

Effects of Mobile Phone Radiation on Reproduction and Development in *Drosophila melanogaster*

David Weisbrot,¹ Hana Lin,² Lin Ye,¹ Martin Blank,³ and Reba Goodman^{1*}

¹Department of Pathology, Columbia University Health Sciences, 630 West 168 St. NYC, New York 10032

²Department of Anatomy, Columbia University Health Sciences, 630 West 168 St. NYC, New York 10032

³Department of Physiology, Columbia University Health Sciences, 630 West 168 St. NYC, New York 10032

Abstract In this report we examined the effects of a discontinuous radio frequency (RF) signal produced by a GSM multiband mobile phone (900/1,900 MHz; SAR ~ 1.4 W/kg) on *Drosophila melanogaster*, during the 10-day developmental period from egg laying through pupation. As found earlier with low frequency exposures, the non-thermal radiation from the GSM mobile phone increased numbers of offspring, elevated *hsp70* levels, increased serum response element (SRE) DNA-binding and induced the phosphorylation of the nuclear transcription factor, ELK-1. The rapid induction of *hsp70* within minutes, by a non-thermal stress, together with identified components of signal transduction pathways, provide sensitive and reliable biomarkers that could serve as the basis for realistic mobile phone safety guidelines. *J. Cell. Biochem.* 89: 48–55, 2003. © 2003 Wiley-Liss, Inc.

Key words: mobile phone radiation; *hsp70*; heat shock protein; *HSP70* promoter; DNA; electron transfer; MAPK cascades

Stress proteins (hsps) are induced by a variety of potentially harmful extracellular stimuli, such as changes in pH, heavy metals, and sudden temperature increase. The role of these proteins is to maintain the structure and function of cellular proteins. *Hsp70* is also induced in cells and tissues by non-thermal, extremely low frequency (ELF) electromagnetic (EM) fields (< 3,000 Hz) [Goodman and Henderson, 1988; Goodman et al., 1994; Goodman and Blank, 1998]. The induction of increased levels of *hsp70* is rapid (within minutes) and at extremely low levels of energy input (14 orders of magnitude lower than a thermal stimulus).

Thus, cells exhibit far greater sensitivity to ELF EM fields than to elevated temperatures.

When cells and tissues are exposed to non-thermal ELF EM fields, heat shock factor 1 (HSF1) trimerizes, and binds to a heat shock element (HSE) on the *HSP70* promoter in a region upstream from the heat shock domain [Lin et al., 1997, 1998a,b; Goodman and Blank, 2002]. The ELF EM field domain on the *HSP70* promoter contains three nCTCTn recognition motifs/response elements [Lin et al., 2001], identical to the eight elements on the *c-myc* promoter that are ELF EM field-responsive [Lin et al., 1994].

The thermal (heat shock) and non-thermal ELF EM field domains on the promoter function independently; the HSE in the heat shock domain is not interchangeable with the HSE in the ELF EM field domain. Site-specific mutagenesis in either domain eliminates the response to that stress only. Inserting nCTCTn sequences into a promoter lacking them makes the gene attached to that promoter ELF EM field-responsive [Lin et al., 2001], and the level of response appears to be related to the number of nCTCTn modules present [Lin et al., 1999, 2001].

The induction of *hsp70* by ELF EM fields also involves elements of the mitogen-activated-phospho-kinase (MAPK) family of cascades,

Abbreviations used: SRE, serum response element; ELK-1, a transcription factor in the MAPK cascade; MAPK, mitogen activated protein kinase; *HSP70*, heat shock gene; hsp, heat shock protein; HSF, heat shock factor; HSE, heat shock element; IE, immediate early; Hz, Hertz; E, electric field; B, magnetic induction field; ELF, extremely low frequency; EM, electromagnetic; RF, radio frequency.

Grant sponsor: Robert I. Goodman Fund.

*Correspondence to: Reba Goodman, Department of Pathology, Columbia University Health Sciences, 630 West 168 St, NYC, NY 10032. E-mail: rmg5@columbia.edu

Received 18 December 2002; Accepted 19 December 2002

DOI 10.1002/jcb.10480

© 2003 Wiley-Liss, Inc.

which are recognized signal transduction systems present in a wide variety of eukaryotes. MAPK pathways consist of distinct cascades of regulator enzymes that serially activate one another to control the expression of specific sets of genes in response to growth factors, cytokines, tumor promoters, and other major biological stimuli.

