Effects of Low Level Radiation on Genetic Material

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Foreward

I have gone through various sites and read through a lot of them. I have copied and pasted information relavent information at this point. In between the itallic print, there is little side notes by me.

Site 1: Genetics and Radiation Physics;
http://www.ehs.unr.edu/rso/radprin.htm

Cells may be damaged by many factors such as life style, chemical exposure, radiation exposure, etc.,. Most cells are capable of repairing damage including damage to the genetic material if given enough time (rate of exposure). But major damage might not be repaired and may result in cell death. Ability to repair damaged cells may depend on the type of chemicals produced by radiation in the cell and/or surrounding the cell. If the chemicals produced are less active and stay away from genetic material (DNA) or other vital components necessary for cell survival, the cells would likely be less suscetible to radiation damage. The rate at which people recover from radiation exposure is not well known and variations among individuals is great.

Radiation causes ionization which that in turn causes physical and chemical effects to the atoms and cells with which it
interacts. Radiation passes through tissue and causes ionization within the cells of the tissue. The ions produced within the cell are electrically charged and chemically active. These charged, chemically active ions tend to react quickly with surrounding atoms and molecules of the cell and alter the cell structure and/or produce chemically active 6.00 radicals. For an example, water is a primary constituent of a living cell. As a result of ionizing radiation interaction, the bonds between hydrogen and oxygen may be broken. The dissociated hydrogen and oxygen from water may not recombine as water molecules but may recombine in many different combinations between oxygen, hydrogen, and electrons i.e. H2O2, HO2, OH, e-, etc. Radiation interaction can happen in any location of a cell such as the DNA or the chromosome, which if damaged, could be fatal for the cell's survival. If a large enough cell population damage occurs, then radiation effects may be immediate and fatal to the living organism. The radiation effects may show up in a matter of days as acute effects, or, years after the exposure as latent effects.

Site 2: RF Safe - This site is particularly good in showing how DNA can be damaged by the thermal and non-thermal effects of low-level radiation.

The Microwave Effect;  
http://www.rfsafe.com/microwave_effect.htm

I believe that the major contributing factor to the 'microwave effect' is actually a reciprocating lorentz-force (force exerted on a charge-carrying substance in the presence of mutually perpendicular electric and magnetic fields - such as in a microwave) exerted on the uneven charge distribution of the DNA/RNA molecule. Thus providing a non-thermal explanation for this phenomenon. If that is the case, then the frequencies involved would almost certainly be very different to the conventional 2450 MHz, since the structures and the forces involved are so different.

Can microwaves disrupt the covalent bonds of DNA? The fundamentals of thermodynamics and physics indicate this is impossible. Numerous studies have concluded that there is no evidence to support the existence of the 'Microwave Effect', and yet, some recent studies have demonstrated that microwaves are capable of breaking the covalent bonds of DNA. The exact nature of this phenomenon is not well understood, and no theory currently exists to explain it.

What's the history behind the theories of microwave effects on heating?

The dielectric effect on polar molecules has been known since
1912 (DeBye 1929). Polar molecules are those which possess an uneven charge distribution and respond to an electromagnetic field by rotating. The angular momentum developed by these molecules results in friction with neighboring molecules and converts thereby to linear momentum, the definition of heat in liquids and gases. Because the molecules are forced to rotate first, there is a slight delay between the absorption of microwave energy and the development of linear momentum, or heat. There are some minor secondary effects of microwaves, including ionic conduction, which are negligible in external heating. Microwave heating is, therefore, not identical to external heating, at least at the molecular level, and the existence of a microwave effect is not precluded simply because the macroscopic heating effects of microwaves are indistinguishable from those of external heating.

