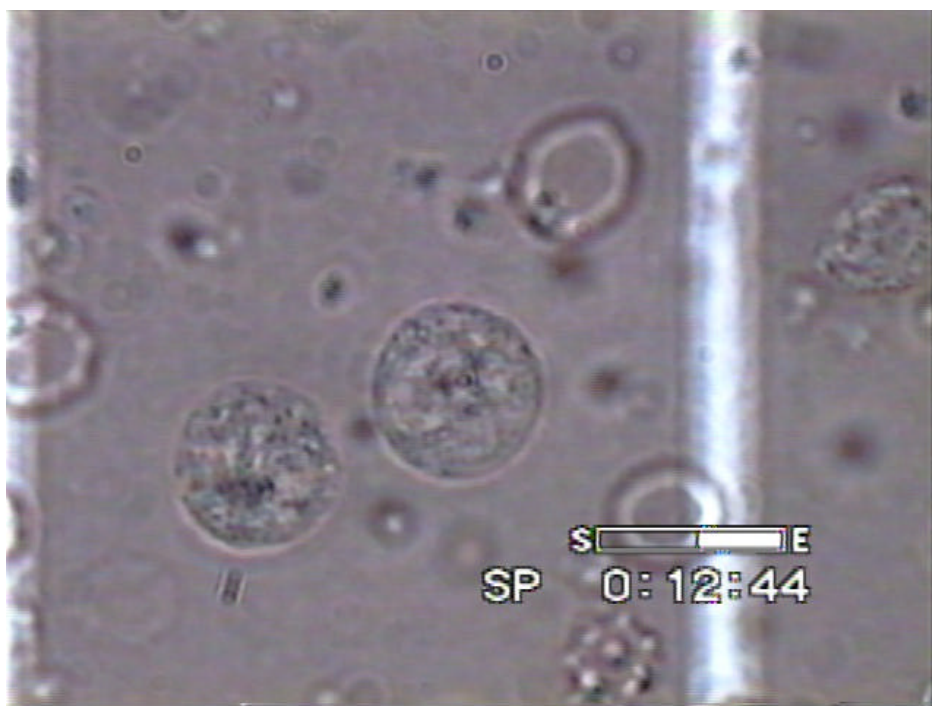


**PROTECTIVE EFFECT  
OF A CRYSTALLO-METALLIC DEVICE  
ON HUMAN PERIPHERAL BLOOD  
LYMPHOCYTES  
EXPOSED TO GSM CELLPHONE HANDSET  
RADIATIONS**



*Dying (centre left) and healthy (centre) lymphocytes.  
A crenating erythrocyte is seen at the bottom of the micrograph (x 960).  
The vertical bar is the haemocytometer gridline (width c. 3 microns).  
The timecode is shown bottom right.*

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## INTRODUCTION

Large scale and rapidly increasing public demand for mobile telephony services has meant a proliferation of cellphone handsets and their associated base stations throughout the United Kingdom and, indeed, throughout the world. There are presently some 30 million UK users, a quarter of whom are under 18 years old, and some 500 million users worldwide. Virtually none of these existed two decades ago, which in terms of human evolution is a negligible time period.

Ever since the introduction of radar in WW2, both public and scientific observation have led to concerns over the possible health hazards of exposure to the radiations inevitably emitted by these installations. Even by the 1970s, a US Congress Committee (ERMAC) concluded that unless an urgent research programme was instituted, industrialised societies may be exposed to adverse *sequelae* affecting the entire population.

Many peer reviewed studies now confirm that RF/MW radiations at levels far below those needed for thermal insulat and well below the present regulatory guidelines are capable of adverse effects on *in vitro* cells, on tissues and organs, on whole live animals, on human beings in a laboratory-controlled environment, and on people in their epidemiological setting. (See Cherry, June 2000 for review). Such studies identify at least nine biophysical endpoints (Table 1).

