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Cutaneous mast cells are altered in normal healthy volunteers sitting in front of ordinary TVs/PCs – results from open-field provocation experiments

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Background: Considerable controversy has surrounded the question of possible biological responses to electromagnetic fields (EMFs) generated from visual display terminals (VDTs), such as personal computers (PCs) and ordinary television sets (TVs). The cellular and molecular mechanisms for such potential harmful health hazards have not yet been understood, although clues from the literature include mast cells and histamine. The aim of this study was therefore to investigate possible biological mast cell responses to TV/PC screens.
Methods: Using the indirect immunofluorescence technique, we studied the presence of histamine-containing mast cells in the dermis of healthy volunteers. Cutaneous biopsies taken before and after exposure to ordinary TV/PC screens for 2 or 4 h were investigated in 13 healthy subjects.
Results: Our present *in vivo* study indicates that normal cutaneous mast cells could be altered by exposure from ordinary TV/PC screens. To our great surprise, we found the number of mast cells in the papillary and reticular dermis to increase, to varying degrees, in 5 out the 13 subjects after such an exposure. A migration of mast cells towards the uppermost dermis appeared as the most important event. Thus, the normally upper "empty zone" of the dermis disappeared, and instead, a higher density of mast cells were found in this zone. These cells also seemed to have a tendency to increase in number towards the epidermal-dermal junctional zone and some of them lost their granular content and the cytoplasm shrunk (=degranulation). These findings could only be seen in the exposed skin. Two of the 13 cases instead showed a decrease in mast cell number, but the shift in mast cells towards the upper dermis was still visible. Twenty-four h after the provocation, the cellular number and location were normalized in all subjects.
Conclusions: By definition, normal healthy volunteers are assumed not to react to a TV/PC screen provocation. To our great surprise, this proved not to be true. The present results might lay a foundation to understand the underlying cause of so-called "screen dermatitis" with special reference to mast cells. However, blind or double-blind experiments using patients ought to be further investigated in order to find out the exact cause for the observed changes. Such causes include the effects of surrounding airborne chemicals, stress factors, etc.

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It has previously been assumed that three visual display terminal (VDT)-dependent factors potentially might affect human health: radiation, ergonomics, and stress factors.[1]

The X-ray radiation emitted from the VDT screen was, however, found to be far below accepted risk levels, even under "worst case" conditions, and has not been considered to be associated with any adverse health effects.[1] Considerable controversy has surrounded the question of possible biological responses to low- or high-frequency electromagnetic fields (EMFs). The mechanisms of any harmful health events, such as in so-called "screen dermatitis", have not been understood. Psychological stress factors are believed to be involved in the development of so-called "screen-induced symptoms".[2] Animal experiments have shown disturbances in endogenous opioid systems, with secondary effects on cholinergic systems, after exposure to low-frequency EMFs.[3] Pulsed EMFs imposed on developing chick embryos resulted in an increase in the frequency of abnormal developments.[4] Mammalian cell lines have been shown to respond to extremely low-frequency EMFs with an increase in the activity of a proliferation-regulating enzyme,[5] and stimulation of the calcium influx;[6] furthermore, EMFs could promote peripheral nerve regeneration both *in vivo* and *in vitro*.[7–12] All obtained data thus indicates that EMFs can directly affect biological systems through different, but yet, unclear pathways, including both physiological and biochemical ones.

Most recently, some interesting studies have been published, e.g. Henshaw et al.[13] revealed an enhanced deposition of radon (^{222}Rn) daughter nuclei (^{214}Po; ^{218}Po) in the vicinity of everyday sources of power-frequency EMFs in normal domestic room air. Since countries such as Sweden and the United Kingdom are very rich in ground-based radon as well as radon exposure from building materials, one may ask whether devices like VDTs are true α-emitters?

In this context one has to also mention the findings of Lai & Singh,[14] who investigated the effects of acute (2-h) exposure to pulsed (2 μs pulse width, 500 pulses/s) or continuous-wave 2,450 MHz radiofrequency electromagnetic radiation on DNA strand breaks in the brain cells of rats. An increase in both types of such DNA strand breaks was observed after exposure to either the pulsed or continuous-wave radiation. Again, it is natural to ask what happens with humans using high-frequency devices such as TVs/PCs, mobile telephones, light tubes, etc.?