During the 1930s the effects of low frequency electromagnetic waves on biological materials were studied in depth by physicists, engineers and biologists. Studies of the effects of microwaves on bacteria, viruses and DNA were performed in the 1960s and included research on heating, biocidal effects, dielectric dispersion, mutagenic effects and induced sonic resonance. Some of the early biophysicists investigating microwave absorption claimed evidence of a 'microwave effect' which was distinct in its biocidal effects from the effects of external heating (Barnes 1977, Cope 1976, Furia 1986). Most biologists in turn claimed there was no evidence of a microwave effect and that the biocidal effects of microwaves were either due entirely to heating or were indistinguishable from external heating (Goldblith 1967, Lechowich 1969, Vela 1978, Jeng 1987, Fujikawa 1991, Welt 1994). These experiments were repeated with increased sophistication right up to the present with the majority consensus being that the microwave effect did not exist.

There were two types of experiments done to test microwave effects: controlled temperature and dry.

In the controlled temperature experiments the researchers controlled the temperature of the irradiated specimen through various timing, pulsing or cooling techniques (Welt 1994, Lechowich 1968). For example, Welt (1994) investigated the effects of microwave irradiation on Clostridium spores and found no additional lethality caused by microwaves that could not be accounted for by conventional heating. However, spores may not be representative of microwave irradiation effects on active growing bacterial cells. The results of this and other experiments showed that controlling the temperature prevented biocidal effects, and this was taken as conclusive evidence that the microwave effect did not exist. However, the assumption that the microwave effect is independent of, and separable from, temperature was always implicit in these studies, but was never
The second type of experiment, the dry experiment, also contains unacknowledged assumptions. Studies have shown that in the absence of water or moisture, biocidal effects of microwaves are severely diminished, or require considerably longer exposures (Jeng 1987, Vela 1979). This was typically taken as evidence that nonthermal microwave effects did not exist, however, since water is the primary medium by which microwaves are converted to heat, the absence of biocidal effects in the absence of water would only indicate that water is necessary for sterilization whether or not heating is the cause. Furthermore, the possibility that the specific frequency used, 2450 MHz, only affects water and not bacteria or spores was overlooked. DNA has a dielectric dispersion, where microwaves are readily absorbed, at much lower frequencies than water (Takashima 1984). The experiments may simply be indicating that the wrong frequency is being used for targeting 'dry' bacteria and spores.

Reconsidering the Microwave Effect:

Most of the studies mentioned above concluded that the microwave effect, if it existed, was indistinguishable from the effects of external heating. However, it was recently demonstrated (Kakita 1995) that the microwave effect is distinguishable from external heating by the fact that it is capable of extensively fragmenting viral DNA, something that heating to the same temperature did not accomplish. This experiment consisted of irradiating a bacteriophage PL-1 culture at 2450 MHz and comparing this with a separate culture heated to the same temperature. The DNA was mostly destroyed, a result that does not occur from heating alone. These photos are borrowed from Kakita et al (1995), permission pending. In the Kakita experiment the survival percentage was approximately the same whether the samples were heated or irradiated with microwaves, but evaluation by electrophoresis and electron microscopy showed that the DNA of the microwaved samples had mostly disappeared. In spite of the evolving complexity of all the previous experiments, electrophoresis had not been used to compare irradiated and externally heated samples prior to this. Electron microscopy had been used to study the bacteriocidal effects of microwaves (Rosaspina 1993, 1994) and these results also showed that microwaves had effects that were distinguishable from those of external heating.

The energy level of a microwave photon is only 10-5 eV, whereas the energy required to break a covalent bond is 10 eV, or a million times greater. Based on this fact, it has been stated in the literature that "microwaves are incapable of breaking the
covalent bonds of DNA" (Fujikawa 1992, Jeng 1987), but this has apparently occurred in the Kakita experiment, even though this may be only an indirect effect of the microwaves. There is, in fact, plenty of evidence to indicate that there are alternate mechanisms for causing DNA covalent bond breakage without invoking the energy levels of ionizing radiation (Watanabe 1985, 1989, Ishibashi 1982, Kakita 1995, Kashige 1995, Kashige 1990, 1994). Still, no theory currently exists to explain the phenomenon of DNA fragmentation by microwaves although research is ongoing which may elucidate the mechanism (Watanabe 1996).

