used in line with our previous study (Verrender et al., 2018). The symptom responses of the 34 items were added to calculate a total symptom score for each of the baseline and exposure intervals in each trial. The primary dependent variables for belief of exposure and symptoms in the provocation trials were calculated as difference scores between the baseline and exposure questionnaires (exposure interval minus preceding baseline) given during each trial (see procedure below); a difference score was used to minimise the influence of baseline variability and potential carry-over effects.

4.4.3.2 Risk perception questionnaire (RPQ)

A self-generated risk perception questionnaire comprising 4 questions was used to assess EMF risk perception. Question 1 assessed concerns about electromagnetic fields in general and question 2 assessed concerns about electromagnetic fields in relation to mobile phones and Wi-Fi. Participants were asked “How concerned are you about the potential health risks of electromagnetic fields in general?” rated on a 7-point Likert scale (1 = not worried at all, 7 = very worried) and “All in all, how threatened do you feel by electromagnetic radiation emissions from mobile phones and Wi-Fi?” rated on a 7-point Likert scale (1 = not threatening at all, 7 = very threatening). To enable standardised measurement of RF-EMF risk perception in relation to mobile phones and Wi-Fi, questions 3 and 4 used picture-guided scenarios which illustrated everyday exposure situations (Freudenstein, Wiedemann, & Brown, 2015). Participants were asked “How dangerous do you think the electromagnetic fields from mobile phones are while you talk on the phone, as illustrated in this picture?” and “How dangerous do you

think the electromagnetic fields are from Wi-Fi routers in close proximity, as illustrated in this picture?" rated on a 7-point Likert scale (1 = not dangerous at all, 7 = very dangerous). The RPQ score was defined as the mean score from all responses.

4.4.3.3 State and Trait Anxiety Index (STAI)

The 40 item version of the STAI (Spielberger, Gorsuch, & Lushene, 1970) was used to assess participant's state and trait anxiety. This comprises two, 20-item forms, assessing state (STAI-Y1) and trait (STAI-Y2) anxiety separately, with items answered on a 4-point Likert scale (1 = not at all, 4 = very much so). Low, medium and high anxiety were defined as being less than minus 1 standard deviation from the mean, between minus 1 standard deviation and plus 1 standard deviation from the mean, and greater than plus 1 standard deviation from the mean, respectively (Witthöft & Rubin, 2013).

4.4.3.4 NEO Five Factor Personality Index (NEO-FFI)

The 60 item NEO-FFI (Costa & McCrae, 1992) was used to assess personality traits: Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Openness to Experience. This measure is beyond the scope of this paper and will not be discussed further.

4.4.4 Design

In an experimental between-groups design, participants were assigned to the alarmist or control video group by a computerised random allocation process. In the alarmist video group, participants viewed a 3 min video appeal to the United Nations from a concerned scientist asking that more precautionary action be taken in regard to EMF exposure "from our favourite gadgets." This included statements about the potential health risks from mobile phone and Wi-Fi signals, as well as sensationalised images of exposure

scenarios (Blank, 2015). In the control group, participants viewed a 3 min segment of a documentary on gravity (Cox, 2013). This video contained no health related content. To minimise the influence of experimenter bias, a researcher not involved in data collection (AD) was responsible for the randomisation and administration of the videos.

For the provocation trials, a randomised, counterbalanced, cross-over design was employed. Each participant's symptoms and belief of exposure was tested under a series of 14 sham and active provocation trials. The first two trials were non-blinded, open-label trials (1 RF-OFF, 1 RF-ON), where both the participant and the researcher were aware of the exposure status. This was verified to the participant using a Nardalert S3 broadband monitor (Narda Safety Test Solutions, Hauppauge, NY). These trials were followed by a series of 12 double-blind, randomised, counterbalanced trials (6 sham, 6 RF-ON). Randomization and counterbalancing was achieved using Excel (randomization command), such that a sham and RF-ON condition were treated as a pair; the conditions for each pair were randomly allocated before assigning the next pair; and no more than three of the same pair-order were permitted.

4.4.5 Procedure

A participant information sheet was sent to people who responded to recruitment flyers. This informed participants that a small percentage of the population report being sensitive to EMF, described some of the symptoms reported by IEI-EMF sufferers and explained that although the scientific evidence has yet to establish a clear relationship between exposure and symptoms, news reports about the possible adverse health effects of RF exposure continue to focus on people who report these symptoms. The general aims of the study were also listed in the information sheet.

Following a telephone screening interview, suitable participants were booked in for one mutually convenient testing session starting at 09:00 am, which lasted approximately 5.5 hr. Upon arrival at the laboratory, participants provided informed written consent and were given a verbal briefing of the ensuing testing session. After being instructed to switch off and leave all electronic devices in a general area of the laboratory, participants were then seated comfortably inside a Faraday cage, where they completed the baseline (Time 1 (T1)) measures of the STAI and RPQ. After completing the T1 questionnaires, participants then watched one of the two videos (based on their randomly assigned group). To maximise attention, participants were instructed to pay attention to the video as they would be required to answer questions about the video as part of a memory test at the conclusion of the study (although no memory test was conducted). After watching the video, participants again completed the STAI and RPQ (Time 2 (T2)). The exposure device was then set up and the provocation trials commenced, beginning with the initial 2 open-label trials (1 RF-OFF, 1 RF-ON), followed by the 12 double-blind trials. Each of the provocation trials lasted 20 min, beginning with a 5 min baseline interval, followed by a 10 min exposure interval (RF-ON or RF-OFF/sham, depending on randomization and counterbalancing), and concluded with a 5 min rest interval before the onset of the next trial. In each trial, participants were required to complete the SESS 2.5 min into the baseline interval and again 7 min into the exposure interval. At the conclusion of the provocation trials, participants were led out of the Faraday cage and asked whether they had any questions or concerns about any aspect of the experiment. No participants reported any concern about the experiment. A 15 min break was given after the 8th provocation trial.

4.4.6 Statistical analyses

Statistical analyses were performed with SPSS Statistics for Windows, Version 23.0 (IBM, Armonk, New York). Where normality tests and visual inspection of the data revealed violations to the assumption of normality, non-parametric tests were employed and the corresponding effect sizes (*ES*) were calculated as *r*, (where 0.1 = small, 0.3 = medium and 0.5 = large (Cohen, 1988)). Where parametric tests were conducted, corresponding effect sizes were calculated as Cohen's *d*, (where 0.3 = small, 0.5 = medium and 0.8 = large (Cohen, 1988)).

4.4.6.1 Preliminary Analyses:

Independent samples *t* tests were used to compare pre-existing (T1) levels of state anxiety (STAI-Y1), trait anxiety (STAI-Y2) and risk perception (RPQ) between the control and alarmist video groups. A Wilcoxon Signed-Ranks test was used to determine whether participants understood the exposure protocol by assessing whether there was a difference in belief of exposure rating of the SESS between the RF-OFF and RF-ON open-label trials.

4.4.6.2 Hypothesis Driven Analyses:

A Wilcoxon Signed-Ranks test was used to determine whether there was a main effect of exposure on the symptom score of the SESS in the open-label trials. To assess whether there was an interaction between video group and symptom score, a symptom difference score (RF-ON minus RF-OFF) was calculated and a Mann-Whitney U test used to compare the difference score between control and alarmist video groups in the open-label trials.

4.4.6.3 Exploratory Analyses:

To verify whether there was no effect of RF-EMF exposure, Wilcoxon Signed-Ranks tests were used to determine whether there was a main effect of exposure on either the belief of exposure rating or the symptom score of the SESS in the double-blind trials. To assess whether there was an interaction between video group and either belief of exposure rating or symptom score, a difference score for each variable was calculated (RF-ON minus RF-OFF). These difference scores were calculated by averaging the belief of exposure, and separately the symptom difference scores (already calculated as the difference between the baseline and exposure intervals) of each variable across the 6 RF-ON and 6 RF-OFF conditions. The averaged RF-ON score was then subtracted from the averaged RF-OFF score. Mann-Whitney U tests were then used to compare each of these variables between the control and alarmist video groups.

Spearman's rho measure of association was used to test whether there was a relationship between belief of exposure and symptoms in the double-blind trials (irrespective of actual exposure condition) for each individual participant. The resultant rho values were then transformed using a Fisher transformation, and a one sample *t* test was used to determine whether these transformed correlations differed from 0. An independent samples *t* test was used to assess whether there was a difference in the Fisher transformed Spearman's rho values between the control and alarmist video groups.

Mann-Whitney U tests were used to assess whether the difference between T1 and T2 STAI-Y1 score, and separately RPQ score, differed between the control and alarmist video groups.

A Jonckheere-Terpstra test was used to determine whether there was a linear trend in the symptom score of the RF-ON open-label trial as a function of pre-existing state anxiety.

4.5 Results

4.5.1 Preliminary Analyses

The means, standard deviations and test statistics for assessing whether there were significant differences between the control and alarmist video groups in relation to pre-existing levels of state anxiety (STAI Y-1), trait anxiety (STAI – Y2) and risk perception (RPQ) are displayed in Table 4.1. No significant differences were detected. Verifying that participants understood the exposure protocol (they believed that they were being exposed in the open-label RF-ON condition and that they were not being exposed in the open-label RF-OFF condition), belief of exposure ratings were significantly higher in the RF-ON (*Median* = 100) compared to the RF-OFF (*Median* = 0) condition, $T = 0.00$, $z = -5.86$ (corrected for ties), N – ties = 44, $p < .001$, $ES = 0.88$. All participants correctly reported that they were confident that the exposure was 'Definitely ON' in the RF-ON condition and 'Definitely OFF' in the RF-OFF condition of the open-label trials.

Table 4.1: Descriptive statistics and tests for differences in pre-existing levels of state anxiety, trait anxiety and risk perception between the two video groups.