**Table 1** Example studies of ill health effects

calcium ion efflux	Bawin & Adey, 1976; Blackman, 1985, 1990
melatonin reduction	Kolomytkin et al., 1995; Rosen, Barber & Lyle, 1998
DNA strand breakage	Lai & Singh, 1997a; Svedenstal et al., 1998
chromosomal aberrations	Garag-Vrhovac et al., 1993; Maes et al., 1993
leukaemia	Dolk et al., 1997a,b; Michelozzi, 1998
solid tumours of brain	Balcer-Kubiczek & Harrison, 1991; Szmigielski, 1991
immune deficiency	Bruviere et al., 1998; Dmoch & Moszczynski, 1998
miscarriage	Lindbohm et al., 1992; Magras & Xenos, 1997
neurological effects	Lilienfeld et al., 1978; Beale, 1997; Mild et al., 1998

These studies are merely representative of a much larger body of evidence. Nevertheless, despite intensive and mainly industry-guided/funded research, the regulatory authorities (ICNIRP, NRPB etc.) seem reluctant to recommend guidelines based on exposures at below thermal insult levels, while accepting that biological effects do exist below these levels.

The Government-appointed Stewart Committee Independent Expert Group reported on the possible health hazards of handsets and masts in May 2000. It recommended a policy of prudent avoidance in line with the European environmental legislation embodied in the 1993 Maastricht Treaty, which states:

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***Title XVI***

***Environment***

***Article 130r***

1. Community policy on the environment shall contribute to pursuit of the following objectives:
- protecting human health
  - preserving, protecting and improving the quality of the environment

The Precautionary Principle is a risk management policy applied in circumstances with a high degree of scientific uncertainty, reflecting the need to take action for a potentially serious risk without awaiting the results of scientific research.

**ALSO**

For countries of the European Union, the Treaty of Rome states that  
*“Community policy on the environment...shall be based on the Precautionary Principle”.*

**The scientific evidence has naturally been challenged by those whose commercial interests might be damaged by acknowledging this evidence. In May 2000, the UK Institution of Electrical Engineers, whose members encompass the power utilities and the telecommunications industries, continued to deny that such effects exist, thereby occupying an increasingly isolated position (Barker et al., 2000).**

**At the same time, a bewildering variety of protective devices have arrived in the marketplace, the operation of many is seldom explained in terms acceptable to normal science. Moreover, such devices have rarely been supported by peer-reviewed publications, and these twin disadvantages have led to scepticism and purchasing resistance.**

The emerging scientific evidence indicates that there is clearly a need for effective devices to protect against adverse EM field and radiation effects

(e.g. Barnothy, 1964; Coghill Steward et al., 1996). This has often been observed both at cellular (Lyle et al., 1983, 1988) and live animal level (de Pomerai et al., 2000) and for a variety of artificially originating frequencies well below those that produce a thermal effect (Szmigielsky, 1988). There is a need to investigate such devices by means of studies using established scientific methods and to formulate ways to explain how they work.

The human peripheral blood lymphocyte has been widely used as a model for assessing the impact of noxious agents, particularly ELF and RF/MW electromagnetic fields. Therefore, they may also be used to assess the extent of protection against these agents within normal science (tests such as applied kinesiology or aura inspection, though often producing impressive results, are not regarded as scientifically acceptable). Moreover, tests for lymphocyte viability are also commonly used and reported in scientific literature.

Against this background, it would seem prudent for the users of mobile telephony handsets to protect themselves by using a device which aims to reduce the adverse effects of cellphone radiation. This study sought to evaluate the device known as Gem Chip (Spiral of Tranquillity), manufactured and marketed by Michael Poynder, using the human peripheral blood lymphocyte as a model.

### **Description of the device**

The Gem Chip consists of a number of organic elements in particulate form, including lithium, boron, sodium, magnesium, aluminium, silicon, phosphorus, potassium, calcium, titanium, chromium, iron, copper, and zinc. These are contained in a plastic droplet with a diameter of 1 cm, and placed on a self-adhesive backing strip for attachment to the front of the handset, near the antenna. Photo 1 shows the device placed on a Philips C12 handset for use in the experiment.