The results of microwave irradiation affected two bacteria, S. aureus and E. coli. The death curves exhibited classic exponential decay with an apparent shoulder, as well as a possible second stage. These curves are based on data from Kakita et al (1999).

The microwave frequency used in the Kakita study was the standard 2450 MHz used in conventional microwave ovens. This is the same frequency that was used in essentially all prior studies, except for the earliest studies (which looked at lower frequencies), and sonic resonant studies, which looked at much higher frequencies. The early studies showed that DNA tended to absorb microwave radiation "in the kilocycle range" (Takashima 1963, 1966, Grant 1978, Grandolfo 1983), but no biocidal effects in the range of 1 MHz to 60 MHz were observed. One notable exception, however, was an early experiment which found that frequencies between 11 and 350 MHz had lethal effects on bacteria, with a peak at 60 MHz (Fleming 1944). As far as could be determined, the contradiction between the results of Fleming and those of Takashima has never been resolved or re-addressed. In any event, there is no evidence in these studies to indicate any undue attention was paid to control the actual absorbed dose or the precise geometry of the irradiation cell, and therefore the differences in the results of these investigators may reflect differences in their cell geometries, among other things.

In summary, it would seem there is reason to believe that the microwave effect does indeed exist, even if it cannot yet be adequately explained. What we know at present is somewhat limited, but there may be enough information already available to form a viable hypothesis. The possibility that electromagnetic radiation in the non-ionizing frequency range can cause genetic damage may have profound implications on the current controversy involving EM antennae, power lines, and cell phones.

A Theory of Microwave Induced DNA Covalent Bond Breakage

A review of the data from the various referenced experiments shows a common pattern -- for the first few minutes of irradiation there is no pronounced effect, and then a cascade of microbial destruction occurs. The data pattern greatly resembles the dynamics of a capacitor; first there is an accumulation of energy,
and then a catastrophic release. It may simply indicate a threshold temperature has been reached, or it may indicate a two-stage process is at work.

A link that may be of interest, but not as directly relevant to my topic:

**DNA single and double strand breaks at levels below the current FCC exposure standard**

So at this point a few days ago, I thought to myself of the researcher who believes that this Microwave Effect that could be the cause of a lot of genetic damage is actually due to the Lorentz Force. That directly hits the topic of Electromagnetism and it explains why damage could be caused by Microwave Radiation even though the energies are not literally enough to break the covalent bonds in DNA. This lead me to do more research on the Lorentz Effect and its relation to the Microwave Effect...

**Site 3: (Really Detailed Stuff);**
http://www.elettra2000.it/scienza/EBC.htm

Although at the cellular level, this is some more supporting evidence:

*The previous discussion deals mainly with mechanisms plausible for explaining bioelectromagnetic effects in the low frequency range.*

*Some authors have addressed the specific possibility that the cell membrane lipids are the target of the electromagnetic field interaction at high frequencies. In general, a cell membrane phospholipid is composed of two parts: one head and two tails with similar chemical and physical composition. It has been recently shown (Seelig et al., 1987) that in some phospholipids the polar heads align themselves nearly parallel (within 30°) to the membrane surface and that the heads' group orientation is identical in artificial bilayers and in biological membranes. Different conformations of a phospholipidic bilayer are possible. The transition between one conformation to the other is affected by the concentration of bound water at lipid-water interface, and therefore it can be induced isothermally by ionic or solute interactions (Cevc and Marsh, 1987), while, at fixed bound water concentration, it can be induced increasing the temperature over a critical phase-transition value, Tt. The presence of charges or dipolar molecules external to the membrane but in the proximity of the heads can modify the bistable nature of the phospholipidic bilayer (Seelig et al. 1987; Cevc and Marsh, 1987; Scherer and Seelig, 1987; Shepherd*
and Buldt, 1978).  These recent experimental results can give support to the Bond and Wyeth (1986, 1987) hypothesis that justifies the results of the Liburdy and Penn experiments (1984, 1986). In these experiments, passive permeability of sodium ions in red blood cells and in lymphocytes increased under microwave exposure (2.45 GHz, continuous wave (CW), 60 mW/g) in correspondence to $T_t$. A phase transition could be involved in this microwave effect, leading to the hypothesis that the phase transition temperature $T_t$ is near to a natural critical temperature $T_c$ at which the system becomes extremely sensitive to small external perturbations.

