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(Accepted 14 November 2005)

doi 10.1136/bmj.38720.687975.55

Are some people sensitive to mobile phone signals? Within participants double blind randomised provocation study

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BMJ 2006;332:886-9

Abstract

Objective To test whether people who report being sensitive to mobile phone signals have more symptoms when exposed to a pulsing mobile signal than when exposed to a sham signal or a non-pulsing signal.

Design Double blind, randomised, within participants provocation study.

Setting Dedicated suite of offices at King's College London, between September 2003 and June 2005.

Participants 60 "sensitive" people who reported often getting headache-like symptoms within 20 minutes of using a global system for mobile communication (GSM) mobile phone and 60 "control" participants who did not report any such symptoms.

Intervention Participants were exposed to three conditions: a 900 MHz GSM mobile phone signal, a non-pulsing carrier wave signal, and a sham condition with no signal present. Each exposure lasted for 50 minutes.

Main outcome measures The principal outcome measure was headache severity assessed with a 0-100 visual analogue scale. Other outcomes included six other subjective symptoms and participants' ability to judge whether a signal was present.

Results Headache severity increased during exposure and decreased immediately afterwards. However, no strong evidence was found of any difference between the conditions in terms of symptom severity. Nor did evidence of any differential effect of condition between the two groups exist. The proportion of sensitive participants who believed a signal was present during GSM exposure (60%) was similar to the proportion who believed one was present during sham exposure (63%).

Conclusions No evidence was found to indicate that people with self reported sensitivity to mobile phone signals are able to detect such signals or that they react to them with increased symptom severity. As sham exposure was sufficient to trigger severe symptoms in some participants, psychological factors may have an important role in causing this condition.

Trial registration ISRCTN81432775.

Introduction

The health effects most often attributed to mobile phone use are non-specific symptoms, the most commonly reported of which are headache, burning, dizziness, fatigue, and tingling.¹ Mechanisms to explain these phenomena remain speculative, and although the pulsing nature of "global system for mobile communication" (GSM) signals has been suggested to be partly to blame,² experiments that have exposed healthy adults to GSM signals under blind conditions have not found any effect on the reporting of symptoms.³

Of particular interest are people who report symptoms almost every time they use a mobile phone.⁴ This phenomenon falls within the broader category of "electromagnetic sensitivity," a medically unexplained condition in which non-specific symptoms are reported after perceived exposure to electrical devices, including mobile phones, visual display units, and power lines. Provocation studies that have exposed people who report electromagnetic sensitivity to electromagnetic fields under blind conditions have so far failed to provide any good evidence linking the



This is the abridged version of an article that was posted on bmj.com on 6 March 2006: <http://bmj.com/cgi/doi/10.1136/bmj.38765.519850.55>

presence of electromagnetic fields to severity of symptoms.⁵

We tested whether people with self reported sensitivity to GSM experienced greater headache severity after double blind exposure to a GSM signal than after exposure to a sham signal. Secondary outcomes included other symptoms and ability to discriminate GSM from sham signals.

Methods

Study design

We exposed people who reported adverse reactions to mobile phone signals (sensitive group) or who did not report such effects (control group) to three conditions: a signal mimicking that produced by a 900 MHz GSM mobile phone, an unpulsed continuous wave signal, and a sham exposure with no signal present. For each participant we randomised the order of exposure type. Exposures were double blind.

Participants

To be eligible for the sensitive group, participants had to report often experiencing headache-like symptoms within 20 minutes of using a 900 MHz GSM mobile phone. Participants who did not attribute any symptoms to mobile phone signals were eligible for the control group. We recruited participants through mailshots organised by an electromagnetic sensitivity support group, advertising by clinicians, posters in general practitioners' surgeries, adverts and articles in the press and specialist health publications, email circulars, and word of mouth.