It has been known for several years that mast cells can be involved in many physiological and pathological reactions in inflammation, allergy, urticaria, psoriasis, itch sensations and pain.[15–17] Cutaneous mast cells are involved in both type I and IV hypersensitivity reactions. In another case-control study (Johansson & Liu, unpublished data), the results in human skin clearly showed that cutaneous mast cells differed both in quantity, quality and distribution pattern in so-called "screen dermatitis" patients as compared to normal healthy volunteers.

Using the same approach in this study, we employed conventional PCs and household TVs as provocation tools to conduct an *in vivo* study on human normal healthy volunteers. The aim of this study was to investigate possible biological mast cell responses to TV/PC screens. By definition, normal healthy volunteers are assumed not to react to such a provocation. To our great surprise, this proved not to be true.

Material and methods

Subjects

Thirteen healthy volunteers (7 male and 6 female, aged 19–34 years old, average 25.7) were selected as the subjects in the study, which was approved by the Committee of Ethics at the Karolinska Hospital. All subjects had no history of dermatoses, allergic diseases or other somatic diseases, and they were all non-smokers.

Test parameters

An ordinary laboratory room without windows was used for the provocation experiment. The room was equipped with 5 conventional PCs (attached to their monitors), 2 ordinary household TVs and 1 portable TV. The temperature in the room was 23–24 °C, and the electric and magnetic fields had a strength of 85 V/m, 35 nA and 310 μT/s in the TVs/PCs "OFF"-position, and a strength of 250–500 V/m, 100 nA and >10,000 μT/s in the TVs/PCs "ON"-position,

as measured at the biopsy spot with a Friman Instrument MF-4 (size of measuring plate: 21.5 mm×65.5 mm; 1 m^2 coil (MF-3) and an RC nT-converting filter (no. 169; Friman Datakonsult AB, Stockholm, Sweden). The subjects were seated at a distance of 40 cm from the TVs/PCs with their backs facing the front of the TVs/PCs. The whole provocation lasted 2 or 4 h. Biopsies from the challenged area were taken in pairs at anatomically symmetric sites right before, immediately after the provocation and at 2, 4 or 24 h after the provocation.

Preparation of tissues

Routine punch (Kai Industries, Japan) biopsies (3 or 4 mm in diameter), including epidermis and dermis, were taken under local anaesthesia (Xylocaine, Astra, Sweden). The biopsies were fixed in 4% carbodiimide (1-ethyl-3–3-dimethylaminopropyl-carbodiimide (Sigma, St. Louis, MO, USA) diluted in phosphate buffer (pH 7.4)) for 2 h at 4 °C.[18] The tissue was then rinsed for at least 24 h in 0.1 M Sörensen's buffer containing 10% sucrose, 0.01% NaN_3 and 0.02% Bacitracin and then sectioned on a Microm cryostat to yield 14-µm-thick sections, thawed onto gelatine-coated slides and processed for indirect immunohistochemistry.

Immunohistochemistry

The indirect immunofluorescence technique was employed.[18,19] The sections were kept in an humid atmosphere, incubated with the primary rabbit histamine antiserum (1:2,000; Milab AB, Malmö, Sweden) overnight at 4 °C, rinsed in phosphate-buffered saline (PBS), incubated for 30 min at 37 °C in rhodamine (TRITC)-conjugated goat anti-rabbit IgG (1:80; Boehringer-Mannheim, Mannheim, Germany), rinsed and mounted. All antisera were diluted in PBS containing 0.3% Triton X-100.

The control of the antiserum specificity was performed by pre-absorption with histamine dihydrochloride (0.1 mM; Sigma). In addition, it has been verified by the manufacturer that the antiserum does not cross-react with norepinephrine, serotonin, vasoactive intestinal polypeptide, glucagon or histidine. To test for any possible non-specific binding of the primary antiserum to Fc receptors in the tissue, a normal rabbit serum (1:100) was used instead of the primary antibody. To control for any possible non-specific reactions of the secondary antiserum, PBS was used on certain sections instead of the primary antibody. Other sections were directly only incubated with the secondary antibody. For observation and photography a Nikon Microphot-FXA fluorescence microscope was used. The results were collected from two independent observers.

