Motorola Funded Counter Research on Microwave DNA Damage

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Abstract

Dr Henry Lai and Dr Narendra Singh used a DNA Comet Assay developed by Dr Singh to determine the microwaves damaged DNA-strands. They found that non-thermal microwave exposures significantly caused single and double DNA stranded breakage in living mice brains. The cellphone company Motorola wanted to prove that these studies were wrong and that microwaves and cell phone radiation do not cause DNA strand breakage. They funded Dr Roti Roti at Washington University, St Louis to replicate the Lai and Singh studies to try to show that they do not produce these effects. Dr Roti Roti used a different, much less sensitive assessment method and used a cell-line not living mice. Hence it is not a replicate study. They claimed not to show any DNA strand breakage from radiation exposures. The analysis of their own published data shows that they actually did show that microwaves and cellphone non-thermal radiation significantly damages DNA-strands and enhances significant repair rates in human cells.

Evidence:

Lai and Singh (1995, 1996, 1997a,b) showed that microwaves caused single and double-stranded DNA breakage in living mice brains using a very advanced assay of DNA strand breakage developed by Dr N.P. Singh at the University of Washington. This is called the microgel electrophoresis or Comet Assay, Singh et al. (1994). The Comet Assay involves migration of segments of DNA down an electric field gradient.

Motorola funded Dr Joseph Roti Roti's group at Washington University, St Louis, to replicate the Lai/Singh DNA damage research and to extend it to cell phone frequencies. "Replication" requires the work to very closely follow the method and conditions of the earlier study, usually carried out by independent researchers who are well qualified. Both groups used 2.45GHz microwaves for exposure. However, the follow-up study used a cell-line (C3H/10T1/2) compared to Lai/Singh's living rats. The St Louis group also used a very different DNA damage assay based on Olive et al. (1992) not Singh et al. (1994). The follow-up study also used a much weaker fluorescent stain, an overall weaker electrophoresis field (0.6V/cm for 25mins c.f. 0.4V/cm for 60mins). Most importantly, they did not use Proteinase K to separate the bound protein from the DNA strands. It is therefore clearly evident that they used a much less sensitive method. They claim that they don’t find any DNA breakage from 2.45GHz and cellphone radiation. Despite using a different and insensitive method, their data shows that they actually did. The first example was from Malyapa et al. (1997a), Figure 5, shown in Figure 1. The sham exposure distribution is very narrow with a maximum at 32 microns. The 2hr distribution has much fewer comet tails less than 25 microns and more above 28 microns. The 2x2 analysis is presented in Table 1.

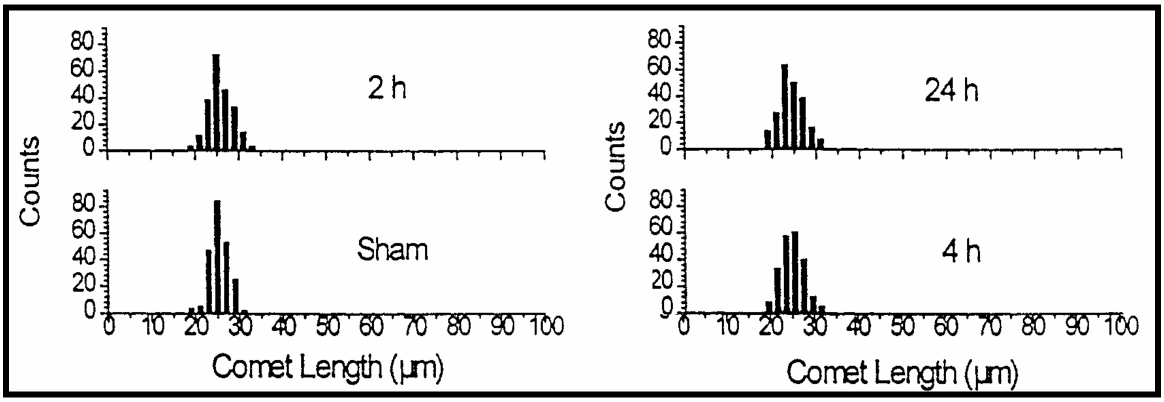

Figure 1: Frequency Distribution of Comet tail lengths for 2.45GHz exposed U87MG cells, Malyapa et al. (1997a).

Table 1: The 2x2 table of results for DNA strand breakage after exposure of U87MG cells to 2.45GHz microwaves, from Figure 1:

	Comet Length Class					
Time	≤28μm	>28μm	RR	95%CI	χ^2	p-value
Sham	196	29	1.00			
2hr	174	51	1.75	1.16 -2.76	7.34	0.0067
4 hr	206	20	0.06	0.40 -1.18	1.90	0.169
24 hr	197	25	0.87	0.53 -1.44	0.28	0.60

The time sequence of variations reveals a significant increase in DNA strand breakage after 2 hours and then the repair process kicks in and over compensates, Figure 2.