These pathways control cell proliferation, metabolism and cell survival in response to tissue injury, infection, malignancy, and other diseases. Also MAPK signaling pathways transduce signals for processes as diverse as mating, cell proliferation, and organ development. Depending on the cellular context, MAPK cascades may provide a switch-like, all-or-none decision between two different responses, or a graded response over a wide range of stimulus strengths. The MAPKs guide cellular maturation and can induce apoptosis. Activation of the MAPK cascades leads to phosphorylation of transcription factors that bind to the upstream regulatory elements on the promoter, relative to the transcription initiation site. There are four major MAPK cascades; the three that are differentially activated by ELF EM fields are diagrammed in Figure 1. They are the extra signal-regulated kinase/mitogen activated protein kinase (ERK/MAPK1/2), the c-Jun NH₂-terminal kinase/stress activated protein kinase (JNK/SAPK) and the p38 MAPK.

In experiments using a variety of mammalian cell lines (e.g., C3H10T1/2, HL60, HeLa, MCF7),

ELF EM fields were found to utilize some components of the p38MAPK and the JNK/SAPK cascades [Lin et al., 1998a,b; Jin et al., 2000]. This also occurs with human endothelial cells in RF fields [Leszczynski et al., 2002]. Induction appears to require the simultaneous and parallel activation of multiple kinases, the phosphorylation of more than one transcription factor and the delivery of signal(s) to the gene through several sequence elements [Downward, 2001; Brivanlou and Darnell, 2002; Hazzalin and Mahadavan, 2002; Ingolia and Murray, 2002; Weston et al., 2002].

RF field studies have similarly shown the stimulation of cell proliferation [Kwee and Raskmark, 1998] and stress proteins following exposure [dePomerai et al., 2000, 2002; Kwee et al., 2001; Shallom et al., 2002]. Thus, levels of stress proteins elevated by ELF and RF fields can serve as biomarkers for establishing biologically relevant safety guidelines for cell phone emissions.

In this article, we report the effects on *Drosophila* development of RF signals from a GSM mobile phone. We measured survival, phosphorylation of the nuclear transcription factor ELK-1, and the binding activity of the serum response element (SRE). SRE is a regulatory sequence required for the growth factor-induced, transient transcriptional activation of the cellular immediate early (IE) genes, *c-fos* and *c-jun*. These two genes, *c-fos* [Phillips et al., 1992] and *c-jun* [Rao and Henderson, 1996], and another IE gene, *c-myc*, [Jin et al., 1997] are over-expressed in response to ELF field exposures. Important in the regulation and control of development, these genes are known to transmute into oncogenes under certain conditions.

MATERIALS AND METHODS

Drosophila melanogaster

Experiments were performed with *Drosophila melanogaster* Oregon R (generously provided by Dr. Gary Struhl, Department of Genetics, Columbia University Health Sciences), maintained as previously described [Goodman et al., 1992a,b,c]. All the adults from the stock were cleared from the culture bottles. Females that emerged within 24 h were later placed into 50 ml cylindrical glass vials (2.5 cm diameter and 10 cm height), with standard food 1 cm thick at the bottom of the vials.

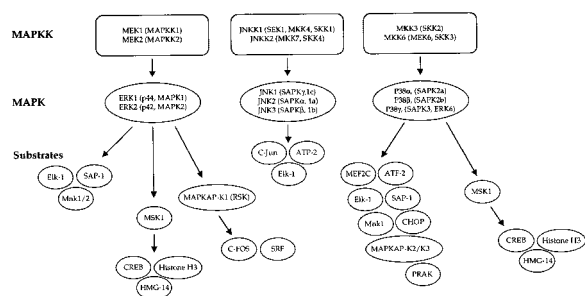


Fig. 1. Diagram of three MAPK cascades and their substrates [modified from Hazzalin and Mahadavan, 2002]. ATF, activating transcription factor; CHOP, C/EBP homologous protein; CREB, cAMP response element binding protein; MAP/ERK, mitogen-activated protein kinase/extracellular signal-regulated kinase; MAPKAP-K, MAPK activated protein kinase; MEF2C, myocyte enhancer factor 2C; Mnk, MAPK interacting protein kinase; MSK, mitogen- and stress-activated protein kinase; PRAK, p38 related/activated protein kinase; Rsk, ribosomal S6 kinase; SAP-1, SRF accessory protein-1; SRF, serum response factor.

Experimental Protocol

We used two types of matings, 1 female and 1 male per vial and also 6 females and 3 males per vial. For the parameters measured, there were no differences between the two types of mating. In each case, the procedure followed was:

- Group 1: Five vials were placed parallel to the antenna of an active GSM phone with a Tecno AO MP12 device (Fig. 2).
- Group 2: Five vials were placed parallel to the antenna of an active GSM phone (Fig. 3).
- Group 3: Five vials were placed parallel to the antenna of a phone with the power off (control).