Exposures

We generated exposures by using the standard GSM handset system used within the UK Mobile Telecommunications and Health Research programme.⁶ The antenna for this headband mounted system was positioned slightly above and behind the left ear and within a few millimetres of the participant's scalp. Both GSM and continuous wave conditions produced a target specific absorption rate adjacent to the antenna of 1.4 W/kg. For the sham exposure, a continuous wave signal was generated but was diverted to an internal load instead of being transmitted through the antenna.

Questionnaires

We assessed severity of symptoms during exposure by using visual analogue scales,⁷ anchored with the phrases "no sensation" and "worst possible sensation." These scales measured headaches; nausea; fatigue; dizziness; skin itching, tingling, or stinging; sensations of warmth or burning on skin; and eye pain or dryness. We also asked participants to record the frequency with which they experienced 11 common symptoms

after a mobile phone call (never, 25% of calls, 50% of calls, 75% of calls, every call).

Procedure

Participants completed baseline visual analogue scale measures at the beginning of each session. Signal exposures lasted 50 minutes, and participants completed visual analogue scale measures after 5, 15, 30, and 50 minutes and 30 minutes after the end of each exposure. After exposure, we asked participants to state whether they believed a signal had been present and their confidence about this (visual analogue scales from "complete guess" to "100% certain"). Testing took place between September 2003 and June 2005 at King's College London, in rooms that were not shielded against outside electromagnetic fields.

Analyses

To analyse symptom severity over time, we used generalised estimating equations.⁸ This approach accommodated the extremely positively skewed distribution of response variables.

Results

We were contacted by 83 potential sensitive participants and 69 potential controls. Of these, 60 participants in each group attended all three testing sessions.

For sensitive participants, the mean reported delay between beginning a call and onset of symptoms in everyday life was 6.5 (SD 6.5) minutes. They reported headache-like symptoms in a mean of 70.4% of calls. The next most common symptoms were skin warmth or burning (43.8% of calls), difficulty concentrating (30.0%), and dizziness (20.8%). Very few control participants reported any symptoms in relation to mobile phone signals.

Fitted models for all response variables during controlled exposure showed highly significant effects for time (both linear and quadratic effects) and for baseline severity (see bmj.com). We found no convincing evidence of an effect of condition or a condition×group effect for any of the symptoms. For headache, burning sensations, skin sensations, and eye pain we found evidence of a main group effect—sensitive participants reported greater severity. In terms of the original visual analogue scale units, this group effect for headache severity equated to an increase of 1.0 (95% confidence interval 0.4 to 2.0) unit (see bmj.com). The figure shows the median headache severity by group for each exposure condition.

We also analysed the number of severe reactions seen in each condition, with a severe reaction defined as a participant requesting that an exposure be terminated early or withdrawing from the study entirely after

Number of participants who believed a signal was present for each experimental condition and mean (SD) confidence (0-100) reported by participants for these "signal present" assessments

Exposure	Controls		Sensitive participants			
	No	Confidence	Completed all three exposures		Completed at least one exposure	
			No	Confidence	No	Confidence
GSM	35/60	36.8 (28.5)	36/60	58.6 (30.8)	41/65	61.2 (31.0)
CW	42/60	39.7 (33.0)	41/60	57.7 (27.8)	45/64	57.8 (28.9)
Sham	41/60	43.9 (31.9)	38/60	64.4 (31.7)	39/63	64.0 (31.3)

CW=continuous wave; GSM=global system for mobile communication.

an exposure. Twenty six such reactions occurred in the sensitive group (9 withdrawals; 17 early terminations), and none occurred in the control group. These reactions were equally distributed between GSM (n=7), continuous wave (n=10), and sham (n=9) conditions ($\chi^2=0.54$, P=0.76). The table shows participants' assessments of whether a signal was present during provocation. The proportion who believed a signal was present during exposure to GSM (60% of sensitive participants, 58% of controls) was slightly less than for the sham exposure (63% of sensitive participants, 68% of controls). Self reported confidence for these judgments did not differ greatly (table).