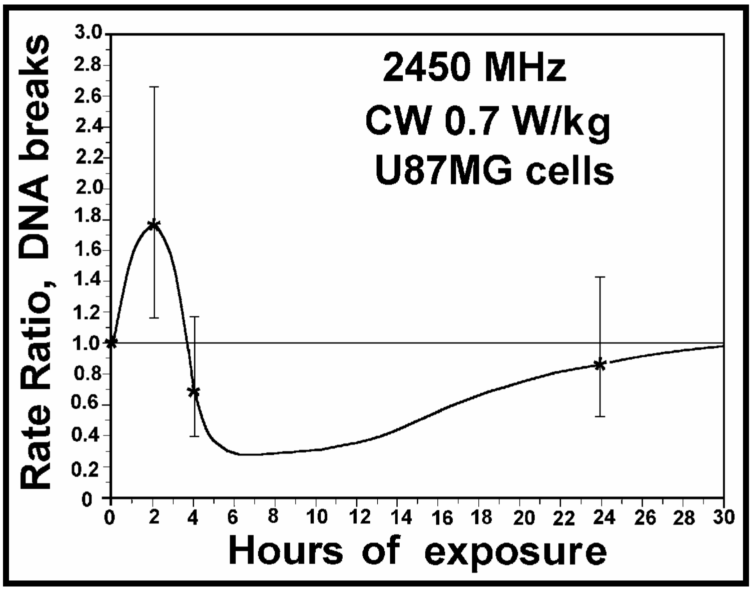

Figure 2: The time sequence of DNA damage and enhanced repair for Figure 5 in Malyapa et al. (1997).

This confirms the Lai and Singh results rather than contradicting them. This shows highly significant , p=0.0062, DNA strand breakage after 2 hours.

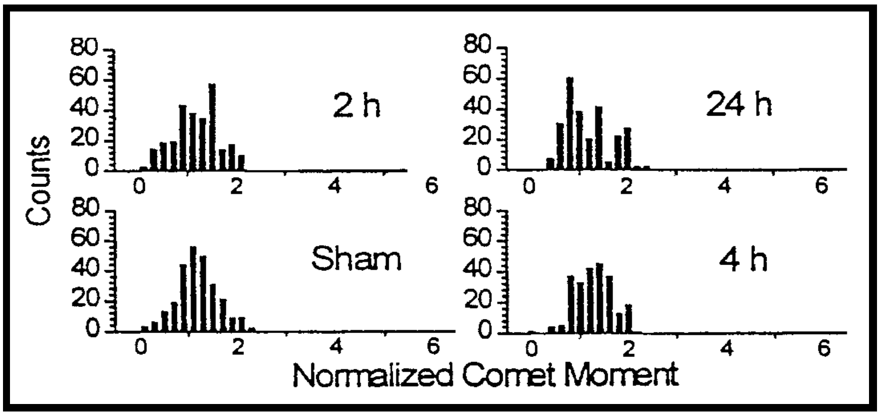

Figure 3: The frequency distribution of normalized comet moment for CW exposure of 2450 MHz at 0.7 W/kg of C3H 10T1/2 cells, Malyapa et al. (1997a) Figure 6.

Table 2: The 2x2 table of results for DNA strand breakage after exposure of C3H 10T1/2 cells to 2.45GHz microwaves, from Figure 16:

	Comet Moment Class					
Time	≤6	>6	RR	95%CI	χ^2	p-value
Sham	194	75	1.00			
2hr	176	101	1.31	1.02 -1.67	4.59	0.0321
4 hr	126	119	1.74	1.38 -2.20	23.31	0.0000014
24 hr	159	132	1.63	1.29 -2.05	18.30	0.0000189

The time sequence from Table 2 is plotted in Figure 4.