*Protective effect of a device on lymphocyte competence in vivo.*



Photo 1

*Close up of the device. The words "The Spiral of Tranquillity" are written around the circumference. The device is 10 mm in diameter.*



Photo 2

*The device in place on the front of a Philips C12 handset, near the antenna. A gold wire leads from the antenna to the sealed lymphocyte culture sample. The handset is held upright while on stand-by using a non-metallic clamp.*

## **METHOD AND MATERIALS**

A 30 ml whole blood sample from a healthy male donor drawn via *v. cubitale* and the peripheral blood lymphocytes were isolated. Firstly, the whole blood was centrifuged twice at 800 g for 30 min and the buffy coat was removed under a laminar flow unit (Labcaire, Cliveden, Avon, UK) to preserve sterile conditions. This was then diluted (1/1) with a balanced saline solution with added glucose and recentrifuged several times at 100 g for 5–7 mins with Histopaque. The white blood cell gradient was removed by micropipetter and re-suspended in RPMI with added antibiotics and antimycotics (Sigma Chemicals, Gillingham, Dorset). The cells were inspected for integrity using light microscopy (Olympus BX50) and left to rest in a double shielded mu-metal container for 3 hours at 20 degrees Centigrade.

Exposure to EM fields and radiation from three identical, fully-charged cellphones (Philips C12) on standby for 14 hrs was achieved by means of a 30 cm gold wire that connected each cellphone to 0.3 ml samples of the isolate, each in sterilized and sealed 2 ml glass phials, under the following conditions:

- a) **Sham exposed.** The phial contained 0.3 ml of the culture and an embedded gold wire which did not protrude from the interior. These were then enclosed in a mu-metal box inside a double skinned mu-metal container maintained at 37° Centigrade ( $\pm 0.1^\circ$  Centigrade). The “exposure” period was 14 hrs. The cellphone was not switched on.
- b) **Protected.** A phial containing 0.3 ml of the same culture was attached to a Philips C12 cellphone on standby for 14 hrs by means of a 30 cm gold wire using non-conducting tape, and the device (Spiral of Tranquillity Gem Chip) was attached to the handset according to maker’s instructions (see Photo 2).
- c) **Positive Control.** A phial containing 0.3 ml of the same culture was attached by means of a 30 cm gold wire to an identical Philips C12 cellphone on standby for 14 hours in a separate room.

After the exposure period, the three samples from the culture were allowed to rest for 24 hours, removed to a laminar flow unit, coded by a third party not connected with the study, and tested blind for competence by means of a cluster test.

### **Lymphocyte competence**

One common assay for testing lymphocyte competence is their response to mitogenic stimulation, where commonly used mitogens include horseradish peroxidase, PHA, concanavalin A, etc. A second type of test is to see whether the cell is viable (i.e. with intact plasma membrane) using a penetrating dye such as trypan blue. Morphologic inspection under a light microscope also establishes whether the lymphocyte is active or not, as the interior can clearly be seen pulsating, but this method is tedious and time consuming.

However, activated cells routinely attach to other cells in which they are interested, including other dead cells of the same type. It is therefore commonplace to find a live and competent lymphocyte attached to or near to a dead lymphocyte, as shown by dye infiltration. In this study, this characteristic was used to compare the numbers of clustered cells per total lymphocyte count as a measure of competence. Cells were considered to be clustered if they were attached to at least one other non-erythrocyte. For each sample, the four outer grids of a haemocytometer (Sigma Brightline, Gillingham, Dorset) were counted as well as the centre grid, in accordance with manufacturer’s instructions.