Flies in all three groups were exposed for 60 min at 11 AM and 60 min at 4 PM daily for a total of 10 days. Pupae and adult flies were

counted daily. Protein was extracted from larvae for analyses of *hsp70* levels, binding activity of SRE and phosphorylation of ELK-1.

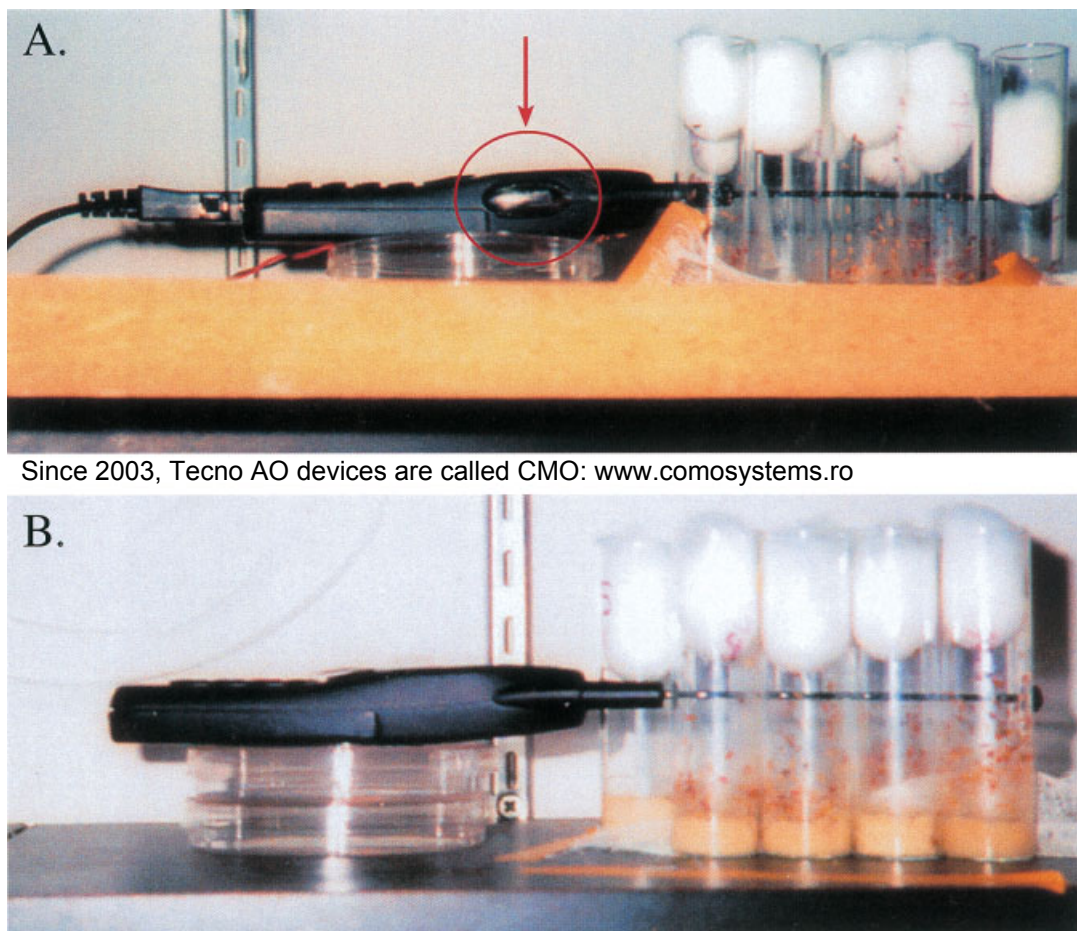
EMF Measurements

In the RF/MW range, the electric (E) field RF/MW Probe EP 330 (PMM) with wide band analyzer—0.1–3 GHz (0.3–300 V/m—conversion in A/m or mW/cm² or W/m²) was used.

In the ELF range, the electric (E) and magnetic (B) field analyzer PMM 80 + ELF Probe EHP 50 (PMM) were used, along with a spectrum analyzer (5 Hz–100 KHz, 0.1 V/m–100 kV/m/10 nT–10 mT/XYZ).

Type of Cell Phone

Bosch World 718 multiband 900/1900; Power Class: Class 4 (GSM-900/2 W); Power Class: Class 1 (GSM-1900/1 W). Frequency used in these experiments was 1,900 MHz.