Results

First, it should be noted that no cutaneous and/or somatic objective or subjective symptoms at all were induced in or reported by the tested volunteers during and after the provocation. This is fully in accordance with the assumption that normal healthy volunteers should not react to the TV/PC provocation situation at all.

The histamine immunoreactivity was only found in cells of the dermis. The immunoreactive cells observed in this study are considered to be mast cells based on their localization, number and cellular morphology, e.g., size and arrangement of granules according to our previous investigations.[18,19] They were seen around dermal appendages such as hair follicles, sebaceous glands, sweat glands and blood vessels, and they all had a similar morphology. Although mast cells were frequently found close to small capillaries and large blood vessels, they were never actually pres-

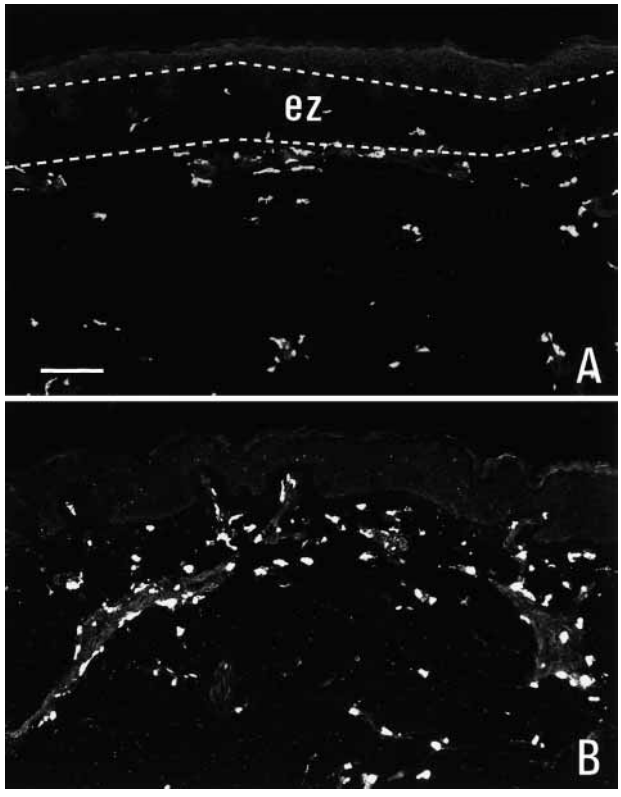

Fig. 1. The distributional alteration of cutaneous mast cells due to the provocation. A) An unexposed biopsy illustrates the relatively empty zone (ez) below the epidermal-dermal junction, where only single cells are seen. Further below this empty zone, mast cells are found in their highest density and gradually decrease towards the deeper dermis. B) In an exposed skin, a cellular migration upwards to the uppermost dermis was induced after exposure, i.e. the normally empty zone disappeared, and, instead, a high density of mast cells is observed in this zone. Bar in A=50 µm. The magnification in A=B.

ent in the vascular wall. The immunolabelling was found exclusively in the cytoplasm of the cells, leaving the ovoid single spherical or oblate spherical nucleus unlabelled, generally displaced to one side of the cells and along the long axis of cell (Fig. 1A). All the positive cells appeared prominently granular and most of them were elongated with cytoplasmic extensions while others were flat or dendritic when examined under high magnification.

In all unexposed biopsies, a relatively empty zone could be seen, which was approximately 100–200 μm wide below the epidermal-dermal junctional zone running parallel to that border (Fig. 1). One could hardly see any mast cells in that zone. This is consistent with our previous results in normal facial skin (Johansson & Liu, unpublished data). Further below the empty zone, mast cells were found in their highest density and gradually decreased towards the deeper dermis.

The main experimental results registered after exposure to the TVs/PCs are shown in Table 1. To our great surprise, the numerical density of mast cells increased in 5 out of the 13 subjects in the papillary dermis and reticular dermis as a response to the provocation; however, the cellular volume seemingly remained unchanged in most cases. Two out of 13 also increased the fluorescence intensity of their histamine granules. Above all, a cellular migration appeared as the most important event in the provocation experiment, i.e. the normally empty zone disappeared, and instead a high density of mast cells was observed in this zone. These cells also seemed to have a tendency to move towards the epidermis and some of them lost their granular content and their cytoplasm shrinked (pointing to a possible degranulation) (Fig. 2).