Discussion

We found no evidence to indicate that self reported sensitivity to 900 MHz GSM mobile phone signals has a biological basis. Nor did we find any evidence to suggest that the pulsing nature of GSM contributes to these symptoms. These findings agree with the large majority of previous blind or double blind provocation studies for electromagnetic sensitivity, which have found no differences in severity of symptoms elicited by active or sham exposure to electromagnetic fields.⁹

The provocation exposure represented a relatively "worst case scenario" mobile phone call, using a high specific absorption rate and lasting eight times longer than the mean call length usually needed to trigger symptoms in our sensitive sample. Interference from participants' reactions to extraneous electromagnetic fields is unlikely: after 30 minutes adjusting to our offices only two participants reported baseline symptoms that might have masked any effects of exposure, and both were excluded. Finally, as we were able to detect changes in symptom severity over time as highly significant, the sensitivity of our visual analogue scales and our statistical techniques seems adequate.

That symptom severity increased during exposure is interesting. These symptoms were not trivial. For some they were so severe that exposures had to be

What is already known on this topic

Non-specific symptoms such as headaches, tingling sensations, and fatigue are sometimes attributed to mobile phone use

No generally accepted mechanisms exist that might explain how mobile phone signals could cause such effects

A minority of people also report being particularly sensitive to mobile phones, experiencing symptoms almost every time they use one

What this study adds

The signals produced by 900 MHz GSM mobile phones do not cause greater subjective symptoms than sham exposures in which no signal is present, even in people who report sensitivity to mobile phones

The symptoms reported by "sensitive" people may be the result of a nocebo effect and may be primarily psychological in origin

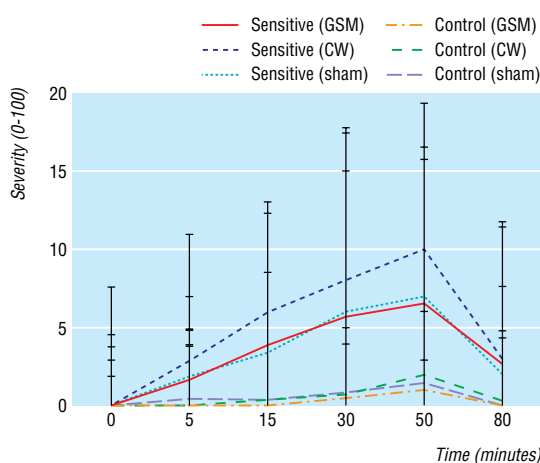
stopped early or the participants withdrew from the study. That apparently realistic symptoms can be induced in provocation experiments despite no difference being seen between active and sham conditions suggests that the acute symptoms reported by sensitive people in everyday life may be the result of a nocebo phenomenon. Such phenomena have previously been observed in relation to a wide range of stimuli,⁹ including headaches induced by providing misleading information about the presence of electrical fields.¹⁰ The mechanisms governing nocebo effects need further study but seem to include conscious expectation of symptoms and the presence of negative affect.^{11 12}

These results do not suggest that attempting to reduce exposure to mobile phone signals will be a useful strategy for patients who report sensitivity to them. In the longer term a danger exists that such a strategy will reinforce a patient's view of himself or herself as being sensitive to electromagnetic fields and put him or her at risk of developing symptoms associated with other electrical stimuli. Instead, it may be better to encourage patients to test alternative non-electromagnetic field related explanations for their symptoms by using principles derived from cognitive behavioural therapy.¹³

We thank everybody who participated in this study, especially those in the sensitive group. We also thank Phil Chadwick from MCL-UK for supplying and calibrating the exposure equipment, and the staff from the Mental Health and Neurology Clinical Trials Unit at the Institute of Psychiatry for doing the randomisation and double blinding of the study.

Contributors: See bmj.com.