Since 2003, Tecno AO devices are called CMO: www.comosystems.ro

Fig. 2. **A:** The exposure system showing five vials parallel to the antenna of an active GSM mobile phone with an attached MP12 device. **B:** The exposure system showing five vials parallel to the antenna of an active GSM mobile (900/1900). [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

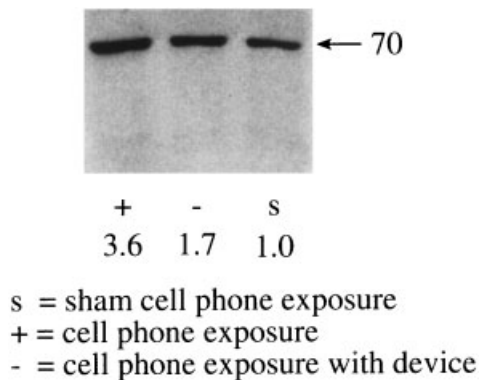


Fig. 3. Stress protein *hsp70* levels in protein isolated from larvae exposed to cell phones (+), an active phone with the device (-), and a sham (s).

Provider Used

Voice Stream activated by an automatic dialing system.

Specific Absorption Rate

Approximately 1.4 W/kg (determined by Bosch World for a human head).

Commercial Mitigation Device

Magnetic Oscillator 0.8/0.9 GHz/1.8/1.9 GHz (model MP12, TecnoZone, Montreal, QC, Canada). According to the manufacturer, the frequency range of the MP12 device is measureable by SQUID only. The main frequencies observed in ELF are 10/12 Hz and 40/45 Hz, and the intensity of the magnetic field is 100/150 Femto-Tesla.

Characterization of Cell Phone Emissions

Emissions from the cell phone were measured under four sets of conditions (measurements were made in air):

1. Mobile phone without the battery charger;
2. Mobile phone with the MP12;
3. Mobile phone with the battery charger;
4. Mobile phone with both the MP12 and the battery charger.

The measurements in the ELF and RF ranges (E field in volts/meter; B field in nanoTesla) are given in Tables I and II. From a comparison of these measurements in pairs, it is possible to determine the effect of the MP12 device with the mobile phone alone or with a battery charger attached.

TABLE I. E Field (V/m) at 1,900 MHz at Center of Antenna and at Speaker

	Switch off/on	Switch on and calling	
		Peak	RMS
Antenna	0	32.6	2.2
Antenna (with MP12)	0	21.6	1.5
Speaker	0	63.7	3.3
Speaker (with MP12)	0	56.9	2.5
Antenna (with charger)	0.4	20.4	1.7
Antenna (with MP12 + charger)	0.4	19.2	1.3
Speaker (with charger)	0.9	30.1	2.3
Speaker (with MP12 + charger)	0.9	22.2	1.9

Measurements were made in three ranges, RF/MW (1,900 MHz), ELF (0–100 Hz) and ELF (0–500 Hz), and at two locations on the mobile phone, the antenna and the speaker:

- RF/MW 1,900 MHz emissions were measured by placing the center of the probe 2.5 cm from the middle of the phone antenna (10 cm/2) and 2.5 cm from the speaker.
- ELF (0–100 Hz, 0–500 Hz) emissions were measured by placing the center of the probe 7 cm from the middle of the antenna.
- ELF (0–500 Hz) emissions were measured by placing the center of the probe 7 cm from the speaker. ELF (0–100 Hz) emissions from the speaker were not measured.

All RF/MW and ELF measurements were for 6 min, with corresponding RMS and maximum peak intensity values. Specific ELF frequency emitted by the cell phone was measured at the speaker. The analyzer showed a frequency at 35 Hz with its harmonics at 105, 175, 245, 315, 385 Hz. The 35 Hz frequency, at 0.85 V/m did not exist when the phone was off, but appeared as soon the phone was switched on, even if not connected to the provider net.

Protein Lysates

Extraction of protein was as previously described [Carmody et al., 2000]. Protein concentrations were determined by Bradford assay with a kit supplied by Bio-Rad Laboratories.

Antibodies

SRE-binding and ELK-1 phosphorylation were determined using an antibody kit (New England BioLabs). Non-phospho-ELK-1 served

TABLE II. ELF Range E Field (V/m) and B Field (nT) Measured at the Antenna

Field	Switch off		Switch on		Switch on and calling			
	E (V/m)	B (nT)	E (V/m)	B (nT)	E (V/m)	B (nT)	E (V/m)	B (nT)
0–100 Hz								
With/without MP12	2.6	33	2.8	33	2.7	116	2.7	80
Charger (with/without MP12)	500	31	500	33	500	280	500	82
0–500 Hz								
With/without MP12	2.5	77	2.8	76	2.8	91	2.8	79
Charger (with/without MP12)	500	74	500	75	500	235	500	77

as a positive control (PC). *Drosophila anti-hsp70.1* was generously provided by Dr. Susan Lindquist, University of Chicago.