In contrast to the results mentioned above, two cases showed a numerical mast cell decrease, but the upward movement was still visible. In addition, in one case, the processes of mast cells were found to be

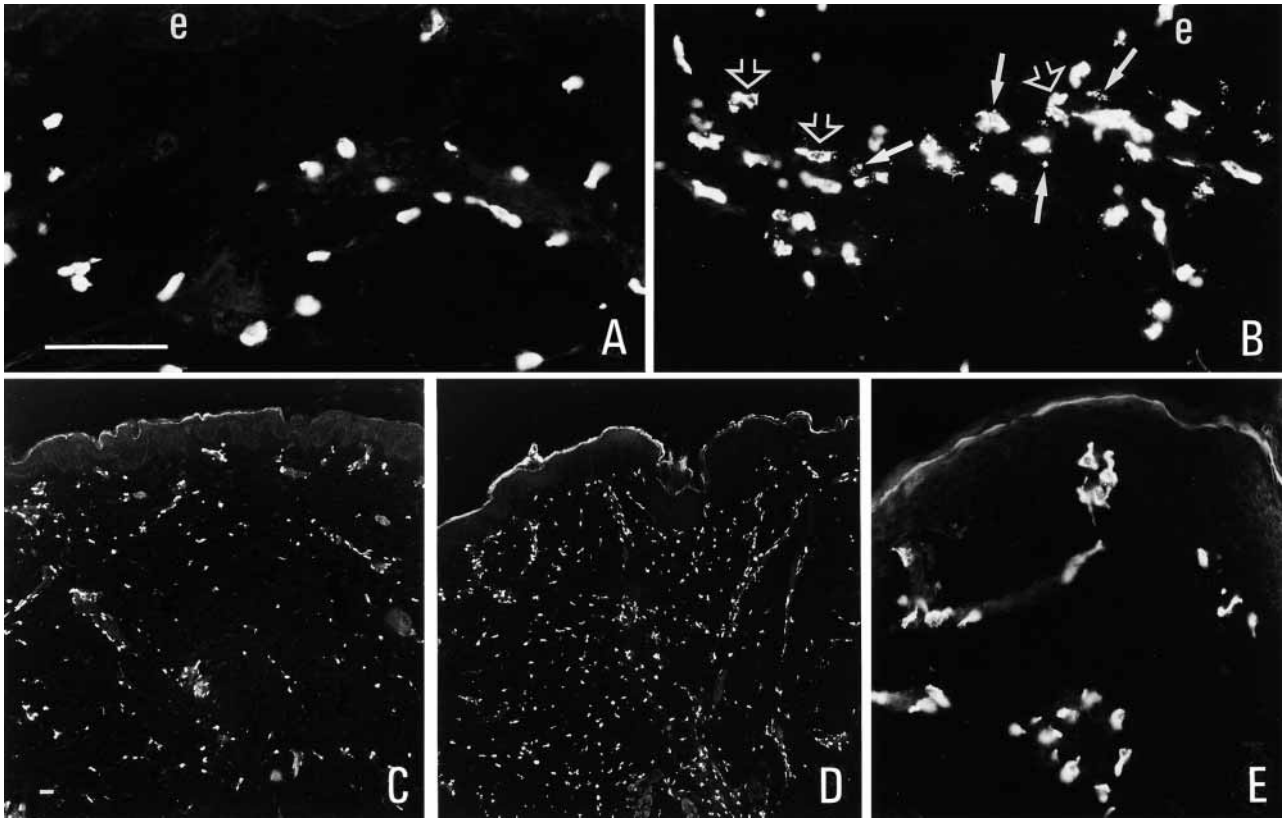

Fig. 2. A typical cutaneous response to TV/PC provocation. Note that biopsies A & C and B & D, respectively, came separately from the same subject. In A, the unexposed biopsy shows the morphology of normal status mast cells. In B, a biopsy from after 4 h of exposure, it is shown that these cells close to the epidermis have degranulated and that their cytoplasm has shrinked. Open arrows point to shrinked mast cells; solid arrows point to discharged histamine granules from mast cells. e=epidermis. C and D are low-power micrographs clearly demonstrating that compared to before (C), after 4-h exposure (D) the number of mast cells has increased and an upward movement of these mast cells has appeared. In E, one case showed an odd morphology of mast cells as a response to the provocation; the processes became more numerous as well as larger, especially in cells that were situated closer to the epidermis. Bars in A and C=50 μm. The magnification in A=B=E, and in C=D.