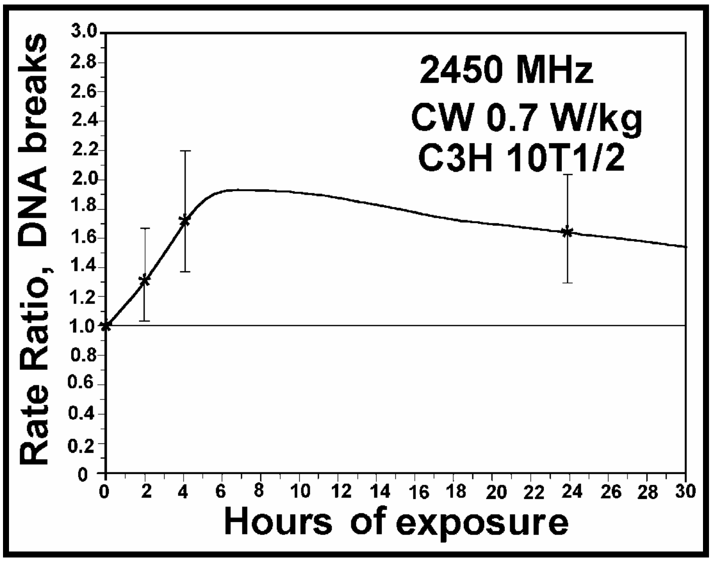

Figure 4: DNA strand breakage Risk Ratio and 95% confidence intervals for the frequency distribution of the Normalized Comet Moment of Malyapa et al. (1997a), Figure 6. The fitted time line is an estimate.

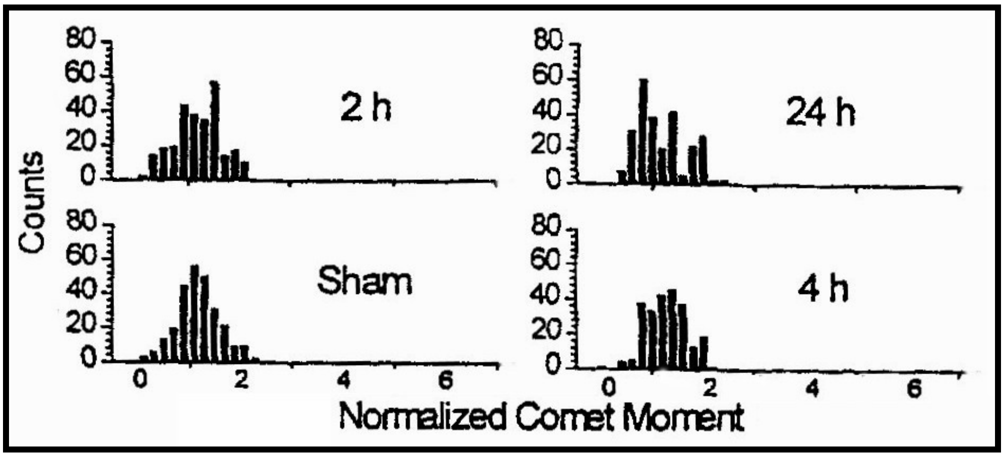

Figure 5: The frequency distribution of normalized comet moment for CW exposure of 847.72 MHz at 0.6 W/kg of U87MG cells, Malyapa et al. (1997b) Figure 2.

Table 3: The 2x2 table of results for DNA strand breakage after exposure of U87MG cells to 847.74 MHz microwaves, from Figure 18:

	Comet Moment Class					
Time	≤6	>6	RR	95%CI	χ^2	p-value
Sham	168	42	1.00			
2hr	138	92	2.00	1.46 -2.74	20.68	0.0000052
4 hr	158	50	1.20	0.84 -1.73	0.99	0.3196
24 hr	195	24	0.55	0.34 -0.87	6.72	0.00956

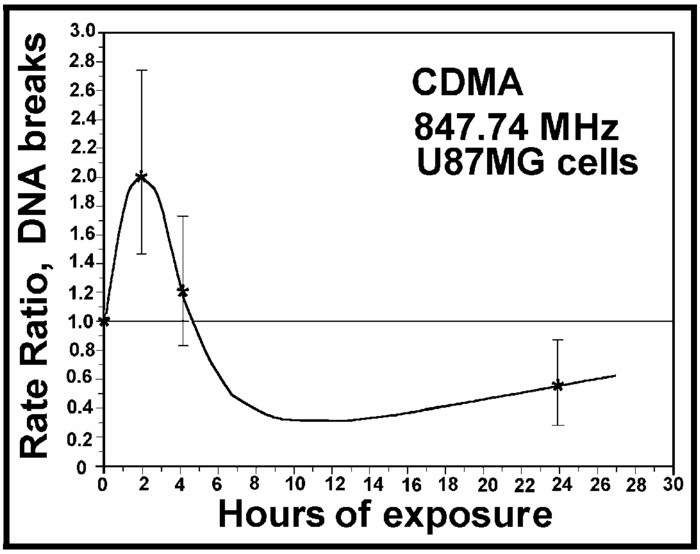

Figure 6: DNA strand breakage Risk Ratio and 95% confidence intervals for the frequency distribution Normalized Comet Moment in Figure 2 of Malyapa et al. (1997b).