Western Blot

Protein lysates were prepared and analyzed as described in Lin et al. [1997]. The intensity of the signal was determined with a Phosphor-Imager 400A (Molecular Dynamics) and quantified using ImageQuant software.

Electrophoretic Mobility Shift Assays

These were performed as described in Lin et al. [1997, 1998a,b].

Temperature

Temperature was monitored with a thermocouple probe (PhysiTemp, Hackensack, NJ) inside the vials. The sensitivity of this system is $\pm 0.01^\circ\text{C}$.

Statistical Analyses

Statistical significance was determined by the *t*-test.

RESULTS

There were significant increases in *hsp70* levels, SRE binding and ELK-1 phosphorylation in larvae exposed to emissions from cell phones. Stress protein *hsp70* levels in protein samples showed a 3.6-fold increase in *hsp70* levels as compared with the sham/inactive cell phone (Fig. 4). The addition of the MP12 device reduced the effect of the cell phone to 1.7-fold. A 3.7-fold increase was measured for SRE binding in protein samples from larvae exposed to cell phones as compared with exposure to a sham/inactive phone. The addition of the MP12 device reduced the effect of the cell phone to sham levels (Fig. 5). There was a 3.9-fold increase in phosphorylation of ELK-1 in protein samples from larvae exposed to cell phones as compared with the protein samples from larvae

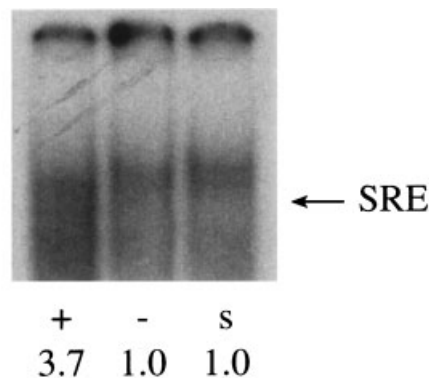
exposed to an inactive phone. The addition of the MP12 device induced a slight reduction of ELK-1 phosphorylation (Fig. 5). The positive control (pc) for phosphorylation showed a 2-fold increase over sham control(s).

Surprisingly, radiation from the GSM mobile phone induced *increased* numbers of *Drosophila* offspring per vial as compared with the inactive mobile phone group. Table III gives the numbers of adults and pupae using two types of matings, and shows the percent pupae developing into adults.

DISCUSSION

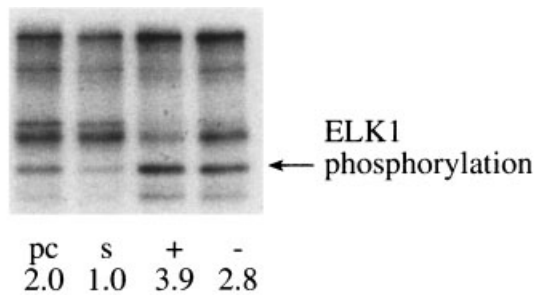
Effects of Cell Phones on Ovulation and Gene Activation

In these experiments, we have shown that cell phone radiation increases levels of the stress protein *hsp70*, the binding activity of SRE, and the phosphorylation of ELK-1. Cell phones emit both ELF and RF fields (Tables I and II).



s = sham cell phone exposure
 + = cell phone exposure
 - = cell phone exposure with device

Fig. 4. Serum response element (SRE) binding in protein isolated from larvae exposed to cell phones (+), an active phone with the device (-), and a sham (s).



p = positive control for phosphorylation
 s = sham cell phone exposure
 + = cell phone exposure
 - = cell phone exposure with device

Fig. 5. Phosphorylation of the nuclear transcription factor ELK-1 in protein samples from larvae exposed to cell phones (+), an active phone with the device (-), a sham (s), and a positive control (pc).

In earlier studies, we showed that ELF induces *hsp70*, and uses the p38MAPK and the JNK/SAPK cascades [Jin et al., 2000; Goodman and Blank, 2002]. RF field studies, using pure microwave signals, have also shown effects on cell function, specifically the stimulation of cell proliferation [Kwee and Raskmark, 1998] as well as increases in *hsp70* [dePomerai et al., 2000, 2002; Kwee et al., 2001; Shallom et al., 2002] and in fertility [dePomerai et al., 2002]. These microwave fields also utilize the p38MAPK and the JNK/SAPK cascades [Leszczynski et al., 2002]. Thus, it appears that both ELF and RF fields are capable of stimulating the same biological mechanisms and affecting the same cell functions.

The above effects on cellular function would be expected to have important influences on the organism. These are reflected in the data of Table III. Exposure to GSM mobile phones

resulted in greater numbers of adults, 22% higher in the 6:3 matings and 50% higher in the 1:1 matings. The increase in the number of adults when growth and development occur during cell phone exposure is probably the result of increased ovulation and/or increased cell divisions.