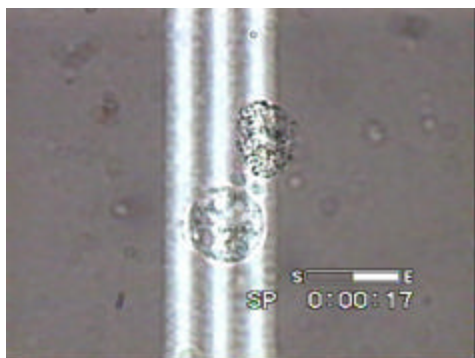


Photo 3.  
*A live lymphocyte (lower cell) investigating a dead cell. This would count as one cluster in this study. The three vertical bars are part of the haemocytometer grid (Sigma Brightline), and are about ten microns across. The time coding is shown on the bottom right for video referencing. Movements of the lymphocytes were also captured as \*.avi computer files to show their liveliness.*

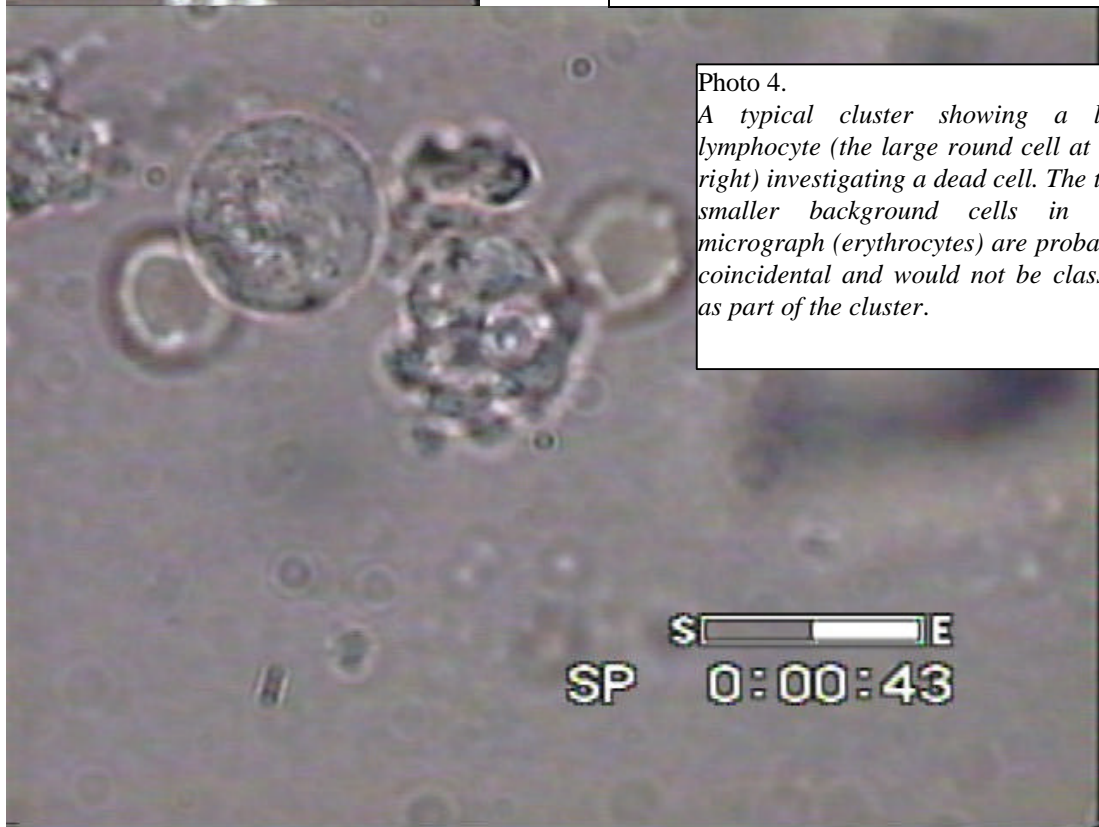


Photo 4.  
*A typical cluster showing a live lymphocyte (the large round cell at the right) investigating a dead cell. The two smaller background cells in the micrograph (erythrocytes) are probably coincidental and would not be classed as part of the cluster.*

## RESULTS

The results of the cell counts are set out numerically below (Table 2)