Dependent variable	Control video N = 22	Alarmist video N = 22	Test statistic for differences between groups
State Anxiety	*M* = 29.00, *SD* = 8.11	*M* = 30.45, *SD* = 9.96	*t*(42) = -0.639, *p* = .527, *ES* = 0.19
Trait Anxiety	*M* = 38.45, *SD* = 11.85	*M* = 36.41, *SD* = 9.79	*t*(42) = -0.624, *p* = .536, *ES* = 0.19
Risk Perception	*M* = 2.35, *SD* = 1.12	*M* = 2.51, *SD* = 1.09	*t*(42) = - 0.477, *p* = .636, *ES* = 0.14

M = mean; SD = standard deviation; ES = effect size.

4.5.2 Hypothesis Driven Analyses:

4.5.2.1 Symptoms in the open-label trials:

Figure 4.1 shows the SESS symptom scores for the RF-OFF and RF-ON open-label trials. Overall, participants had significantly higher increases in symptom scores in the RF-ON condition (*Median* = 17.00) compared to the RF-OFF condition (*Median* = -0.50), *T* = 77.00, *z* = -4.476 (corrected for ties), *N* - ties = 40, $p < .001$, *ES* = 0.71.

4.5.2.2 Effect of video group on symptoms in the open-label trials:

The symptom scores in the RF-OFF condition were equal between the alarmist (*Mean* = 11.59 *Median* = -3) and control (*Mean* = 11.45 *Median* = 0) video groups, validating the comparison of symptom difference scores between the two groups. Figure 4.2 shows the symptom difference score (RF-ON – RF-OFF) for the control and alarmist video groups

in the open-label trials. The symptom difference score was higher in the alarmist (*Median* = 25.50) compared to the control (*Median* = 5.00) video group, and the interaction between symptom difference score and video group was significant, U = 159.50, z = -1.738, p = .041 (one-tailed), ES = 0.26[3].

[3] One significant outlier was removed from this analysis. The interaction between symptom difference score and video group only reached trend level when including this outlier, U = 181.50, z = -1.421, p = .078 (one-tailed), ES = 0.24.

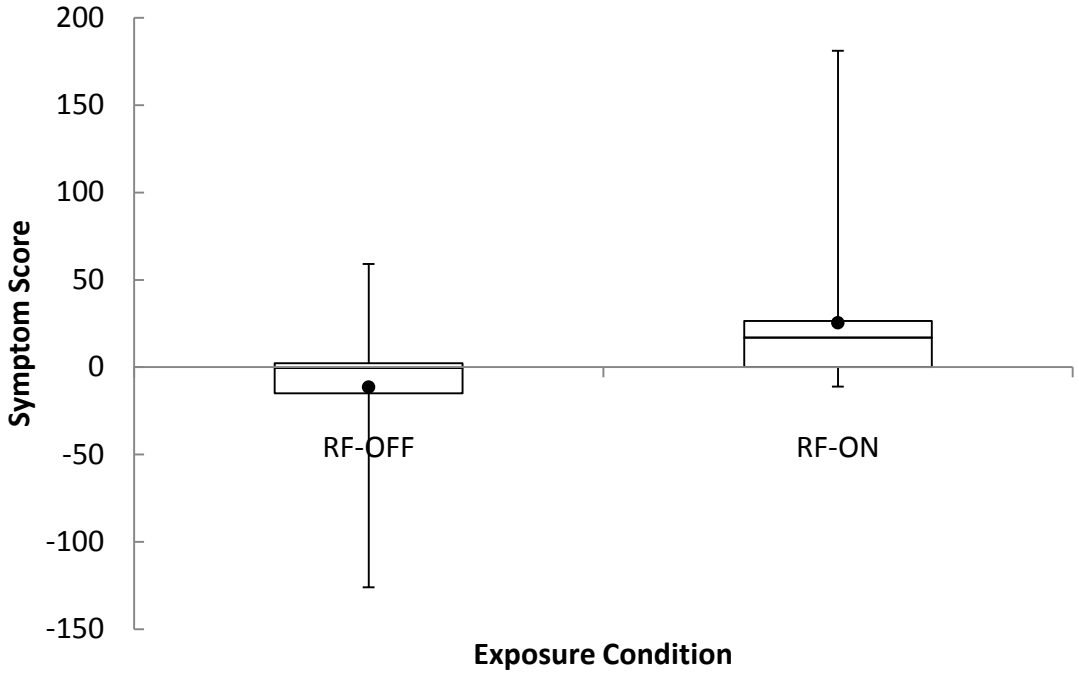

Figure 4.1 The mean (dot), median (line), interquartile range (box) and range (whiskers) of the SESS symptom scores are shown for the RF-OFF and RF-ON open-label trials.

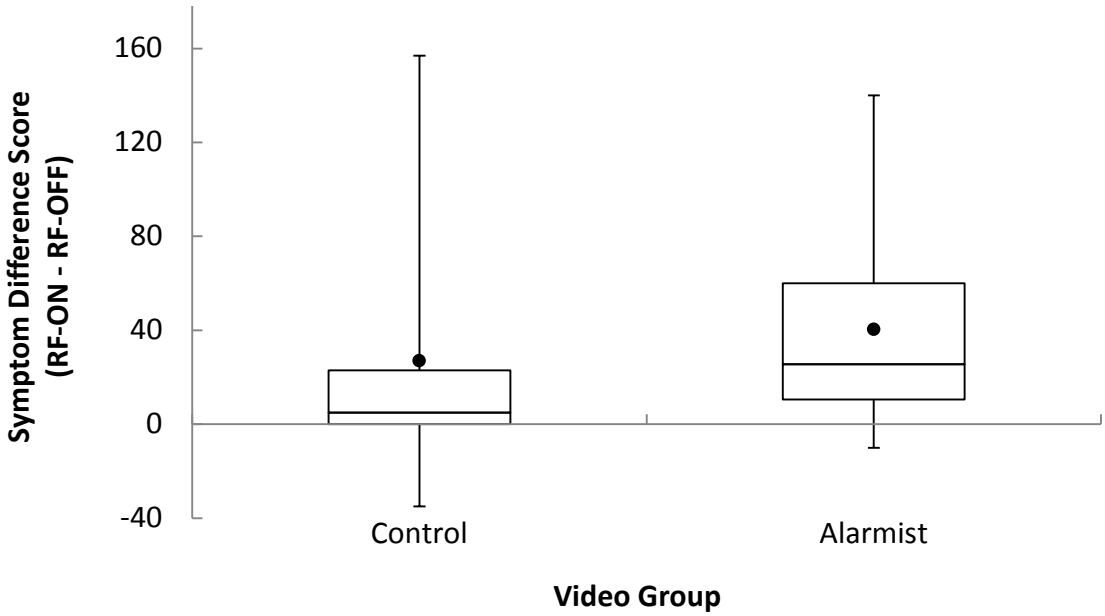

Figure 4.2: The mean (dot), median (line), interquartile range (box) and range (whiskers) of the difference in SESS symptom score (RF-ON – RF-OFF) are shown as a function of video group in the open-label trials[3].

4.5.3 Exploratory Analyses

4.5.3.1 Effect of exposure on belief of exposure and symptoms in the double-blind trials: The SESS belief of exposure ratings in the RF-ON and sham double-blind trials are shown in Figure 4.3. Overall, there was no difference in belief of exposure rating between the RF-ON (*Median* = 34.58) and sham conditions (*Median* = 38.33), T = 331.00, z = -1.062 (corrected for ties), N – ties = 40, p = .144 (one-tailed), ES = 0.17, indicating that there was no main effect of exposure on belief of exposure rating. Three participants correctly identified at greater than chance levels (within-subjects), when they were and were not being exposed in the double-blind trials, with 1 participant getting 75% correct and 2 participants getting 83% correct. No other participants could correctly identify when they were being exposed. Given a chance level of 5% and that there were 44 participants, it would be expected that 2.2 participants would correctly identify the conditions by chance.

The SESS symptom scores in the RF-ON and sham double-blind trials are shown in Figure 4.4. Overall, there was no difference in symptom score between the RF-ON (*Median* = 10.33) and sham conditions (*Median* = 10.33), T = 398.00, z = -0.906 (corrected for ties), N – ties = 43, p = .183 (one-tailed), ES = 0.14, indicating that there was no main effect of exposure on symptoms.

Spearman's rho measure of association showed that the relationships between belief of exposure and symptoms in the double-blind trials were highly variable between participants. These values ranged from -.276 to .882 in the control video group and -.675 to .852 in the alarmist video group. Following a Fisher transformation, a one-sample t test found that the transformed rho values were significantly greater than 0, $t(43) = 6.862$, $p < .001$, ES = 1.03.

4.5.3.2 Effect of video group on belief of exposure and symptoms in the double-blind trials:

The belief of exposure difference score (RF-ON – Sham) also not differ between the control (*Median* = -0.33) and alarmist (*Median* = 4.25) video groups, $U = 186.50$, $z = -1.303$, $p = .096$ (one-tailed), $ES = 0.20$, indicating that there was no interaction between video group and belief of exposure rating. The symptom difference score (RF-ON – Sham) also did not differ between the control (*Median* = -1.25) and alarmist (*Median* = 1.92) video groups, $U = 218.00$, $z = -0.563$, $p = .287$ (one-tailed), $ES = 0.08$, indicating that there was no interaction between video group and symptoms.

4.5.3.3 Effect of video on state anxiety and risk perception:

The difference in state anxiety (STAI-Y1) from T1 to T2 was significantly higher in the alarmist (*Median* = 3.50) compared to the control (*Median* = -.50) video group, $U = 135.50$, $z = -2.505$, $p < .01$ (one-tailed), $ES = 0.38$. The difference in risk perception (RPQ) from T1 to T2 was also significantly higher in the alarmist (*Median* = 1.00) compared to the control (*Median* = 0.00) video group, $U = 75.50$, $z = -3.946$, $p < .001$ (one-tailed), $ES = 0.60$.