The above effects could occur at the level of the chromosomes. *Drosophila* salivary gland chromosomes, exposed to ELF fields, show increased transcriptional activity at 73 of the more than 200 transcriptionally active chromosomal regions [Goodman et al., 1992a,b,c]. On chromosome 3R, for example, these include the *hsp70* puff at 87AD, the *hsp90* puff at 93AD, and the maternal restricted transcripts at 83AD and 98DF. All four chromosomes displayed increased transcriptional activity at all loci where actin, tubulin, and myosin genes have been mapped. These three "housekeeping" genes are active during growth and development, particularly during cell divisions. Their upregulation in response to mobile phone exposure could account for the significantly increased numbers of offspring, since females were exposed before, during and after egg laying.

Biological Implications of EM Emissions on Mechanism

The physical measurements of cell phone emissions have important biological implications. In the two ELF ranges, the magnetic field (B field) is in the 200–500 nT range (Tables I and II) which is the threshold for effects on enzyme reactions [Blank and Soo, 2001], and close to the 300–400 nT threshold determined for epidemiological studies on leukemia [Ahlbom et al., 2000; Greenland et al., 2001; Milham and Ossiander, 2001]. Thus, biological effects from the ELF emissions alone are to be expected.

TABLE III. Numbers of Adults and Pupae Using Two Types of Matings

Types of matings	(n = No. of experiments)					
	6 Females: 3 males/vial			1 Female: 1 male/vial		
	n	Adults	n	Pupae	Adults	%
Inactive phone	20	81.5 ± 4.49	20	37.9 ± 4.7	34.7 ± 2.7	91.6
Active phone	19	99.45 ± 4.04 ^a	19	63.8 ± 6.8 ^a	51.9 ± 5.92 ^a	81.3
Active phone + MP12	27	94.9 ± 4.05	23	52.3 ± 5.6	40.9 ± 4.36	78.2

^aDifference between inactive phone and mobile phone exposures is significant at the 0.01 level in adults. Addition of MP12 lowered the effect of the mobile phone, but not to inactive phone/control levels.

Since 2003 Tecno AO devices has called CMO: www.comosystems.ro

Experiments with the MP12 mitigation device, suggest that RF fields from the cell phones may also play an important role in biological changes. The presence of the MP12 device had no significant effect on the intensity and composition of the spectrum and amplitude of ELF frequencies (Table I), but did cause significant changes in RF frequencies (Table II). It is clear that the biological changes in the presence of the MP12 device must be due to RF field effects.

Biological criteria for realistic cell phone safety standards

There are many reports of non-thermal biological effects from pulsed, low-intensity, microwave radiation, using both in vivo and in vitro models. In vitro studies include increases in chromosome aberrations and micronuclei in human blood lymphocytes [Garaj-Vrhovac et al., 1992], increased ornithine decarboxylase activity [Litovitz et al., 1993], single and double strand DNA breaks [Lai and Singh, 1996], increases in cell proliferation [Kwee and Raskmark, 1998], increased levels of the stress protein *hsp70* [DePomerai et al., 2000] and nonthermal activation of the *hsp27/p38MAPK* stress pathway [Leszczynski et al., 2002].

Studies using in vivo models provide additional examples, including increased permeability of blood-brain barrier in rats [Persson et al., 1997], promotion of lymphoma in transgenic mice [Repacholi et al., 1997], and pathological effects induced by embryonic and post-natal exposure to EMF radiation by cellular mobile phones [Youbicier-Simo and Bastide, 1999].

It is important to note that all but the last of these studies have used exposures from pure RF fields. The present study, therefore, adds to the demonstration that biological effects occur with an actual cell phone. The experiments reported here can be reproduced since cell phones can be readily purchased and the ELF and RF fields we utilized are well characterized.

Although biological effects are well established, the possibility of adverse health effects from exposure to the radiation of GSM cell phones is still under discussion [Goldsmith, 1995, 1997; Hyland, 1998, 2000; Foster et al., 2000; Rothman, 2000]. The biological effects suggest that some precautions are necessary, and the current safety guidelines for GSM cellular telephones specify limited radiation intensity to prevent tissue heating based on the body's thermoregulatory mechanisms. How-

ever, it is now clear that the low intensity pulsed microwave radiation currently emitted by GSM telephones exerts non-thermal influences on living organisms long before heating occurs. The pathways activated by both ELF and RF fields do not respond to changes in temperature, so they must be activated by the EM fields. Studies using ELF stimulation have shown that there are two distinct regions on the *hsp70* promoter—one responds to EM fields and a different region to thermal stimulation [Lin et al., 2001].