**Table 2: Haemocytometer Grid Lymphocyte counts**

<b>Grid</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>
<i>Exposed</i>					
Total (a)	37	48	65	57	113
Clusters (b)	8	13	9	17	12
a/b (%)	21.6	27.1	13.8	29.8	10.6

*Protective effect of a device on lymphocyte competence in vivo.*

<b><i>Protected</i></b>					
Total (a)	14	11	5	13	10
Clusters (b)	2	6	2	5	2
a/b (%)	14.3	54.5	40.0	38.5	20.0
<b><i>Sham Exposed</i></b>					
Total (a)	27	26	27	10	9
Clusters (b)	8	8	9	4	3
a/b (%)	29.6	29.6	33.3	40.0	33.3

To test whether these data are statistically significant, the correct test is the difference between proportions (Ball and Buckwell, 1991 (2<sup>nd</sup> Ed.) p. 151). The test statistic is:

$$z = \frac{(p_1 - p_2) - 0}{\text{standard error}}$$

where the standard error is

$$\sqrt{\frac{\pi(1-\pi)}{n_1} + \frac{\pi(1-\pi)}{n_2}}$$

$$\text{and } \pi = p = \frac{n_1 p_1 + n_2 p_2}{n_1 + n_2}$$

The calculation is  $p = (59 + 17)/(320 + 53) = 0.204$

so the standard error is  $\sqrt{(0.204 \times 0.796)/320 + (0.204 \times 0.796)/53}$

thus  $SE = 0.060$ , therefore  $z = (0.184 - 0.321 - 0)/0.060 = -2.28$

Since  $z$  is less than  $-1.645$  (the level for significance in a one-tailed test at 5%), we can reject the null hypothesis in this case, and the data are significantly different.

## **DISCUSSION**

Table 2 shows a significantly lower mean proportion of clusters in the exposed group (18.4%) compared with the unexposed (sham exposed) group (32.3%), implying that the exposed lymphocytes were less competent or less active. Given that the protected cells were exposed to the same level of radiation for the same period, the null hypothesis would argue that there should be no difference in competence levels if these are reflected in cluster formations. However, the cluster proportion of the protected group was much higher than 18.4%, namely 32.1%, and very similar to the sham exposed sample.

The second notable feature is the evident variation in the numbers of cells per unit volume in each of the samples: the exposed sample cell count was 320, whereas the sham exposed count was only 99 and the protected group was even less at only 53. These differences require explanation. One possibility is that the procedure was not uniform. In each case, however, the cultures were well stirred before the samples were taken from both the mother culture and from the individual phials, and as a result one would not expect such consistent differences in the counts.

A second possibility is that the cellphone-exposed lymphocytes multiplied in response to the radiation stimulus by multiplying, the same way it would react to a mitogen, whereas the “protected” cells did not perceive the stimulus as any such stressor.

The literature provides some support for this second possibility: lymphocytes are reported to multiply more than usual in the presence of ELF EM fields (Mooney, Smith et al., 1986; Emilia, Torrelli et al., 1985; Cantini, Cossarizza, et al., 1986; Cossarizza, Monti et al., 1989). However, their competence is also lowered with such exposures (Lyle, Ayotte et al., 1988).

There are a number of scientific studies pointing to adverse stressor-like bio-effects from radiation at levels that are lower than those required for inducing thermal effects. Though by no means complete, the following brief overview describes the main evidence that devices such as that investigated here should be used by the public.

### **Subcellular studies**

Damage to DNA (mutagenic as opposed to genotoxic action) is expressed in several ways including the number of single or double strand breaks, chromosomal aberrations, or sister chromatid exchange (SCE). Only one recent study (on human lymphocytes from RF antenna workers) found increased chromosomal aberration (Maes et al., 1993, 1995). Moreover, most recent studies examining effects on sister chromatid exchange have used a lymphocyte model. Stewart reviewed eight of these but only one found any increased frequency of SCE post exposure (Khalil et al., 1993). By contrast, four of five studies on micronuclei formation did find effects.