4.5.3.4 Relationship between belief of exposure and symptoms in double-blind trials as a function of video group:

The relationship between belief of exposure and symptoms (Fisher transformed Spearman's rho values) did not differ between the control ($M = .47$, $SD = .48$) and alarmist ($M = .54$, $SD = .50$) video groups, $t(42) = -.443$, $p = .660$, $ES = 0.07$.

4.5.3.5 Relationship between pre-existing state anxiety and symptoms in open-label trials:

Figure 4.5 shows the SESS symptom scores as a function of anxiety group. No significant trend between symptom scores and higher levels of pre-existing anxiety was detected, $J = 210.00$, $z = -.838$, $p = .402$, $ES = 0.13$.

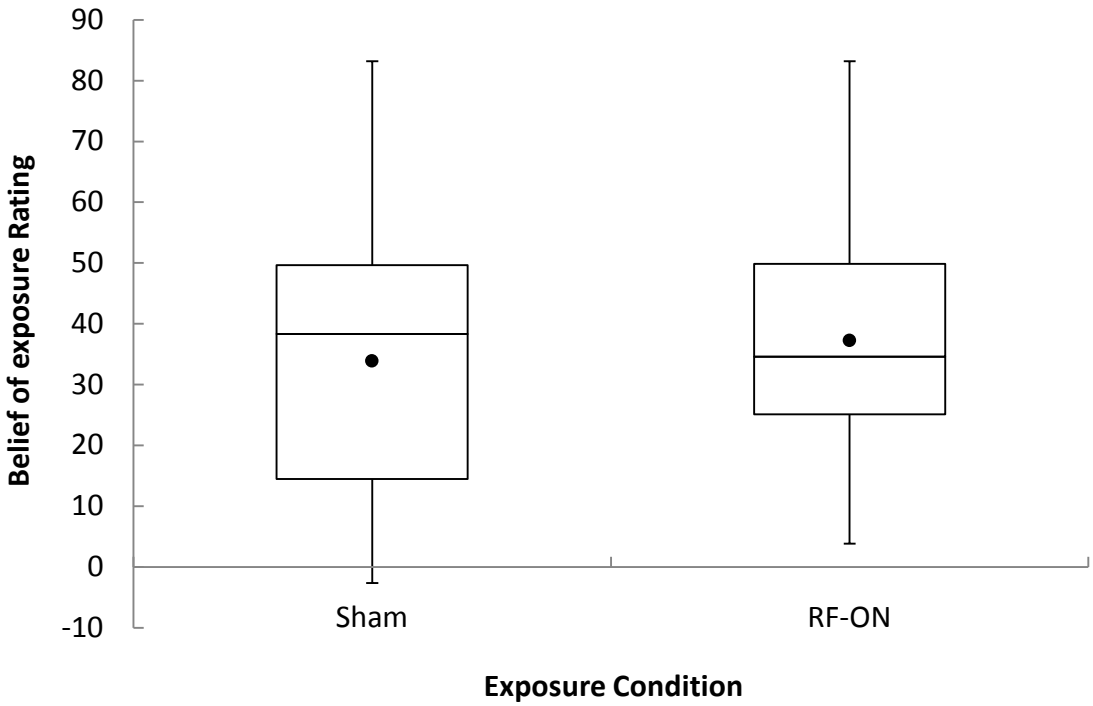

Figure 4.3: The mean (dot), median (line), interquartile range (box) and range (whiskers) of the SESS belief of exposure rating are shown for the sham and RF-ON double-blind trials.

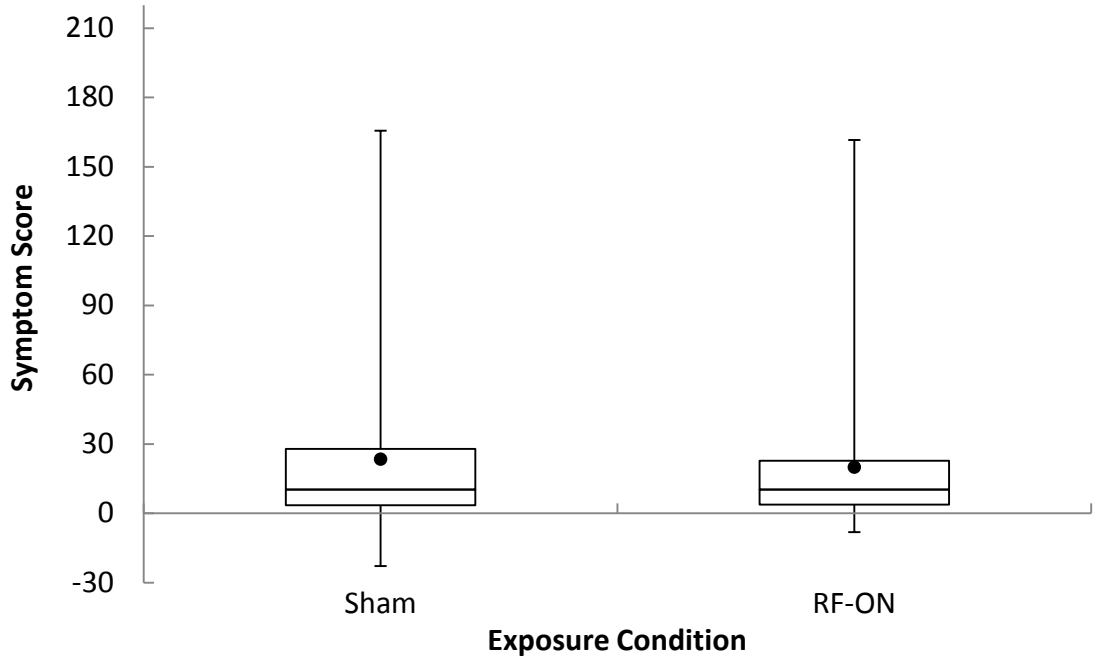

Figure 4.4: The mean (dot), median (line), interquartile range (box) and range (whiskers) of the SESS symptom score are shown for the sham and RF-ON double-blind trials.

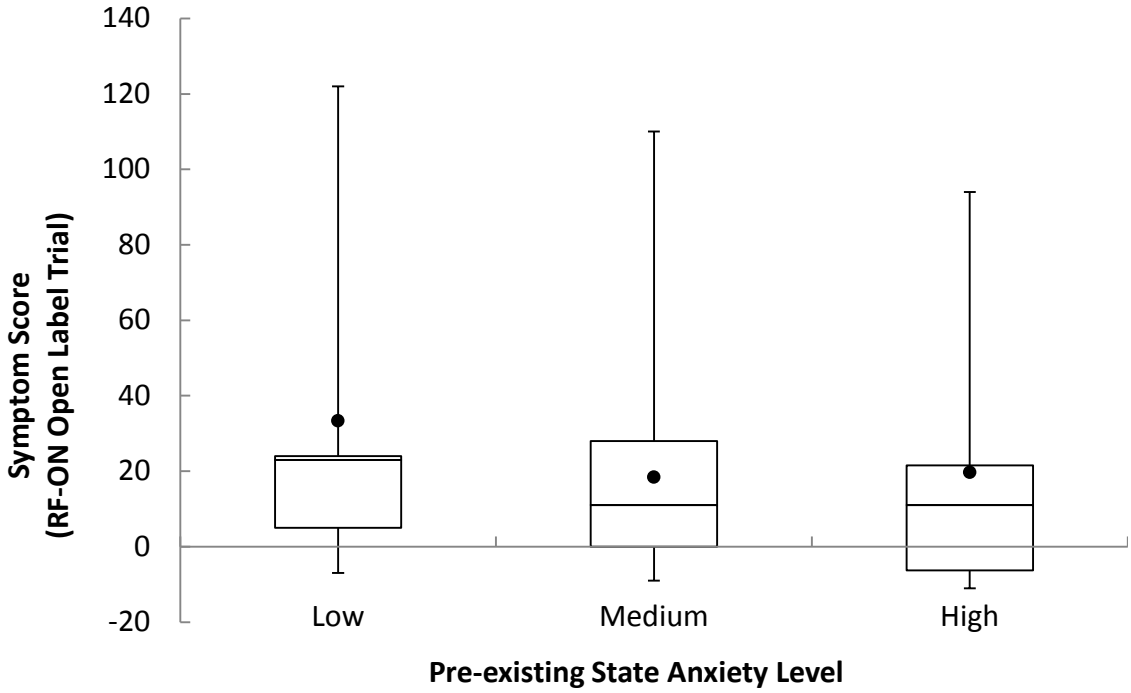

Figure 4.5: The mean (dot), median (line), interquartile range (box) and range (whiskers) of the SESS symptom scores in the RF-ON open-label trial are shown as a function of pre-existing state anxiety level.

4.6 Discussion

While there has been growing evidence that the symptoms reported by IEI-EMF sufferers are likely the result of a nocebo effect (Eltiti, Wallace, Ridgewell, et al., 2007; Nam et al., 2009; Oftedal et al., 2007; van Moorselaar et al., 2017; Verrender et al., 2018; Wilén et al., 2006), there has been limited understanding of the factors which contribute to such a response. Although a number of studies have shown that explicit suggestions about the adverse effects of EMF exposure can increase concern and negatively influence people's beliefs about EMF exposure (Barnett et al., 2007; Köteles et al., 2016; Nielsen et al., 2010; Wiedemann et al., 2014; Wiedemann et al., 2013; Wiedemann & Schütz, 2005; Wiedemann et al., 2006; Witthöft et al., 2017) (factors which are considered to be key in contributing to a nocebo response (Webster et al., 2016)), it has remained relatively unclear whether the negative beliefs induced by such communications can result in greater symptom formation following a perceived exposure to EMF. The purpose of this experiment was to investigate whether perceived EMF exposure would elicit symptoms in a healthy population and to assess whether messages that emphasise 'adverse health effects of EMF exposure' can induce a nocebo response, including for those without high pre-existing levels of state anxiety. In the provocation trials, both active and sham EMF exposures were first demonstrated to participants in an initial non-blinded, open-label trial, before a series of double-blind, randomised, counterbalanced trials were conducted. By demonstrating that the experiment contained a 'no exposure' condition, the present study was able to more clearly determine whether any observed increases in symptoms were the result of a nocebo effect.