Two other figures of frequency distributions in Malyapa et al. (1997a and b) were digitized and analysed using a 2x2 analysis of the Risk Ratio, Chi Squared and p-values, using a cut-level slightly above the middle of the distribution. The following time courses of DNA breakage and repairs resulted. Figure 3 shows the frequency distribution of normalized comet moment for CW exposure of 2450 MHz at 0.7 W/kg of C3H 10T1/2 cells, Malyapa et al. (1997a), Figure 6.

Table 2 and Figure 4 show significantly increased DNA strand breakage for more than 24 hours after a non-thermal microwave exposure of 0.7 W/kg of 2450MHz CW microwaves of C3H 10T1/2 cells.

The third example is derived from Figure 2 in Malyapa et al. (1997b) in which a CDMA cell phone signal, with an exposure of SAR = 0.6W/kg of U87MG cells, Figure 5.

Table 3 shows an extremely significant increase in DNA strand breakage 2 hours after the cellphone radiation exposure, $p<0.00001$. The time sequence, Figure 6, shows the same general pattern as also seen for U87MG cells exposed to 2.45 MHz radiation in Figure 2 above.

These results confirm the Lai and Singh results and confirm that microwave radiation and cell phone radiation significantly damages DNA strands and induces repair and significant repair after 4 hours in some cases. The C3H 10T1/2 cells show much slower DNA repair rates than the U87MG cells, indicating a cell-specific characteristic. It is also well known that the damage and repair rates are strongly dependent on the position in the cell cycle, Durante et al. (1994).

The results of Malyapa et al. (1997a,b) puts the results of Phillips et al. (1998) into context. Phillips et al. (1998) found highly significant ($p<0.0001$) DNA-strand breakage at 0.0024 W/kg exposure to cell phone radiation. They also found significant DNA-strand repair ($p<0.0001$) with other exposure regimes at a similar SAR level. Significant DNA-strand repair is initiated by DNA-strand breakage. This is why earlier assays were based on looking for induced DNA repair as an indicator of DNA damage, Meltz (1995).

The lower sensitivity of the assay used by Malyapa et al. is directly demonstrated by the comet tail lengths. The longest comet tail lengths were 32 microns for Malyapa et al. and 250 microns for Lai/Singh. Despite the lower sensitivity of the Malyapa et al. assay, the results confirm the initial results of Lai and Singh (1995), and Phillips et al. (1998) that pulsed microwaves, including cell phone radiation, significantly enhances DNA-strand breakage, and therefore is genotoxic and causes mutations, cancer and enhanced Apoptosis rates.

References:

Durante, M., Gialanella, G., Grossi, G.F., Mappo, M., Pugliese, M., Bettega, D., Calzolari P Chiorda, G.N., Ottolenghi, A., and Tallone-Lombardi, L., 1994: "Radiation induced chromosome aberrations in mouse 10T1/2 cells: dependence on the cell cycle state at the time of irradiation". Int J Radiat Biol 65(4): 437-447.

Lai, H. and Singh, NP., 1995: "Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells". Bioelectromagnetics 16: 207-210.

Lai, H. and Singh, NP., 1996: "Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation". Int. J. Radiation Biology 69 (4): 51 3-521.

Lai, H., and Singh, NP., 1997a: "Melatonin and N-tert-butyl-a-phenylnitrone Block 60 Hz magnetic field-induced DNA single- and double-strands Breaks in Rat Brain Cells." Journal of Pineal Research 22:152-162.

Lai, H., and Singh, NP., 1997b: "Melatonin and Spin-Trap compound Block Radiofrequency Electromagnetic Radiation-induced DNA Strands Breaks in Rat Brain Cells." Bioelectromagnetics 18:446-454.

Malyapa, R.S., Ahern, E.W., Straube, W.L., Moros, E.G., Pickard, WE. and Roti Roti, J.L., 1997a: "Measurement of DNA damage after exposure to 2450 MHz electromagnetic radiation". Radiation Research 148: 608-617.

Malyapa, R.S., Ahern, EW., Bi, C. Straube, W.L., Moros, E.G., Pickard, W.F. and Roti Roti, J.L., 1997b: "Measurement of DNA damage after exposure to electromagnetic radiation in the cellular phone communication frequency band (835.62 and 847.74 MHz)". Radiation Research 145: 618-627.

Meltz, M.L., 1995: "Biological effects versus health effects: an investigation of the genotoxicity of microwave radiation". In: Radiofrequency Radiation Standards, NATO ASI Series (B.J. Klauebberg, Grandolfo and Erwin Eds). New York, Plenum Press, 1995: 235-241.

Phillips J.L., Ivaschuk, 0., Ishida-Jones, T., Jones, RA., Campbell-Beachler, M. and Haggnen, W., 1998: "DNA damage in molt-4 T-lymphoblastoid cells exposed to cellular telephone radiofrequency fields in vitro". Bioelectrochem Bioenerg 45:103-110.