Table 1. The protocols of cutaneous mast cell variation after 2 or 4 h screen provocation compared with unexposed biopsies

Subject	2 or 4 hours after provocation				After a 24-h delay			
	No.	CV	GI	McM	No.	CV	GI	McM
1.	↑↑	–	-	3+	–	–	–	0
2.	↑↑	↑	–	1+	–	–	–	0
3.	↑↑	–	–	1+	–	–	–	0
4.	↓	–	–	1+	–	–	–	0
5.	–	–	–	1+	–	–	–	0
6.	–	–	↑	0	–	–	–	0
7.	–	↑	–	0	–	–	–	0
8.	–	–	–	0	–	–	–	0
9.	–	↑	↑	2+	–	–	–	1+
10.	↓	–	–	1+	–	–	–	0
11.	↑↑	–	–	1+	–	–	–	1+
12.	–	–	–	0	–	–	–	0
13.	↑↑↑	–	–	2+	↑	–	–	0

–=no change; ↑=increase; ↓=decrease; 0=not present; +=was induced to a mild (1+), medium (2+) or strong (3+) degree; CV=cellular volume; GI=granular intensity; McM=mast cell migration into the uppermost dermis

more numerous as well as larger, especially in cells that were situated closer to the epidermis, as a response to the provocation (Fig. 2E). The morphology of the cutaneous capillaries, sebaceous glands as well as hair follicles always remained unchanged. Finally, it should be noted that 24 h later, all of the subjects with changes reported above revealed a normal pattern.

There was no significant difference between biopsies that were taken instantly after exposure or after 2 or 4 h post-provocation delay. Furthermore, no differences could be seen if the provocation time was 2 or 4 h.

Discussion

Mast cells are effectors of IgE-mediated immune reactions because they have high-affinity receptors for the Fc-portion of IgE.[20] Their ability of rapid response to allergens or poison stimuli may be considered as a first line of defence in protecting the skin from being infiltrated by micro-organisms and other potentially harmful agents. They contain and release, after activation, a wide array of pro-inflammatory mediators affecting structures and cells, and conversely their differentiation and function are affected by their environment.

Histamine is synthesized in mast cells from histidine and is stored within the mast cell secretory granules by forming a complex with the glycosaminoglycan side chains of heparin. In mammalian connective tissue, including the skin, mast cells have generally been regarded as the major source of histamine.[21] It has been found that there were statistically significant correlations between the mast cell number and histamine content.[19,21–24] Therefore, most of the histamine immunoreactivity cells are equal to classical mast cells.

In the present study, we found a special distribution in the papillary dermis, i.e. an empty zone could easily be identified, like in the face (Johansson & Liu, unpublished data), which has not been reported in the back before. This zone may be considered as a 'buffer' zone for immune reactions; once an antigenic substance or stimulus intrude into this area, immunocytes (as well as mast cells) migrate into this zone to participate in the immunological response.

Apart from the fact that mast cells could be provoked to increase their infiltration in the dermis, another prominent finding in this investigation is, of course, the upward migration. It could be induced in most cases (9 out of 13; see Table 1), although two cases showed a decrease of mast cell number. In this context, it may be noted that Donnellan et al.[25] have shown clear-cut effects on a mast cell analogue, RBL-2H3, of EMFs at 835 MHz. The rate of DNA synthesis and cell replication increased, the actin distribution and cell morphology became altered, and the amount of β-hexosaminidase released in response to a calcium ionophore was significantly enhanced, in comparison to unexposed cultures. There were no effects seen on the levels of cytoskeletal protein synthesis or β-actin mRNA. However, the amount of Ras in the membrane fraction of exposed cells increased. The morphological changes persisted following subculture for at least 7 days in the absence of further exposure.[25] This work has now also been extended to yet another mast cell line, namely the HMC-1, and at 864.3 MHz.[26] In their study, the authors reported effects on the localization of the protein kinase C, and expression of 3/588 genes screened. The affected genes included the proto-oncogene c-kit, the transcription factor nucleoside diphosphate kinase B and the apoptosis-associated gene DAD-1. In addition, stress response genes were variably upregulated. No significant effect on cellular morphology or on F-actin distribution was detected. The conclusion of the publication[26] was that the low-power microwave exposure used may act on the HMC-1 cells by altering gene expression via a mechanism involving activation of protein kinase C, and at temperatures (=26.5 °C) well below those known to induce heat-shock responses.