A number of preliminary tests were first used to establish whether the experiment was valid and whether the experimental manipulation had worked. These checks demonstrated that there were no differences in pre-existing levels of state anxiety, trait anxiety and EMF risk perception between the alarmist and control video groups. Further, these tests verified that participants understood the exposure protocol, as they correctly indicated that they were being exposed in the RF-ON open-label trial and not being exposed in the RF-OFF open-label trial.

The results of the provocation trials revealed the crucial role of awareness and belief in the presentation of symptoms during perceived exposure to EMF. In the open-label trials, participants reported higher symptom scores in the RF-ON trial compared to the RF-OFF trial. In the subsequent double-blind trials, however, there was no difference in either belief of exposure or symptom scores between the RF-ON and sham conditions. These findings demonstrate that knowledge and/or awareness of the exposure condition was essential for producing an effect on symptoms. In addition to this, belief of exposure was found to be positively associated with higher symptom scores in the double-blind trials, giving further indication that a nocebo effect, rather than EMF exposure itself, was responsible for the increase in symptoms. In regards to the effect of messages which emphasise the 'adverse health effects of EMF exposure', the present study found that participants who viewed the alarmist video had higher symptom scores in the open-label trials than participants who viewed the control video. While, in contrast to Witthöft and Rubin (2013), the present study found that the effect of the video on symptom score was not moderated by pre-existing levels of state anxiety, it is important to note that the effect of the video on symptom score in the present study was only trend level when including an outlier in the sample. This may indicate that the

effect of the video was strongly influenced by the individuals in the study. This corresponds to the notion that both situational factors (such as viewing a particular media report) and dispositional factors (such as personality traits) interact to influence people's worries about the potential health hazards of modern life, though further research is required to clarify the personality traits which may be involved in moderating this effect (Witthöft et al., 2017). It is important to note, however, that sample size may also play a considerable role in the interaction between the type of video viewed and symptom score, and a larger sample size would have increased the chance of identifying an effect of the video in the present study. In line with previous research, the present study also found that participants who viewed the alarmist video had a larger increase in state anxiety and risk perception from baseline relative to those who viewed the control video (Bräscher, Raymaekers, et al., 2017; Witthöft et al., 2017; Witthöft & Rubin, 2013). This provides further support to the notion that sensationalised media reports are capable of increasing people's concerns and worries about exposure to EMF. Nonetheless, it is also possible that pre-existing beliefs about the relative harmfulness of EMF exposure may have influenced the results of this study via a ceiling effect. Future studies could usefully address this issue by using pre-screening to allocate participants into "high" and "low" risk perception groups, within each of the control and alarmist video groups.

Overall, the results of the present study corroborate those of IEI-EMF provocation studies, and demonstrate that the belief of being exposed, rather than EMF exposure itself, is sufficient to trigger symptoms in healthy participants, including those without high pre-existing levels of anxiety. This is supported by the fact that 77% of participants reported higher symptoms in the open-label RF-ON trial compared to the RF-OFF trial,

while no difference in symptom score was detected in the double-blind trials. Although the size of the effect on symptoms in the open-label trials in the present study was not as large as the effect observed for IEI-EMF sufferers in a previous study (*ES* > 3.6) (Verrender et al., 2018), the effect observed in the present study was still quite large (*ES* = 0.71), and is larger than the traditionally used nomenclature of Cohen (1988) (who treats the largest category of effect size as >.05). This may indicate that the nocebo response displayed by IEI-EMF sufferers during a perceived exposure situation is a normal human response. In addition to this, the results of the present study not only support those of previous studies suggesting that sensationalist media reports about perceived environmental hazards can raise concerns and negative beliefs about EMF, but also demonstrate that such reports may be contributing to a symptomatic nocebo response. This is analogous to the conclusions reached by studies investigating whether media health warnings can influence symptom expectations after exposure to infrasound from wind turbines or chemical pollution (Crichton, Dodd, Schmid, Gamble, & Petrie, 2014; Winters et al., 2003) and further emphasises the importance of disseminating accurate scientific and health information in order to reduce the likelihood of symptomatic nocebo responses in the community more generally.

A number of potential limitations should be considered when interpreting the results of the present study. First, as the sample was mainly comprised of a relatively young healthy population, the present study is unable to comment on whether similar effects of alarmist media would be observed in a more general population sample. While online advertisements were used in an attempt to attract greater interest in the study, future studies could address this issue by using local newspapers and radio stations to recruit more broadly from the community. Second, as the RF-OFF condition always preceded

the RF-ON condition in the open-label trials, the possibility that part of the increase in symptoms in the open-label trials (independent of the media content) was due to the elapsed study time cannot be completely ruled out. However, it is important to note that participants in provocation studies generally do not report such large increases in symptoms as a function of time (Schmidt, Wolfs-Takens, Oosterlaan, & van den Hout, 1994). In addition to this, it is possible that effects on risk perception and symptoms may be triggered by any message on EMF and health, irrespective of whether it is an alarmist or positive message. However, Crichton and Petrie (2015a) found that positively framed health information may reverse or dilute the effect of negative expectations formed by alarmist media in the context of infrasound exposure, which suggests that the frame of the message is important for symptom perceptions. Nevertheless, future studies could include a third 'positive' video group to address this issue. Finally, due to feasibility requirements, the present study was limited to assessing acute symptom responses to acute exposures and the experiment was conducted in a laboratory setting using a Faraday cage. It is important that these factors are taken into consideration when interpreting the results of the present study, as these conditions may not reflect typical everyday exposure scenarios.

Although the present study has provided further evidence that symptoms attributed to EMF exposure are likely the result of a nocebo response, one of the major difficulties in treating IEI-EMF is the stigma attached to the notion that the condition is a psychological illness. While cognitive-behavioural therapy has been shown to be efficacious in treating IEI-EMF (Rubin, Das Munshi, & Wessely, 2006), simply telling sufferers that their symptoms do not have a toxicological cause is not reassuring and is unlikely to completely alleviate symptoms (Rief, Heitmüller, Reisberg, & Rüddel, 2006). For instance, although van Moorselaar et al. (2017) found that providing

individual feedback on the results of double-blind provocation studies reduced IEI-EMF participants certainty about responding to acute EMF exposures, the feedback did not materially change IEI-EMF sufferers perception of being sensitive to EMF in their everyday life. Likewise, Nieto-Hernandez, Rubin, Cleare, Weinman, and Wessely (2008) found that providing feedback to IEI-EMF sufferers about their ability to discriminate between active and sham exposures had no influence on subsequent symptom levels or perceived sensitivity to EMF. Explaining the nocebo response may, however, offer an alternative solution. Recently, Crichton and Petrie (2015b) found that participants who reported symptomatic experiences during infrasound exposure returned mood and symptom levels to baseline levels in a subsequent exposure after they had received an explanation of the nocebo response with supporting scientific evidence. Whether such an effect could be replicated in people who experience IEI-EMF, however, remains unclear. Explaining that the nocebo response experienced by IEI-EMF sufferers is a normal human response may also offer a useful approach for addressing the condition in the future. Nonetheless, it is conceivable that explaining psychological mechanisms to people who claim to experience IEI-EMF may be interpreted as offensive or lacking credibility. It may thus be more appropriate (and more accurate) to emphasise that although EMF has not been shown to cause symptoms, that this does not mean that IEI-EMF symptoms are necessarily due to the nocebo effect; they may also relate to an undiagnosed medical condition (Dieudonné, 2016) which would require attention from a medical professional. Discussing the potential aetiology of symptoms with IEI-EMF sufferers is thus a difficult task, and one that requires further investigation.

4.7 References

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5. CHAPTER 5: GENERAL DISCUSSION

Despite decades of research, the aetiology of IEI-EMF has remained extremely controversial. While much of the existing literature has not found convincing evidence of a relationship between EMF exposure and the symptoms reported by IEI-EMF sufferers (Röösli, Frei, Mohler, & Hug, 2010; Rubin, Nieto-Hernandez, & Wessely, 2010), it has been estimated that between 1.5 – 13.3% of the population report experiencing the condition. While disagreement has existed over the cause of IEI-EMF, it has generally been agreed that sufferers are experiencing real symptoms which significantly impair their daily functioning and quality of life (Johansson, Nordin, Heiden, & Sandström, 2010; Rubin, Hillert, Nieto-Hernandez, van Rongen, & Oftedal, 2011). The continuing aetiological debate, however, has limited the development of effective treatments and support for those who experience the condition, and this has warranted the need for further investigation.

The overall aim of the present doctoral research was to clarify the determinants of IEI-EMF by investigating whether toxicogenic or psychogenic processes can explain the symptoms reported by IEI-EMF sufferers. Specifically, the research contained in this thesis encompassed three human provocation studies, each designed with considerable methodological improvements on the extant literature, to determine whether individuals can be sensitive to EMF exposure and to further explore the potential role of psychological processes and alarmist media reports in the presentation of symptoms attributed to EMF exposure. The following chapter discusses the findings and contribution of each study to the literature, and outlines the implications of this research for the development of effective treatments and interventions for IEI-EMF sufferers.