The rapid induction of *hsp70* within minutes, together with identified components of signal transduction pathways, provide biologically relevant, sensitive and reliable biomarkers that could serve as the basis for establishing realistic safety guidelines for mobile cell phone emissions.

ACKNOWLEDGMENTS

We thank Technolab, Research Center for providing the Bosch mobile phones, Voice Stream service, the MP12 device and made measurements presented in Tables I and II. We thank Dr. Richard Ambron, Department of Anatomy, Columbia University, for antibodies to ELK-1 and SRE. We thank Eve Vagg, Department of Photography and Illustration, Columbia University, for figure preparation and A.J Kremer for critical reading of the manuscript.

REFERENCES

- Ahlbom H, Day N, Feychting M, Roman E, Skinner J, Dockerty J, Linet M, McBride M, Michaelis J, Olsen JH, Tynes T, Verkasalo PK. 2000. A pooled analysis of magnetic fields and childhood leukemia. *Brit J Cancer* 83:692–698.
- Blank M, Soo L. 2001. Electromagnetic acceleration of electron transfer reactions. *J Cell Biochem* 81:278–283.
- Brivanlou AH, Darnell JE. 2002. Signal transduction and the control of gene expression. *Science* 295:813–818.
- Carmody S, Wu XL, Lin H, Blank M, Goodman R. 2000. Cytoprotection by electromagnetic f-field-induced *hsp70*: A model for clinical application. *J Cell Biochem* 79:453–459.
- DePomerai D, Daniells C, David H, Allan J, Duce I, Mutwaki M, Thomas D, Tattersall J, Jones D, Candido EPM. 2000. Non-thermal heat shock induced by microwaves. *Nature (Lond.)* 405:417–418.
- DePomerai D, Dawe A, Djerbib L, Allan J, Brunt G, Daniells C. 2002. Growth and maturation the nematode *Caenorhabditis elegans* following exposure to weak microwave fields. *Enzyme Microbial Technol* 30:73–79.
- Downward J. 2001. The ins and outs of signaling. *Nature* 411:759–762.
- Foster KR, Vecchia P, Repacholi MH. 2000. Science and the precautionary principle. *Science* 288:979–981.