The recent finding that small magnetic particles of magnetite (Fe_3O_4) are present in various biological tissues opens the arena for new speculations on interaction mechanisms.[27] A cluster of cells, denoted magnetocytes or Jurkat cells (a human leukemic T-cell line)[27] and bacteria *Magnetospirillum magnetotacticum* have been reported to contain magnetite, and low-frequency magnetic fields have been shown to increase inositol 1,4,5-triphosphate levels in the Jurkat cell line.[28] Earlier reports from the same group have shown that when a weak 50-Hz magnetic field was applied, the Jurkat cells responded with intracellular

calcium oscillations.[29] The results suggested that the magnetic fields interfered with the signal transduction, although neither target molecules nor molecular mechanisms are at present known. Perhaps some cutaneous mast cells also may contain magnetite and such cells may be involved in possible interactions of environmental EMFs and skin. When there exists an EMF, they might migrate toward the magnetic source, i.e., towards the epidermis, to degranulate their histamine content. Since this effect only happens at the cellular level, it may not be strong enough to immediately cause cutaneous objective/subjective symptoms of long-lasting nature. Support for this idea may be given by the observation that, even if a subject had cellular changes directly after the provocation, 24 hours later they had become normal again (cf. Table 1). But, repeated chronic exposure to magnetic fields may be able to cause symptoms such as itch, smarting, redness, papules, etc.[30] This is correspondent to the fact that mostly when the so-called "screen dermatitis" patients leave their VDT work, the symptoms are relieved or, more or less, disappear.

In conclusion, although the subjects in the present investigation did not report any cutaneous and/or somatic objective or subjective symptoms during as well as after the provocation, 7 out of 13 still had profound cellular changes in their dermal mast cell population. This would mean that normal human skin very well could biologically react to external EMFs generated from TVs/PCs. What this would imply for the whole human being could only, at this stage, be guessed at. Since the subjects did not verbally complain it is not likely that psychological and/or psychosomatic events have taken place, rather the cellular changes point to a UV, microwave, or other high-frequency radiation emitted by the TVs/PCs. The assumption that normal healthy volunteers should not react at all to the TV/PC provocation situation thus proved to be wrong. And, maybe the observed cellular changes are actually just plain radiation damages?