The potential limitations of this research are also discussed, and directions for future studies are highlighted.

5.1 Contribution to the Literature

5.1.1 No evidence of an adverse functional consequence of RF-EMF

A number of studies have consistently shown that exposure to RF-EMF, similar to that emitted by a mobile phone, can influence the brain's electrical activity, specifically in the spontaneous resting alpha (Croft et al., 2008; Croft et al., 2010; Curcio et al., 2005; Leung et al., 2011; Perentos, Croft, McKenzie, Cvetkovic, & Cosic, 2007; Regel, Gottselig, et al., 2007) and sleep spindle (Huber et al., 2000; Huber et al., 2002; Loughran et al., 2005; Regel, Tinguely, et al., 2007; Schmid et al., 2012) frequencies of the electroencephalogram (EEG). Given the close relationship between the EEG and cognition, and given that IEI-EMF sufferers often report memory and concentration difficulties as symptoms which they attribute to EMF exposure, Study 1 (Chapter 2, Verrender, Loughran, Dalecki, McKenzie, & Croft, 2016) was developed as a means of establishing whether exposure to RF-EMF below the established safety guidelines is capable of eliciting these effects in humans. While a number studies assessing various aspects of cognitive and behavioural functioning have found inconsistent, but mostly null results (Barth, Ponocny, Gnambs, & Winker, 2012; Barth et al., 2008; Valentini, Ferrara, Presaghi, De Gennaro, & Curcio, 2010), the methodological issues inherent in previous research may have limited the ability of these studies to detect real effects. If clear adverse effects on cognitive performance could be demonstrated in healthy participants, this could offer a starting point for determining the most sensitive endpoints with which to test IEI-EMF participants. Thus, to address the potential limitations of previous studies, and as a means of developing a potential sensitive

objective test for IEI-EMF sufferers, Study 1 was designed with considerable methodological improvements to determine whether RF-EMF exposure could influence cognitive performance in a dose-dependent manner.

The first stage of Study 1 required identifying possible methodological constraints in previous studies and developing appropriate improvements to overcome these issues. For instance, previous studies have generally relied on cognitive performance measures, such as the N-back task, because of their perceived face validity or as a means to replicate previously reported effects. However, these measures have generally been unable to account for individual variation in cognitive performance (Regel & Achermann, 2011), or have been found to be affected by learning effects (Haarala et al., 2005; Haarala et al., 2004; Regel, Gottselig, et al., 2007; Regel, Tinguely, et al., 2007). This means that the cognitive performance tasks utilised in previous studies have not been able to reliably measure the potential effects of RF-EMF exposure on cognition due to large error variance (Regel & Achermann, 2011). To overcome this issue, the cognitive performance tasks utilised in Study 1 were individually calibrated to each participant's cognitive ability, and sufficiently long practice blocks were conducted before each experimental session to reduce the influence of potential learning effects. In addition to this, Type I and Type II error was minimised by treating the data using an index of the participants' response sensitivity and bias, as adapted from signal detection theory (Stanislaw & Todorov, 1999). This treatment takes into account how well a participant can discriminate between trials (sensitivity) and the participant's general tendency to respond with a 'yes' or 'no' button press (bias), giving a better indication of task performance. Further to these improvements on the cognitive performance tasks, Study 1 also reduced the influence of thermally induced variability by clamping skin

temperature to a thermo-neutral state. This is potentially important, because it is possible that whole body thermoregulatory processes play an integral role in mediating the changes in the brain's electrical activity and any functional effects resulting from exposure to RF-EMF given that the primary mechanism of interaction between RF and the human body is thermal (Adair & Black, 2003). Together, the improvements implemented in Study 1 were intended to increase the sensitivity of the experiment, and thus the ability to detect potential effects.

The results of Study 1 indicated that exposure to pulse modulated RF-EMF, similar to that emitted by a mobile phone, may influence cognitive performance. Specifically, a small improvement in reaction time was observed on the Sternberg working memory task during exposure compared to sham. The improvement in performance, however, was not found to be dose dependent, and the relatively small effect size means that it may not be important in relation to normal variation in cognitive performance. While a number of studies have not found that pulse modulated RF-EMF influences behavioural measures of cognitive performance (Haarala et al., 2003; Haarala et al., 2004; Haarala et al., 2007; Krause, Pesonen, Bjornberg, & Hamalainen, 2007; Leung et al., 2011), the methodological improvements employed in Study 1 may have increased the sensitivity of the experiment and thus enhanced the ability to detect potential effects. However, without further replication, it is unclear whether the improvement in performance observed in Study 1 represents more than a chance finding. Consequently, despite accounting for a number of methodological limitations, Study 1 did not provide sufficiently convincing evidence that exposure to RF-EMF can influence cognitive performance; it thus did not identify any sensitive cognitive performance endpoints with which to test IEI-EMF participants.

5.1.2 No evidence of a relationship between IEI-EMF symptoms and EMF exposure

As Study 1 did not identify an appropriate objective cognitive performance measure with which to test IEI-EMF participants, and indeed, did not find convincing evidence that exposure to RF-EMF can elicit adverse functional effects on cognitive performance, Study 2 (Chapter 3, Verrender, Loughran, Anderson, et al., 2018) was designed as a series of individual case studies to test whether exposure to RF-EMF results in an increase in IEI-EMF participants self-nominated symptoms compared to sham, and additionally, to determine whether IEI-EMF individuals could accurately detect the presence of EMF emissions under double-blind conditions. Despite much of the existing literature indicating that the symptoms experienced by IEI-EMF sufferers are the result of a nocebo effect (Röösli et al., 2010; Rubin et al., 2010), some researchers and many IEI-EMF sufferers have raised concerns about the way in which IEI-EMF provocation studies have been conducted and have argued that methodological issues have influenced the results of previous studies (Leszcynski, 2018; Weller, 2014). These concerns have often been related to questions about whether laboratory testing environments adequately reflect the conditions in which symptoms are normally developed, and worries about whether previous studies have adequately accounted for the heterogeneous nature of IEI-EMF (including between-subject differences in both symptoms and EMF triggers).

To address these concerns, Study 2 incorporated a number of important methodological improvements. First, to reduce the stress and anxiety experienced by participants in a laboratory experiment, and to minimise potential confounding effects associated with inadvertent environmental EMF exposures on the way to a laboratory, Study 2 utilised a

portable exposure device which enabled double-blind testing to take place in environments where participants generally felt safe and asymptomatic, such as in their own home. Second, to limit potential statistical confounds, the study took an idiographic, case-study approach to testing, and used a sufficient number of sham and active exposure trials to determine statistically, within each participant, whether any symptom/exposure relationship was significant. Further to this, the study included a consideration of each participants IEI-EMF history, which involved using a similar RF-EMF exposure to the one which the participant claimed triggers symptoms as well as verifying that the exposure triggered symptoms in an open-label trial. In addition, the testing protocol could be modified, if necessary, to match the reported symptom onset and recovery periods for each participant, thus accounting for potential inter-individual heterogeneity in symptom onset and recovery times. Finally, the design incorporated a fully counter-balanced protocol in order to reduce time of day and time on task effects.

Yet, despite accounting for a number of potential limitations of previous IEI-EMF provocation studies, the results of the case studies presented in Study 2 failed to demonstrate that the symptomatic response of self-reported IEI-EMF sufferers is affected by EMF exposure, nor that IEI-EMF sufferers could detect the presence of RF-EMF emissions at greater than chance levels. While all three case study participants displayed an increased symptom severity and were confident that they could detect the presence of RF-EMF in the RF-ON compared to RF-OFF open-label trial, no significant differences in symptom severity or exposure detection were found between the RF-ON and sham conditions in the double-blind trials. Notably, in each case, a significant relationship between a participant's belief that they were being exposed (irrespective of the actual exposure condition) and their symptomatic response was observed, giving a

strong indication the symptoms experienced were more closely related to a nocebo response. In line with the overall pattern of results of previous provocation studies investigating IEI-EMF (Rubin et al., 2010), these findings confirm that awareness and/or belief of exposure status, rather than the EMF exposure itself, is more closely associated with IEI-EMF symptoms. This not only provides strong support for the psychogenic theory of IEI-EMF, but also demonstrates that the lack of evidence for a relationship between symptoms and EMF exposure observed in previous studies was not due to the methodological concerns raised by some researchers and IEI-EMF sufferers.

5.1.3 Nocebo responses may reflect a normal human response and may be exacerbated by alarmist media coverage

As Study 2 provided further evidence to support the psychogenic theory of IEI-EMF, it became crucial to understand whether the nocebo response exhibited by IEI-EMF sufferers is a 'normal' human response, and to determine the factors which may contribute to such a response. If healthy participants were found to exhibit a similar response to those who suffer from IEI-EMF during a perceived threatening exposure to EMF, then this may remove the stigma attached to the notion that the condition is a psychological illness and may help to overcome the current reluctance of IEI-EMF sufferers to seek psychological treatment. In addition, although there has been consistent evidence that both precautionary and mainstream media messages about the potential harmful effects of EMF exposure can negatively influence people's beliefs and raise concerns about EMF exposure (Barnett, Timotijevic, Shepherd, & Senior, 2007; Nielsen et al., 2010; Wiedemann, Boerner, & Repacholi, 2014; Wiedemann et al., 2013; Wiedemann & Schütz, 2005; Wiedemann, Thalmann, Grutsch, & Schütz, 2006;

Witthöft et al., 2017), it has been relatively unclear whether these negative beliefs directly result in greater symptom formation and detection following a perceived exposure to EMF. This is because previous research has either not assessed the effect of explicit suggestions of risk from EMF exposure (which may induce negative expectations) (Szemerszky, Köteles, Lihi, & Bárdos, 2010), has assessed somatosensory perception and not symptom perception (Bräscher, Raymaekers, Van den Bergh, & Witthöft, 2017), or has not included a verified non-exposure condition, which may have resulted in insufficient statistical power to detect effects in non-anxious healthy participants (Witthöft & Rubin, 2013). Thus, it has remained unclear as to whether the negative beliefs induced by explicit suggestions about EMF exposure contribute to a symptomatic nocebo response.