Funding: This study was funded by the Programme Management Committee (PMC) of the Mobile Telecommunications and Health Research (MTHR) programme (www.mthr.org.uk), an independent body set up to provide funding for research into the possible health effects of mobile telecommunications. The MTHR is itself jointly funded by the UK Department of Health and the mobile telecommunications industry. The PMC contributed to the study design by proposing a reduction in overlap with other ongoing studies by focusing on symptom reporting, an increase in sample size, and an alteration in inclusion criteria to allow more highly sensitive people to participate. It had no



Median headache severity (error bars show interquartile range) during provocation with global system for mobile communication (GSM), continuous wave (CW), and sham exposures for sensitive and control participants. For clarity, graph does not include data relating to exposures that were terminated early, although these data were included in analyses

role in the collection, analysis, or interpretation of the data, writing of the report, or decision to submit the paper for publication. The views expressed in this paper are those of the authors and not necessarily those of the funders.

Competing interests: None declared.

Ethical approval: The South London and Maudsley NHS Trust Research Ethics Committee granted approval for the study.

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- doi 10.1136/bmj.38765.519850.55

DRUG POINTS

Nicorandil may be associated with gastrointestinal ulceration

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Nicorandil (Ikorel; Rhône-Poulenc Rorer, Guildford) is associated with mouth and anal ulcers,¹⁻³ but we are not aware of any previous reports of association with gastrointestinal ulceration. A 69 year old woman had had percutaneous coronary intervention and a stent inserted into her left anterior descending artery two years before presentation. She had recurrent angina, and her general practitioner increased her dose of nicorandil to 30 mg twice a day. She had another percutaneous coronary intervention for in-stent re-stenosis, after which she presented to her general practitioner with mouth and anal ulcers and was referred for gastrointestinal evaluation. She was taking aspirin, a β blocker, nitrate, and a statin. Upper and lower gastrointestinal endoscopy showed, in addition to her oral and anal ulcers, multiple ulcers of the small and large intestines and multiple biopsies showed non-specific ulceration. A provisional diagnosis of inflammatory bowel disease was made and she was due to be started on steroids and immunosuppressive treatment. She was seen in our clinic for cardiac review, and, at that time, she had anal ulcers and a large ulcer, 15 mm in diameter, at the base of her tongue. We stopped nicorandil, and her ulcers healed, and she had no further problems and remains well.

Full investigations to rule out other pathologies and causes of ulceration are important. Ulceration related to

nicorandil usually resolves itself on stopping nicorandil, but reducing the dose may promote ulcer healing.⁴ This is important as nicorandil is usually used as a third line treatment in patients with severe coronary artery disease, and it may not be possible to stop it completely without recurrence of anginal symptoms.

This case suggests that nicorandil might be associated with intestinal ulceration, in addition to ulceration of the mouth and anus, because no other cause was identified, and the ulcers resolved after stopping nicorandil. The Medicines and Healthcare Products Regulatory Agency's website lists two cases with possible relation to nicorandil: one of oesophageal ulceration and another of fatal small intestinal ulceration. Similarly, a report of perforation of the terminal ileum putatively linked it to nicorandil.⁵

Funding: None.

Competing interests: None declared.

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BMJ 2006;332:889

The 1918 influenza pandemic: a survivor's tale

Patrick Collins, my uncle, was a cavalry soldier in the first world war. In 1918 he was one of the few survivors in his Cheshire regiment.

When the influenza pandemic struck, it went through his camp like wildfire. Patrick noticed that virtually all those who stayed in camp died, presumably from the secondary staphylococcal pneumonia which killed so many. When he developed the first signs of influenza, he begged that he be allowed to leave camp with three days' rum ration and a tent. He managed to drag himself and his tent up a hill away from the camp, and there he

sweated and shivered and was delirious for several days, sustained by his rum allowance.

Patrick was one of the few survivors. He lived until the age of 92. He was a wonderful storyteller to his children, nephews and nieces, grandchildren, and great-grandchildren. The one story he never told us was this one. His eldest daughter, in whom he had confided, told the story at his funeral in 1987.

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