- Garaj-Vrhovac V, Fucic A, Horvat D. 1992. The correlation between the frequency of micronuclei and specific aberrations in human lymphocytes exposed to microwave radiation in vitro. *Mutat Res* 281:181–186.
- Goldsmith JR. 1995. Epidemiological evidence of radio-frequency radiation (microwave) effects on health in military, broadcasting and occupational studies. *Int J Occup Environ Health* 1:47–57.
- Goldsmith JR. 1997. TV broadcast towers and cancer: The end of innocence for radiofrequency exposures. *Am J Ind Med* 32:689–692.
- Goodman R, Blank M. 1998. Magnetic field stress induces expression of hsp70. *Cell Stress Chaperones* 3:79–88.
- Goodman R, Blank M. 2002. Insights into electromagnetic interaction mechanisms. *J Cell Physiol* 192:16–22.
- Goodman R, Henderson A. 1988. Exposure of salivary gland cells to low-frequency electromagnetic fields alters polypeptide synthesis. *Proc Natl Acad Sci USA* 85:3928–3932.
- Goodman R, Weisbrot D, Uluc A, Henderson A. 1992a. Transcription in *Drosophila melanogaster* salivary gland cells is altered following exposure to low frequency electromagnetic fields: Analysis of chromosome 3R. *Bioelectromagnetics* 13:111–118.
- Goodman R, Weisbrot D, Uluc A, Henderson A. 1992b. Transcription in *Drosophila melanogaster* salivary gland cells is altered following exposure to low frequency electromagnetic fields: Analysis of chromosomes 3L and X. *Bioelectrochem Bioenerg* 28:311–318.
- Goodman R, Weisbrot D, Uluc A, Henderson A. 1992c. Transcription in *Drosophila melanogaster* salivary gland cells is altered following exposure to low frequency electromagnetic fields: Analysis of chromosome 3R. *Bioelectromagnetics* 13:111–118.
- Goodman R, Blank M, Lin H, Khorkova O, Soo L, Weisbrot D, Henderson A. 1994. Increased levels of hsp70 transcripts are induced when cells are exposed to low frequency electromagnetic fields. *Bioelectrochem Bioenerg* 33:115–120.
- Greenland S, Sheppard AR, Kaune WT, Poole C, Kelsh MA. 2001. A pooled analysis of magnetic fields, wire codes, and childhood leukemia. *Epidemiology* 11:624–634.
- Hazzalin CA, Mahadavan LC. 2002. MAPK-regulated transcription: A continuously variable gene switch? *Nat Rev Mol Biol* 3:30–40.
- Hyland GJ. 1998. Non-thermal bioeffects induced by low intensity microwave irradiation of living systems. *Engineering Sci Educ* 7:261–269.
- Hyland GJ. 2000. Physics and biology of mobile telephony. *Lancet* 356:1833–36.
- Ingolia NT, Murray AW. 2002. History matters. *Science* 297:248–249.
- Jin M, Lin H, Han L, Opler M, Maurer S, Blank M, Goodman R. 1997. Biological and technical variables in MYC expression in HL60 cells exposed to 60Hz electromagnetic fields. *Bioelectrochem Bioenerg* 44:111–120.
- Jin M, Blank M, Goodman R. 2000. ERK1/2 phosphorylation, induced by electromagnetic fields, diminishes during neoplastic transformation. *J Cell Biochem* 78:371–379.
- Kwee S, Rasmak P. 1998. Changes in cell proliferation due to environmental non-ionizing radiation: 2. Microwave radiation. *Bioelectrochem Bioenerg* 44:251–255.
- Kwee S, Rasmak P, Velizarov S. 2001. Changes in cellular proteins due to environmental non-ionizing radiation
1. Heatshock proteins. *Electro-Magnetobiology* 20:141–152.
- Lai H, Singh NP. 1996. Single and double strand breaks after acute exposure to radiofrequency radiation. *Int J Radiat Biol* 69:513–521.
- Leszczynski D, Joenvaara S, Reivinen J, Kuokka R. 2002. Non-thermal activation of the hsp27/p38MAPK stress pathway by mobile phone radiation in human endothelial cells: Molecular mechanism for cancer- and blood-brain barrier-related effects. *Differentiation* 70:120–129.
- Lin H, Goodman R, Henderson A. 1994. Specific region of the *c-myc* promoter is responsive to electric and magnetic fields. *J Cell Biochem* 55:1–8.
- Lin H, Opler M, Head M, Blank M, Goodman R. 1997. Electromagnetic field exposure induces rapid transitory heat shock factor activation in human cells. *J Cell Biochem* 66:482–488.
- Lin H, Han L, Blank M, Head M, Goodman R. 1998a. Magnetic field activation of protein-DNA binding. *J Cell Biochem* 70:297–303.
- Lin H, Head M, Blank M, Jin M, Goodman R. 1998b. Myc-mediated transactivation of HSP70 expression following exposure to magnetic fields. *J Cell Biochem* 69:181–188.
- Lin H, Blank M, Head M, Goodman R. 1999. Magnetic field-responsive domain in the human HSP70 promoter. *J Cell Biochem* 75:170–176.
- Lin H, Blank M, Goodman R. 2001. Regulating genes with electromagnetic response elements. *J Cell Biochem* 81:143–148.
- Litovitz TA, Krause D, Penafiel M, Elson EC, Mullins JM. 1993. The role of coherence time in the effect of microwaves on ornithine decarboxylase activity. *Bioelectromagnetics* 14:395–403.
- Milham S, Osslander EM. 2001. Historical evidence that residential electrification caused the emergence of the childhood leukemia peak. *Med Hypotheses* 56:290–295.
- Persson BRR, Salford LG, Brun A, et al. 1997. Blood brain barrier permeability in rats exposed to electromagnetic fields used in wireless communication. *Wireless Networks* 3:455–461.
- Phillips JL, Haggren W, Thomas WJ, Ishida-Jones T, Adey WR. 1992. Magnetic field induced changes in specific gene transcription. *Biochim Biophys Acta* 1132:140–144.
- Rao S, Henderson AS. 1996. Regulation of *c-fos* is affected by electromagnetic fields. *J Cell Biochem* 63:358–365.
- Repacholi MH, Baster A, GebSKI V, Noonan D, Finnie J, Harris AW. 1997. Lymphomas in E?-Pim 1 transgenic mice exposed to pulsed 900 MHz electromagnetic fields. *Radiat Res* 147:631–640.
- Rothman KJ. 2000. Epidemiological evidence of health risks of cellular telephones. *Lancet* 356:1837–1840.
- Shallom JM, DiCarlo A, Ko D, Penafiel M, Nakai A, Litovitz TA. 2002. Microwave exposure induces hsp70 and confers protection against hypoxia in chick embryos. *J Cell Biochem* 86:490–496.
- Weston CR, Lambright DG, Davis RJ. 2002. MAP Kinase signaling specificity. *Science* 296:2345–2347.
- Youbicier-Simo BJ, Bastide M. 1999. Pathological effects induced by embryonic and postnatal exposure to EMF's radiation by cellular mobile phones. *Radiat Protect* 1:218–223.