To address these issues, Study 3 (Chapter 4, Verrender, Loughran, Dalecki, Freudenstein, & Croft, 2018) tested whether perceived EMF exposure could elicit symptoms in a healthy population sample, and whether viewing an alarmist video emphasising the 'adverse effects of EMF exposure' could exacerbate a nocebo response in a healthy population. In this study, participants were first randomly assigned to view either an alarmist video, which emphasised the 'adverse effects of EMF exposure', or a control video completely unrelated to EMF health effects, before completing a series of 2 open-label (RF-ON and RF-OFF) and 12 randomised, double-blind, counterbalanced provocation trials (6 RF-ON, 6 Sham). Importantly, like Study 2, the open-label trials were used to verify that the experiment contained both active and sham exposure conditions. By demonstrating that the experiment contained a 'no exposure' condition, Study 3 was able to more-clearly determine whether any observed increases in symptoms in healthy participants were the result of a nocebo effect.

In line with Study 2, the results of Study 3 showed that healthy participants reported higher symptom scores in the RF-ON compared to the RF-OFF open-label trial. However, in the subsequent double-blind trials, no difference in either belief of exposure or symptoms was found between the RF-ON and sham conditions. Belief of exposure was also found to be positively associated with higher symptom scores in the double-blind trials, giving further indication that a nocebo effect, rather than EMF exposure itself, was responsible for the increase in symptoms. These results again demonstrate that knowledge and/or awareness of the exposure condition is essential for producing an effect. The large effect sizes observed for the relationship between belief of exposure and symptoms in the open-label trials of Study 2 (*ES* = 3.6) and Study 3 (*ES* = .71) also provide robust support for the notion that a nocebo effect can explain symptoms attributed to EMF exposure, and suggest that the nocebo response exhibited by IEI-EMF sufferers may be a normal human response. Participants who viewed the alarmist media video were also found to report higher symptom scores in the open-label trials; and a greater increase in state anxiety and risk perception from baseline, than those who viewed the control video. This indicates that viewing sensationalist media reports about perceived environmental hazards both raises concerns and negative beliefs about EMF exposure, and may also contribute to a symptomatic nocebo response.

While these findings provide further support for the psychogenic theory of IEI-EMF, they are also consistent with a large amount of evidence which has demonstrated that health warnings can increase concerns about environmental exposures and elicit symptomatic nocebo responses (Crichton, Chapman, Cundy, & Petrie, 2014). For instance, Winters et al. (2003) found that participants who had received warnings about environmental pollution reported more symptoms to a foul smelling (but

physiologically irrelevant) odour stimulus than participants who had received no prior information about environmental pollution. Similarly, Crichton, Dodd, Schmid, Gamble, and Petrie (2014) demonstrated that healthy participants who had received information about an expected negative physiological effect of wind turbine infrasound reported symptoms that aligned with that information during exposure to both active and sham infrasound. In a follow up study, Crichton and Petrie (2015a) found that the framing of information is important in eliciting an effect. In that study, positively framed health information about the effects of infrasound exposure was shown to reverse an initial symptomatic nocebo response that was generated by negatively framed information. Clearly, the type of information disseminated in public has profound effects on expectations and the experience of nocebo responses. In a powerful demonstration of the role of expectations in producing symptoms, Landgrebe et al. (2008) deceived IEI-EMF participants into thinking that they were being exposed to EMF. The results showed that the deception not only lead to expectations which resulted in symptom formation, but was also accompanied by activations of the brain regions known to be involved in pain perception. This not only demonstrates the role of expectations in producing a nocebo response, but also provides a psycho-physiologically plausible mechanism as to why symptoms develop and are detected during nocebo responses. Given the strong indications of the determinative role of nocebo effects and negative expectations in eliciting symptoms attributed to EMF, it is possible that the consistent misrepresentation of the scientific evidence in the mainstream media (Claassen, Smid, Woudenberg, & Timmermans, 2012; Eldridge-Thomas & Rubin, 2013) may contribute to a symptomatic nocebo response.

5.1.4 Implications for developing effective treatments and interventions for IEI-EMF sufferers

Given the historical lack of evidence for an association between exposure to low level EMF and adverse health effects, the development of effective treatments for people who experience IEI-EMF has remained relatively challenging. While many public health organisations and governments have recommended exposure reduction strategies to those who are concerned about their EMF exposure, the current evidence does not provide any support to the notion that such strategies are effective for treating IEI-EMF. The difficulty in developing treatments and interventions for those who experience IEI-EMF has also been limited by sufferers' firm belief that the condition is caused by exposure to EMF and the stigma attached to the notion that the condition is psychological in origin, with the suggestion that symptoms are more closely related to a psychosomatic phenomenon often attracting derision from IEI-EMF advocacy groups. Overall, the results of the present thesis have provided strong support for the psychogenic theory of IEI-EMF, which has several implications for the development of effective treatments and support for IEI-EMF sufferers.

For example, exposure reduction strategies are one of the most commonly adopted interventions employed by IEI-EMF sufferers. Generally, exposure reduction strategies involve minimising or avoiding the use of EMF emitting technologies. However, many exposure reduction strategies involve considerable social and/or financial cost without any tangible benefit. For instance, although rooms and buildings can be shielded from EMF through the use of metallic paints or the construction of Faraday cages, these methods are often extremely expensive, and vary greatly in their ability to attenuate RF-EMF. Similarly, products typically sold by IEI-EMF advocacy groups which claim to

reduce EMF exposure from personal devices, such as protective stickers for personal devices or protective clothing fabrics, are often ineffectual or have not been supported by empirical tests of their claims (Leitgeb, Cech, Schröttner, & Kerbl, 2008; Rubin, Das Munshi, & Wessely, 2006). In a recent survey study, Hagström, Auranen, and Ekman (2013) found that 76% of IEI-EMF respondents reported the reduction or avoidance of EMF as an action they had taken to manage their condition and that this behaviour helped in their full or partial recovery. Yet, as the current thesis has found no support for the toxicogenic explanation of IEI-EMF, exposure reduction strategies are likely to be ineffective, and may only provide a limited placebo-like solution that would only be useful in specific and limiting circumstances, such as in an IEI-EMF sufferers' home. Moreover, electromagnetic sanitation through exposure reduction or avoidance behaviour may be counterproductive, as it may reinforce an IEI-EMF sufferers' belief that their symptoms are caused by EMF and worsen their condition when they perceive that they are in an environment with EMF (Rubin et al., 2006). In addition to exposure reduction, complementary and alternative medicine treatments are also popular amongst people who suffer from IEI-EMF (Huss & Röösli, 2006). Such treatments, however, have rarely been evaluated in controlled studies with IEI-EMF participants, and thus their effectiveness in treating the condition remains to be determined. Of the few studies that have assessed the use of acupuncture in treating patients with 'environmental illnesses' (Arnetz, Berg, Anderzen, Lundeberg, & Haker, 1995) and antioxidant vitamin supplements to treat individuals with IEI-EMF (Hillert, Kolmodin-Hedman, Eneroth, & Arnetz, 2011), no specific therapeutic benefits were found.

In an attempt to find an explanation and solution for their symptoms, many IEI-EMF sufferers often consult with general practitioners (GPs) and other first-line health

professionals. Although data concerning the use of health services by IEI-EMF sufferers in Australia is lacking, survey studies evaluating the use of GP consultations in European nations have shown that the majority of GPs in these countries have been consulted at least once by IEI-EMF sufferers (Huss & Röösli, 2006; Kowall, Breckenkamp, Heyer, & Berg-Beckhoff, 2010; Leitgeb, Schröttner, & Böhm, 2005; Slottje et al., 2017). For instance, Leitgeb et al. (2005) reported that at least two thirds of GPs who responded to the survey were frequently consulted by IEI-EMF sufferers in Austria, while Huss and Röösli (2006) found that 69% of respondent GPs reported having at least one consultation about symptoms attributed to EMF. Alarmingly, many of these survey studies have revealed that health professionals generally have a poor understanding of the current scientific consensus regarding EMF exposure and health risks and that many consider a causal relationship between EMF and health complaints to be at least to some degree plausible. For example, Leitgeb et al. (2005) found that 96% of GP respondents either 'totally' or 'to some degree' believed in the health relevant role of EMF exposure in producing symptoms or illness. Similarly, Huss and Röösli (2006) found that respondent GPs judged the relationship between EMF exposure and symptoms to be plausible in 54% of cases. Although it is concerning that health professionals may be fostering people's beliefs that EMF exposure is the cause of their symptoms, this may be due to the GPs own insufficient knowledge, or a limited understanding of the scientific evidence. For instance, Leitgeb et al. (2005) reported that only 25% of medical practitioners had consulted the scientific literature for information about the potential health risks of EMF, while only 4% reported receiving information from health agencies or governments. In addition to this, it is also possible that GPs have evaluated the relationship between EMF exposure and symptoms to be plausible on the basis that they are taking a precautionary or preventative approach for their

patients in an area which they themselves perceive to be scientifically uncertain. Nevertheless, given that the studies presented in the current thesis provide no support for the role of EMF in producing symptoms, and given the apparent contradiction between physicians' opinions and the positions of the researchers and the world's leading health authorities on EMF health risks, it is clear that greater effort is needed to communicate the current position of science to these first-line health professionals so that they can begin to discuss this issue and develop appropriate alternative interventions with their patients (Slottje et al., 2017).