Three of four recent studies on DNA reported effects. The most frequently discussed subcellular studies are those of Drs Henry Lai and N. P. Singh from Washington University, Seattle, who used a novel assay (the Comet assay) and reported both single and double strand breaks in rat brain tissues after 4 hours exposure at 2.45 GHz (equivalent to an SAR of 0.6 and 1.2 W/kg). The effect has not been replicated other studies (Malyapa et al., 1997a,b), but some questions remain over the differences in protocol used in the studies.

Not so well publicised, but with greater support for replication, are the studies on ornithine decarboxylase (ODC), a key enzyme that regulates the synthesis of substances called polyamines, which can trigger DNA synthesis, cell growth, and differentiation. These studies report a rise in ODC levels in response to amplitude modulated MW (Byus et al., 1988; Litovitz, et al., 1993; Penafiel et al., 1997).

Gene expression studies have focused on the c-fos and c-jun oncogenes. Out of six studies reviewed by Stewart, only one (Walters et al., 1995) found no effect. Most effects were seen when the SAR levels were around 1.5 W/kg or higher. However De Pomerai and colleagues at Nottingham University reported that after 7 hrs exposure to a mobile phone at 750 MHz, the heat shock proteins of a transgenic nematode worm were elevated (Daniells et al., 1998; de Pomerai et al., 1999).

Though evocative and important when taken together, these subcellular studies alone do not mean that bio-effects from cellphone or mast exposure are harmful. They do, however, convince that bio-effects are possible well below thermal limits.

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## **Cellular studies**

These are the easiest, cheapest, and quickest to carry out, but the standard criticism is that it is not easy to extrapolate the effects on live people from an *in vitro* study. Nevertheless, a large number of cellular studies taken together present a formidable argument, and this is what is already in place when considering the bio-effects of base stations. Typical of those in the literature is that by Siannette Kwee who used cells from human amniotic fluid and human skin fibroblasts to report that after only 20 minutes exposure to a 960 MHz radiation RF field of only 0.021–0.21 SAR mW/kg (about the same strength as mobile phone base stations and much lower than cellphone emissions), a decrease in cell growth occurred compared with controls (Kwee and Raskmark, 1998).

A similar study from Moscow, Russia (Gapeyev, Kolomyceva et al, 2000) reported that after 20 minutes exposure at 150  $\mu\text{W}/\text{cm}^2$  and 42 GHz, the phagocytic activity of human neutrophils declined by 50%. Most of the white blood cells of the immune system are neutrophils, which act by encapsulating and ingesting the toxin of interest (phagocytosis). The effect was far faster than suppression caused by injecting the chemical carbon tetrachloride.

### **Live animal studies**

These are expensive and generally long term projects which often depend on the life span of the chosen animal model. The criticism of such studies, arguably more pertinent in radiation studies where resonance effects will vary depending on the size of the irradiated object, is that one cannot argue that factors affecting an animal will have the same consequences on human subjects. Although there are several well designed studies showing increased cancer subtypes in animals exposed to cellphone radiation (e.g. Repacholi et al., 1997; Guy, Chou et al., 1985; Huang and Mold, 1980; Szmigielski, 1982; etc.) and a number of anecdotal reports, there are few peer reviewed studies relating to base station exposure levels. Those cited above, however, are still at levels (e.g. circa 100  $\mu\text{W}/\text{cm}^2$ ) that are well below those needed for thermal insult.

A major 1993 World Health Organisation review of electromagnetic fields from 300 Hz to 300 GHz (Environmental Health Criteria No. 137) lists 170 animal studies relevant to high frequency exposure, mainly at 2.45 GHz. This is somewhat higher than the 1.8 GHz frequency used in cellphone transmissions, but not significantly so. Of the studies reviewed by WHO, adverse effects were seen in approximately 130.