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References

1. Wheater RH. Health effects of video display terminals. Council of Scientific Affairs, American Medical Association, Chicago. JAMA 1987; 257: 1508.
2. Lidén S, Berg M. Skin problems in users of video display terminals. Discrepancy between subjective symptoms and objective signs. Acta Derm Venereol (Stockh) 1991; Suppl 156: 18.
3. Lai H, Carino MA, Horita A, Guy AW. Effects of a 60 Hz magnetic field on central cholinergic systems of the rat. Bioelectromagnetics 1993; 14: 5.
4. Litovitz TA, Montrose CJ, Doinov P, Brown KM, Barber M. Superimposing spatially coherent electromagnetic noise inhibits field-induced abnormalities in developing chick embryos. Bioelectromagnetics 1994; 15: 105.
5. Mattsson M-O, Mild KH. Biological effects of electromagnetic fields – Studies of gene regulation and proliferation characteristics [Abstract]. Contractors Review. Stockholm: AMFO, 1994.
6. Barbier E, Dufy B, Veyret B. Stimulation of Ca^{2+} influx in rat pituitary cells under exposure to a 50 Hz magnetic field. Bioelectromagnetics 1996; 17: 303.
7. Rusovan A, Kanje M. Stimulation of regeneration of the rat sciatic nerve by 50 Hz sinusoidal magnetic fields. Exp Neurol 1991; 112: 312.
8. Rusovan A, Kanje M, Mild KH. The stimulatory effect of magnetic fields on regeneration of the rat sciatic nerve is frequency dependent. Exp Neurol 1992; 117: 81.
9. Rusovan A, Kanje M. D600, a Ca^{2+} antagonist, prevents stimulation of nerve regeneration by magnetic fields. NeuroReport 1992; 3: 813.
10. Rusovan A, Kanje M. Magnetic fields stimulate peripheral nerve regeneration in hypophysectiomized rats. NeuroReport 1992; 3: 1039.
11. Sisken BF, Kanje M, Lundborg G, Herbst E, Kurtz W. Stimulation of rat sciatic nerve regeneration with pulsed electromagnetic fields. Brain Res 1989; 485: 309.
12. Sisken BF, Kanje M, Lundborg G, Kurtz W. Pulsed electromagnetic fields stimulate nerve regeneration *in vitro* and *in vivo*. Restorative Neurol Neurosci 1990; 1: 303.
13. Henshaw DL, Ross AN, Fews AP, Preece AW. Enhanced deposition of radon daughter nuclei in the vicinity of power frequency electromagnetic fields. Int J Radiat Biol 1996; 69: 25.
14. Lai H, Singh NP. Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation. Int J Radiat Biol 1996; 69: 513.
15. Goldstein SM, Wintroub BU. The cellular and molecular biology of the human mast cell. In: Fitzpatrick TB, Eisen AZ, Wolff K, Freedberg IM, Austen KF, eds. Dermatology in general medicine. 4th edition. New York: McGraw-Hill, 1993; 359.
16. Keele CA. Chemical causes of pain and itch. Ann Rev Med 1970; 21: 67.
17. Mayrhofer G, Pitts R. A comparison of some properties of mast cells and other granulated cells. In: Befus AD, Bienenstock J, Denburg JA, eds. Mast cell differentiation and heterogeneity. New York: Raven Press, 1986; 141.
18. Johansson O, Virtanen M, Hilliges M, Yang Q. Histamine immunohistochemistry: a new and highly sensitive method for studying cutaneous mast cells. Histochem J 1992; 24: 283.
19. Johansson O, Virtanen M, Hilliges M, Yang Q. Histamine immunohistochemistry is superior to the conventional heparin-based routine staining methodology for investigations of human skin mast cells. Histochem J 1994; 26: 424.
20. Miller L, Blank U, Metzger H, Kinet J-P. Expression of high-affinity binding of human immunoglobulin E by transfected cells. Science 1989; 244: 334.

21. Graham HT, Lowry OH, Wahl N, Priebat MK. Mast cells as sources of tissue histamine. J Exp Med 1955; 102: 307.
22. Eady RAJ, Cowen T, Marshall TF, Plummer V, Greaves MW. Mast cell population density, blood vessel density and histamine content in normal human skin. Br J Dermatol 1979; 100: 623.
23. Riley JF, West GB. The presence of histamine in tissue mast cells. J Physiol 1953; 120: 528.
24. Riley JF, West GB. Skin histamine. Its location in the tissue mast cells. Arch Dermatol 1956; 74: 471.
25. Donnellan M, McKenzie DR, French PW. Effects of exposure to electromagnetic radiation at 835 MHz on growth, morphology and secretory characteristics of a mast cell analogue, RBL-2H3. Cell Biol Internat 1997; 21: 427.
26. Harvey C, French PW. Effects on protein kinase C and gene expression in a human mast cell line, HMC-1, following microwave exposure. Cell Biol Internat 2000; 23: 739.
27. Galt S, Ottoson U, Bergqvist B, et al. Magnetite – a possible interaction mechanism [Abstract]. Contractors Review. Stockholm: AMFO, 1994.
28. Korzh-Sleptsova IL, Lindström E, Mild KH, Berglund A, Lundgren E. Low frequency MFs increased inositol 1,4,5-triphosphate levels in the Jurkat cell line. FEBS Lett 1995; 359: 151.
29. Lindström E, Lindström P, Berglund A, Mild KH, Lundgren E. Intracellular calcium oscillations induced in a T-cell line by a weak 50 Hz magnetic field. J Cell Physiol 1993; 156: 395.
30. Johansson O, Liu P-Y. "Electrosensitivity", "electrosupersensitivity" and "screen dermatitis": preliminary observations from on-going studies in the human skin. In: Simunic D, ed. Proceedings of the COST 244: Biomedical Effects of Electromagnetic Fields – Workshop on Electromagnetic Hypersensitivity. Brussels/Graz: EU (DG XIII), 1995; 52.