Helping patients to consider alternative explanations for their symptoms has been found to be effective across a range of similar conditions that are characterised by the presentation of non-specific symptoms without identifiable cause, such as chronic fatigue syndrome (van Hout, Wekking, Berg, & Deelman, 2003). Yet, the stigma attached to the notion that IEI-EMF is more closely related to a psychological illness has proven to be a difficult challenge to overcome, and simply telling IEI-EMF sufferers that their symptoms do not have a toxicological cause is not an effective way to alleviate symptoms (Rief, Heitmüller, Reisberg, & Rüddel, 2006). For example, Nieto-Hernandez, Rubin, Cleare, Weinman, and Wessely (2008) found that providing feedback to IEI-EMF sufferers about their lack of ability to discriminate between active and sham exposures had no influence on subsequent symptom levels or perceived sensitivity to EMF. Likewise, van Moorselaar et al. (2017) found that while feedback on provocation study results reduced IEI-EMF sufferers' certainty about their ability to respond to acute exposure scenarios, the overall feedback did not materially change their perceptions of being sensitive to EMF. As Study 2 and Study 3 have demonstrated the crucial role of awareness and belief of exposure in producing symptoms attributed to

EMF, interventions which focus on these psychological processes, such as Cognitive Behavioural Therapy (CBT), may offer an alternative avenue for treatment research and development.

CBT is a form of structured psychotherapy designed to change unhelpful or unhealthy thoughts and behaviours (Neenan & Dryden, 2014). It is a problem-focused and individualised approach that focuses on remedying immediate problems, but it also attempts to develop long-term strategies to replace thoughts and behaviours that interfere with a person's happiness and satisfaction with their life (Neenan & Dryden, 2014). CBT has been found to be an effective treatment for a range of psychological issues, such as depression and anxiety, and it has also been used extensively to reduce somatic symptoms in somatosensory disorders and to reduce the side-effects of medications (e.g. nausea associated with chemotherapy for the treatment of cancer) (Neenan & Dryden, 2014). Given that CBT has also been shown to be efficacious in treating other conditions characterised by medically unexplained symptoms (Edwards, Stern, Clarke, Ivbijaro, & Kasney, 2010; Escobar et al., 2007; Sharpe et al., 1996; Speckens et al., 1995), it may also be an appropriate approach for treating IEI-EMF. However, as only a limited number of studies have assessed the efficacy of CBT as an intervention for IEI-EMF sufferers, the potential benefits of this treatment option remain to be adequately clarified.

To date, only four studies have assessed the efficacy of CBT as an intervention for suffering from IEI-EMF. After assessing symptoms and quality of life outcomes in IEI-EMF sufferers following CBT, three of these studies found reductions in self-ratings of hypersensitivity (Hillert, Arnetz, Hedman, & Dölling, 1998), disability (Andersson et al., 1996), symptoms (Andersson et al., 1996; Harlacher, 1998), and overall perception

and degree of suffering (Harlacher, 1998), while one study did not report any significantly better outcomes than the control condition (Hillert, Savlin, Levy Berg, Heidenberg, & Kolmodin-Hedman, 2002). However, while the majority of these studies generally indicate that CBT may be an effective treatment for those who suffer from IEI-EMF, a number of possible methodological issues have limited our understanding of the long term efficacy of CBT as an IEI-EMF treatment (Rubin et al., 2006). For example, no studies have conducted follow-up assessments of patients for more than 6 months, so it is unclear how long any beneficial effects of CBT last in these cases (Rubin et al., 2006). Furthermore, it is possible that the beneficial effects reported in these studies were actually associated with the general non-specific effects of receiving psychotherapy (Rubin et al., 2006). Although this could be interpreted as a useful placebo-like effect, and would not be an issue while therapy is ongoing, it is unclear if or for how long these benefits would continue post-therapy. Future studies, therefore, could usefully clarify the effectiveness of CBT as an intervention for people suffering from IEI-EMF by employing longer-term follow-up assessments.

While CBT may prove to be an effective treatment for IEI-EMF sufferers, the results of Study 2 and Study 3 also indicate that other psychological interventions, which focus on the underlying factors which drive nocebo responses, may provide another alternative avenue for treatment research and development. Generally, a nocebo response occurs when conscious or subconscious negative expectations trigger or exacerbate adverse symptoms in response to an exposure that is not known to cause those effects (Bräscher, Kleinböhl, Hölzl, & Becker, 2017; Hahn, 1997). These expectations may be induced by explicit suggestions about the potential effects of an exposure (Benedetti, Lanotte, Lopiano, & Colloca, 2007; Webster, Weinman, & Rubin, 2016) or by learning through

classical conditioning (Bräscher, Kleinböhl, et al., 2017). Consequently, interventions which focus on modifying conscious or subconscious negative expectations may provide an effective means for treating people who suffer from IEI-EMF.

For instance, there is some emerging evidence that providing a scientific explanation of the nocebo response and its mechanisms following a provocation trial may offer an effective way to change people's conscious expectations about their IEI symptoms. Recently, Crichton and Petrie (2015b) found that participants who reported symptoms during infrasound exposure returned mood and symptom levels to baseline levels in a subsequent exposure after they had received an explanation of the nocebo response with supporting scientific evidence. This suggests that providing an explanation of the nocebo response was able to modify people's response during a subsequent provocation trial. Given the results of Study 3, it may also be possible that explaining that the nocebo response experienced by IEI-EMF sufferers is a normal human response (rather than an abnormal psychological disorder) may also help in modifying conscious expectations; however, further investigation is required to determine the efficacy of such an intervention with IEI-EMF participants. If, however, classical conditioning is one of the main factors driving a nocebo response (as suggested by some theorists, for example, Barsky, Saintfort, Rogers, & Borus, 2002; Webster et al., 2016), then interventions based on systematic desensitization, in which repeated exposure to EMF is paired with relaxation techniques, may also diminish a possible conditioned response to environmental stimuli (Rubin et al., 2006). The efficacy of such an intervention with IEI-EMF patients, however, remains to be empirically tested, and it is unclear whether this type of treatment would be suitable for people who experience symptoms

associated with a broad range of EMF sources compared to those who only experience symptoms associated with specific devices.

The way health information is framed may also modify conscious or subconscious expectations about environmental exposures. For example, there is evidence that has shown that framing health information about environmental exposures in a neutral or benign way can ameliorate IEI symptoms. Crichton and Petrie (2015a) found that participants who formed negative expectations from media warnings about infrasound from wind turbines reported increased symptoms and deterioration in mood during simultaneous exposure to infrasound and audible wind farm noise, yet those who formed positive expectations derived from information about the therapeutic effects of infrasound experienced improvements in symptoms and mood. This demonstrates the malleability of symptomatic responses, and highlights the important role of message framing on expectations and placebo/nocebo effects. Yet, while these studies indicate that interventions which focus on modifying conscious or subconscious expectations can effectively reduce symptoms associated with other IEI conditions, as no similar studies have been conducted with people who report experiencing IEI-EMF, it remains unclear as to whether such effects could be replicated in IEI-EMF sufferers.

5.2 Potential Limitations

Although considerable effort was made to address the limitations of previous research in each of the studies contained within the present thesis, a number of possible issues need to be taken into consideration when interpreting the results of the present thesis. Many of these issues relate to potential limitations associated with the ecological validity of human provocation studies and the methods used to assess IEI-EMF symptoms more generally.

The first potential limitation relates to exposure duration. While the majority of people with IEI-EMF report that their symptoms typically occur within minutes to hours after exposure, some report that their symptoms are the result of longer exposures or of an accumulation of exposures over time (Hocking, 1998; Röösli, Moser, Baldinini, Meier, & Braun-Fahrländer, 2004). Generally, to make them feasible, provocation studies are limited to using short term, acute exposures. As the exposure intervals implemented in the studies contained in the present thesis were limited to 30 minutes or less, the conclusions drawn from these studies cannot be used to comment on possible long term, chronic effects of EMF exposure. However, while the more chronic forms of IEI-EMF remain under-investigated using the provocation study paradigm, it is important to note that there has been no convincing evidence from epidemiological studies that long term exposures have an adverse effect on human health either (Röösli et al., 2010). Although many advocates of the toxicogenic theory of IEI-EMF argue that the condition may be a result of a 'build up' of chronic exposures over time (for e.g. over several days/weeks/months), this could only be determined empirically using a sufficient amount of active and sham exposure conditions (as per the methodology of Study 2). While the amount of time it would take to conduct such provocation trials would make this extremely difficult (and likely not feasible), given the complexities of subjectively trying to summate average exposure periods over long intervals with a multitude of potential confounding factors (in daily life), it seems unreasonable that IEI-EMF sufferers could reliably claim that their symptoms were the result of chronic exposures to EMF. Therefore, acute exposure trials not only offer the most feasible way of testing the claims of IEI-EMF sufferers, they also offer the most direct means of testing the claims made by IEI-EMF sufferers.