Typical among the earliest animal studies were those of Prausnitz and Suskind (1962), which studied the effects of chronic (19 months) microwave irradiation on mice, and those of Frey and Feld (1975), which investigated rat responses to exposure at 1.2 GHz. The former used a power density of 1 mW/cm<sup>2</sup> and a frequency of 9.2 GHz, and reported higher testicular atrophy among the 60 rats exposed vs. the 40 rats of the control group, *inter alia*. The latter reported penetration of the anaesthetised rat blood brain barrier as measured by fluorescein.

Many of the animal studies during the following decade or so used 2.45 GHz, the frequency of microwave ovens. In the 1990s, studies began to concentrate more on power densities and frequencies pertinent to cellphones, but their findings often mirrored the findings of earlier studies at different exposure characteristics. A recent study at Lund University, Sweden, for example (Salford et al., 2000), involving over a thousand rats also reported that the blood brain barrier was made more permeable as a result of exposure to RF/MW radiation (915 MHz) at levels below SAR values of 1 mW/kg. The authors concluded that *“It cannot be excluded that non-users in the vicinity of the cellular phone users may be influenced by these weak effects. Likewise, the radiation from antennas of base stations may be harmful at longer distances than hitherto suspected”*.

Arguably, the best-known of the cellphone-related studies on live animals (among eleven promotional studies reviewed in 1999 by Stewart’s expert group) was that conducted by Repacholi et al. (1997) on transgenic mice. The results showed a two-fold increase in the incidence of lymphoma in the exposed group, which was exposed to 900 MHz for 1 hour each day for 18 months at power densities equivalent to an SAR of 0.008 to 4.2 W/kg. However, none of the other promotional studies in the 1990s reported adverse effects.

The possibility that effects, where found, may be connected with resonance effects makes it difficult to extrapolate arguments from small mammal studies to human beings. The small size of such animals means that their resonant frequencies will be higher than those for humans.

## **Human studies**

These are more convincing since they report the effects of exposure under controlled conditions, where some, if not all, potential confounding factors can be ruled out. Such studies are beginning to emerge in the literature. Preece et al. (1999) and Koivisto (2000) both reported decreased reaction times during

exposure to cellphone handsets, but the studies were poorly designed with few volunteers, and were badly analysed in statistical terms.

Another area of interest has been the effect on EEG records. Approximately half a dozen studies (Reiser, 1995; Mann & Roschke, 1996; Urban et al., 1996; Krause et al., 2000, etc.) suggest that brain function is affected by cellphone radiation, though there are no equivalent data on masts.

Other recent studies have reported a lowering of blood pressure during or after exposure to cellphone frequencies (Braune et al., 1998a,b). These support the original Russian studies on RF exposure in the 1960s and 1970s (e.g. Drogihina, 1966; Sadcikova, 1974) which uncovered similar results.

## **Epidemiology**

A Swedish epidemiological study of brain tumours (Hardell, Nasman et al., 2000) has just reported that the use of cellphones significantly increases the risk of a solid tumour of the brain on the side of the head on which a cellphone is used by 2.5 times. This does not mean that exposure to base station power densities would have the same effect.

A much larger joint Norwegian-Swedish self-reporting questionnaire study of 11,000 users (Hannsen, Mild et al., 1998) found that the odds ratio of incidence of five parameters, including headaches and cheek warmth, increased with the duration of daily exposure. In Sweden, 13% of users suffered headaches (30% in Norway)

Although no peer-reviewed epidemiological study showing adverse bio-effects from base stations exists, several large studies of people living near RF/MW installations do give grounds for concern:

- a) The North Sydney, Australia, study (Hocking, 1996)
- b) The UK High power transmitters study (Dolk et al., 1997a,b)
- c) The Chinese tri-University study (Chiang, Yao et al., 1989)
- d) The Polish Military studies (Szmigielski, 1988, 1998)
- e) The Schwarzenberg transmitter study (Alpeter and Abelein, 1995)
- f) The Skrunđa radar study (Kolodynski and Kolodynska, 1996)
- g) Serdiuk and Serdiuk (1989)

Further analysis of these indicates the radiation levels that are necessary to invoke adverse health effects.

Dr Neil Cherry of Lincoln University, New Zealand, argued (in a 136 page document, July 1997) that the appropriate level for protection should be as low as  $0.1 \mu\text{W}/\text{cm}^2$  if cancer risks are to be avoided and  $0.01 \mu\text{W}/\text{cm}^2$  if children's impairment chronic fatigue syndrome and other bio-effects are to be avoided.

His conclusions were based on five principal studies reporting adverse health effects:

<i>Study</i>	<i>Subject</i>	<i>Exposure range (<math>\mu\text{W}/\text{cm}^2</math>)</i>	<i>Risk ratio range</i>
Szmigielsky, 1988/96	Polish army	<7–14	3.0–13.9
Lilienfeld et al. 1976	Moscow Embassy	<0.1–2.4	1.7–5.0
Robinette, 1980	Korean War	?	1.9–3.3
Dolk et al. 1997a,b	21 UK sites	<0.05–1.6	1.01–3.57
Hocking 1996	North Sydney	<0.04–1.6	1.61–2.74

However, he was also aware of the Chinese tri-University study (Chiang, Yao et al., 1989) and the Skrunda radiolocator studies (Kolodynski et al, 1996).

The NRPB produced a document (NRPB R321) measuring the levels of radiation near selected cellphone base stations (Mann, Cooper et al., 2000). It is a misleading document, since it argues that the 17 sites measured are typical, whereas in fact they are well down the scale in terms of power density. They were only chosen because of public concern in the localities of interest, but it is not stated that these were all the sites about which the public were showing concern, so arguably the sample is biased from the start. Furthermore, by expressing their results as a fraction of their own permitted guidelines, the NRPB are prejudicially inferring the correctness of their own recommendation.

Nevertheless, some interesting facts emerged from R321 and it also provided a beneficial and detailed non-technical overview of how mobile telephony works. None of the sites measured were microcell masts, all were macrocell masts. Whereas most macrocells emit power in the order of 20-50 W, only seven of the 17 masts measured emitted power above 10 W and only three above 20 W, so the sample was not very representative.

The maximum power density found ( $8 \text{ mW}/\text{m}^2$ ) was 60 m from a mast sited on a school building. Power density generally ranged between  $10 \mu\text{W}/\text{m}^2$  and  $1 \text{ mW}/\text{m}^2$  on all but four of the outside locations, meaning that the remaining 87 measurements were taken indoors, again leading to generally lower readings.

Where the readings exceeded  $1 \text{ mW/m}^2$ , the base station emissions dominated the total from all other origins, and other environmental signals had little effect. Finally, there is some evidence that the signal strengths did not reduce with distance.

Comparing these figures with exposures seen in epidemiological studies reporting ill health in the sample populations persuades one that exposures are already relatively high (the recommendations of NRPB and ICNIRP were not used as they are based on thermal considerations). In biophysical terms, there is a plausible explanation of how radiation in the order of  $10 \text{ } \mu\text{W/cm}^2$  (a level well below that of the cellphone in active use) can perturb common organic ions such as calcium, potassium or sodium. This may provide insight into the interaction mechanism of the device of interest, but is still not well understood.

## **CONCLUSIONS**

**The results of this study suggest that the device being tested had a significantly protective effect on lymphocyte competence and inhibited the stressor-like proliferative reaction of the cells *in vitro*.**

**Though the mechanism of interaction is not well understood, there is support for the view that MW fields of the strength emitted by cellphones are capable of perturbing organic ions, of which this device is partly constructed.**

**Further tests on live human subjects are necessary to establish whether other adverse health effects being reported by frequent cellphone users (headaches, asthenia, sleep disturbance, etc.) can be lessened during ordinary use. This test would support a claim that the device has beneficial effects on immune competence.**

## **SCIENTIFIC REFERENCES**

These are available on application. Most of those cited in the text can be found in THE INDEPENDENT EXPERT GROUP REPORT on Mobile Phones, Chairman Sir William Stewart, together with a detailed overview of the issue, published by HSMO in May 2000.