The use of simulated exposure signals may be another possible limitation. As no consistent pattern in the types of EMF emitting sources that are claimed to trigger IEI-EMF symptoms has been identified, provocation studies have generally relied on exposure systems which simulate the emissions produced by everyday devices. The studies contained in the present thesis used exposure systems which either simulated mobile handset-like exposure (Study 1) or generated a signal which was digitally modulated in similar manner to signals from Wi-Fi routers and 3G/4G mobile phones (Study 2 and Study 3). While it is often argued that simulated exposure signals can be used to reliably test whether a well-characterised exposure is associated with an adverse effect (Boutry et al., 2008; Regel & Achermann, 2011), some researchers have criticised the use of simulated exposure signals and argue that experimental findings could only be ecologically valid if relevant commercially available EMF emitting devices were used (Panagopoulos, Johansson, & Carlo, 2015). Unlike everyday devices (such as commercially available mobile phones), which produce a relatively localised yet variable SAR distribution which can be reduced by orders of magnitude with greater separation from the exposure (Loughran, McKenzie, Anderson, McIntosh, & Croft, 2008), simulated exposure devices produce controlled and precise exposures. Although it may be possible that the unpredictable and heterogeneous nature of everyday EMF emissions is an important factor for eliciting an adverse effect (Oftedal, Straume, Johnsson, & Stovner, 2007; Rubin et al., 2010), it is important to note that there is currently no evidence to support this notion. Despite this, it may be advantageous for studies to use simulated exposures if they are attempting to develop an understanding of whether exposures with a specified dosimetric value can affect health (Loughran et al., 2008). For instance, in Study 1, a planar exposure system was used to generate a mobile phone handset-like signal to assess whether EMF exposure could influence cognitive

performance in a dose dependent manner. Because the SAR distribution produced by the exposure system was more homogenous than that produced by a typical mobile phone, the findings cannot be used to definitively comment on whether cognitive performance is influenced by exposure to EMF emitted by *mobile phones*, but rather can only reflect whether RF-related bioeffects occur at the maximum exposure level within the exposed hemisphere. However, in respect to the endpoints of the present thesis, the use of a planar exposure system in Study 1 was appropriate, as exposure of the whole hemisphere maximised the chance of finding any possible effects on cognitive performance. Similarly, while the portable exposure device used in Study 2 and Study 3 generated a signal that would not typically be emitted by everyday devices (as the signal band was reserved for industrial, scientific and medical use), initial non-blinded open-label trials were used to verify that participants believed that they were being exposed and that they responded to the signal, which verified that it was appropriate for the purposes of the study.

Some IEI-EMF advocates also view the use of subjective measures of symptoms as a major limitation of provocation studies, and argue that such measures are unreliable and insufficient to either prove or disprove the existence of a causal link between the reported symptoms and EMF (Leszcynski, 2018). While both Study 2 and Study 3 used questionnaires to assess participant's subjective symptom experiences, it is important to note that such questionnaires are designed to specifically test the claims of IEI-EMF sufferers, and that no objective test for symptom experiences has been developed and no formal diagnostic tests have been established to identify people who experience IEI-EMF. Although Leszcynski (2018) argues that physiology based research examining molecular responses of human tissues and organs will provide the evidence for

individual sensitivity to EMF, it first needs to be established that the exposures claimed to elicit a symptom response are actually capable of eliciting a symptom response. As no toxicogenic relationship between adverse health effects and exposure to EMF has been established, and due to the extremely heterogeneous nature of IEI-EMF, there is no reason to expect that empirical tests assessing whether one of a vast number of physiological endpoints is influenced by EMF exposure would contribute to our understanding of the condition. In this sense, only double-blind provocation studies with appropriate verification procedures (as implemented in Study 2) are able to test the symptom claims of IEI-EMF sufferers.

In addition to the aforementioned issues, a number of other possible limitations apply to the specific studies contained within this thesis. First, the statistical analyses in Study 1 controlled for comparison-wise error by restricting the planned contrasts to degrees of freedom error without multiple comparison adjustment (Tabachnick & Fidell, 2013). However, this method does not control for experiment-wise error, and so it is still possible that the results of Study 1 were influenced by Type 1 error. Second, despite using an improved methodology, the results of Study 2 cannot be generalised across the entire IEI-EMF population, given the relatively small number of participants who completed the study. Recruitment of sufficient numbers of participants is one of the most challenging aspects of conducting provocation studies involving IEI-EMF participants, as reflected by the relatively small sample sizes of previous studies (median number of IEI-EMF participants in previous studies = 19) (Rubin et al., 2010). However, given the consistency within the literature, it is unlikely that the low sample sizes are the reason for the overall failure of provocation studies to detect a toxicogenic relationship between EMF exposure and symptoms (Rubin et al., 2010). For instance,

after pooling the results from five separate provocation studies, Roosli (2008) still failed to detect a significant effect of exposure. The reluctance to participate in provocation studies may be due to scepticism of the scientific process, concerns about the possible adverse effects caused by voluntary exposure, and/or of distrust of the research group conducting the investigation. Nevertheless, the considerable methodological improvements employed in Study 2 meant that it was appropriately designed to detect partial IEI-EMF responses within each case separately. Finally, it is possible that pre-existing beliefs about the relative harmfulness of EMF exposure may have influenced the results of Study 3 via a ceiling effect. While it is impossible to control for the beliefs formed by situational factors (such as viewing a particular media report before an experimental session), pre-screening could be used to allocate participants into "high" and "low" risk perception groups before any experimental manipulation has taken place, which would allow this issue to be controlled for experimentally in future study designs.

5.3 Future Directions

Given that a considerable amount of research has determined that IEI-EMF is of a psychogenic origin, it is important that future research is directed towards targeting the factors that contribute to nocebo responses. This needs to be a multifaceted approach, which involves not only the communication of the current scientific evidence to frontline medical professionals, but also the dissemination of accurate information to the community more broadly. As the present thesis found that the nocebo effect was exacerbated by alarmist media emphasising the 'adverse effects of EMF' in healthy participants, it is regrettable that some commentators and advocates of the toxicogenic theory continue to discuss IEI-EMF without sufficient evaluation of the literature (e.g. BioInitiative Working Group, 2012) as this appears to promote or exacerbate nocebo

effects in society more broadly. Evidently, the scientific community need to develop effective ways to communicate the current state of science (Rubin et al., 2011). Moreover, further research is needed to clarify how personality traits may interact with situational factors (such as viewing a particular media report) to influence peoples worries and expectations regarding EMF exposure in order to identify people who may be more susceptible to alarmist media reports and resultant nocebo effects.

In addition to this, future research could benefit by focusing on developing effective interventions and support for those who suffer from IEI-EMF. This could be achieved by first clarifying the efficacy of psychological approaches to treating IEI-EMF (Rubin et al., 2006), irrespective of whether these are CBT or non-CBT based interventions. While some evidence has shown that CBT may be an effective strategy for treating IEI-EMF (Andersson et al., 1996; Harlacher, 1998; Hillert et al., 1998), non-CBT related interventions may also provide a useful direction for future research. In particular, given that Crichton and Petrie (2015a) found that positively framed health information may reverse or dilute the effect of negative expectations formed by alarmist media in the context of infrasound exposure, future studies could profit from exploring whether positively framed messages about EMF exposure can ameliorate symptoms triggered by negative expectations. However, as many who experience IEI-EMF firmly believe that EMF is the cause of their symptoms, caution should be applied when providing psychological interventions or explanations, as such strategies may be interpreted as being offensive or lacking credibility. Thus, alongside studies assessing the efficacy of psychological approaches to treating IEI-EMF, it would be important for future research to focus on developing effective communication of the research between scientists, health professionals and IEI-EMF sufferers.

5.4 Conclusions

Overall, the studies presented in this thesis have found no support for the view that toxicogenic processes can explain symptoms attributed to EMF exposure. Instead, the results of the present thesis strongly indicate that psychological factors play an important role in triggering, maintaining, or exacerbating symptoms in response to perceived exposure to EMF.

In particular, while the present research found that exposure to mobile phone-like RF-EMF may improve cognitive performance on a working memory task, the slight improvement in performance observed in Study 1 did not provide convincing evidence that exposure to mobile phone-like RF-EMF can influence cognitive performance in a dose dependent manner, let alone adversely (Chapter 2, Verrender, Loughran, Dalecki, McKenzie, & Croft, 2016). This result is significant because it provides further support to the overall evidence that while exposure to RF-EMF within the established exposure guidelines (ICNIRP, 1998) can trigger subtle biological and physiological effects (as has been reported previously in relation to the EEG), these changes do not necessarily constitute an adverse effect on health or functioning. Given this finding, Study 1 also did not identify any sensitive cognitive performance endpoints with which to test IEI-EMF participants. The present doctoral research also demonstrated that awareness and belief of exposure, rather than EMF exposure itself, is crucial in eliciting symptoms attributed to EMF (Chapter 3, Verrender, Loughran, Anderson, et al., 2018; Chapter 4, Verrender, Loughran, Dalecki, Freudenstein, & Croft, 2018). While this not only provides further support for the psychogenic theory of IEI-EMF, the fact that healthy participants were found to exhibit a similar symptomatic nocebo response to IEI-EMF sufferers suggests that this type of response may be a normal human response (Chapter

4, Verrender, Loughran, Dalecki, Freudenstein, & Croft, 2018). This has important implications for developing effective treatments for IEI-EMF sufferers, and may help to remove the stigma attached to the notion that IEI-EMF is a psychological issue. Finally, this research also showed that viewing an alarmist media report that emphasised the 'adverse effects of EMF exposure', increases anxiety and concerns about EMF exposure and may also exacerbate nocebo responses in otherwise healthy people (Chapter 4, Verrender, Loughran, Dalecki, Freudenstein, & Croft, 2018). This demonstrates the clear role of negative expectations in eliciting symptomatic nocebo responses and highlights the need for the dissemination of information which accurately reflects the current state of science.

The research presented in this doctoral thesis has considerable implications for the development of effective treatments and support for those who suffer from IEI-EMF. First, it suggests that it is important for interventions to focus on helping IEI-EMF sufferers to consider alternative explanations for their symptoms, and second, that interventions attempt to modify the negative expectations which contribute to symptomatic nocebo responses. In addition to this, given that explicit suggestions, negative expectations and nocebo responses have been found to play a determinative role in eliciting symptoms attributed to EMF exposure, the evidence presented in this thesis clearly indicates that greater effort is needed to communicate the current state of science, not only to medical professionals, but also to the community more broadly.

5.5 References

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