The Lorentz Effect and it's Part (still at the cellular level):

The historically initial surge of interest on biological microwave effects and related mechanism has been followed by studies at lower frequencies, as they appear to be more fruitful and interesting because the experimental results are quite available. Interaction with ions has been proposed as a possible explanation of em field induced flux of calcium observed "in vivo" and in vitro in different excitable cells, as already discussed in previous sections, (see the review made by Adey, 1984). These results include calcium-ion influx or efflux from the cerebral cortex of cats "in vivo" (Kaczmarek and Adey, 1974) and from isolated chick cerebral hemisphere (Bawin et al., 1975) produced by modulated VHF. Similar subsequent results in the ELF-range (Bawin and Adey, 1976, 1977) or with ELF modulated microwaves (Adey et al., 1982) have been confirmed and partially expanded by other authors (Blackman et al., 1979, 1985; Athey, 1981; Merrit et al., 1982; Dutta et al., 1984). In particular, Blackman et al. (1985) showed that em-field induced alterations of ion fluxes strongly depend on the value of the static magnetic field; so that two experiments using the same em-field (amplitude and frequency) may give rise to distinct results if the environmental static magnetic field is different. A mechanism that can explain Blackman's results was proposed by Chiabrera et al. (1985b-1987) by analysing the em field local effects on proteins and ions that interact with each other on the surface of the cell. They considered the action of electric and magnetic forces on different ionic messengers and binding sites. The contribution of the magnetic induction has been added to an earlier model (Chiabrera et al., 1984a), according to a suggestion of Liboff (1985). The binding process is clearly a statistical event: the binding site moves on the cell surface, while the external messenger (ligand) moves in the microenvironment outside the cell. An applied em field modifies the velocity and the path of the external messenger near the binding site and will therefore change the association and dissociation rate constants of the receptor-ligand binding action, altering ion flux through the membrane.
The problem of a messenger ion (mass $m$ and charge $q$) which travels near a binding site or across a membrane channel has been approached from the classical point of view. In this case, the variable of interest can be the square of the time average ion displacement $r$ or the square of the time average of the ion velocity. The equation that gives the ion dynamics is obtained by introducing the Lorentz's force in the classical Langevin equation (Langevin-Lorentz model):

where: $b$ is the Langevin "collision frequency", which models the average energy loss of the ion, due to its interaction with the thermal bath (e.g. interactions with water molecules); $w_{2end}$ models the endogenous force of the binding site $F_{end} = -mw_{2end} r$ as a linear harmonic oscillator; $g = q/m$ is the ion charge to mass ratio; $n$ is a white noise random force divided by $m$, whose average value is zero, which models the random fluctuation induced by the thermal bath. Its spectral density, for each degree of $0.00\text{dom}$ of the ion is $2kT b/m$, being $k$ the Boltzmann's constant and $T$ the absolute temperature. $B = BO + B_{1\text{sen}} w t$ is the magnetic induction.

The aforesaid equation has been studied under various simplifying assumptions and using numerical approaches by many authors (see papers in Chiabrera et al., 1985a; Blank and Findl, 1987; Durney et al., 1988 and D'Inzeo et al., 1992), mainly in the case of the Helmoltz coil exposure system and in the case of a TEM exposure Cell (Crawford cell).

The ion displacement and velocity exhibit characteristic resonances for values of $w$ close to multiple values of the dynamic cyclotron frequency $B_{1/2}(p))$ (twice the corresponding Larmor frequency) associated to the intensity of the time-varying component of the magnetic induction. These resonances occur for $BO = 0$ or for values the cyclotron frequency $(gBO/(2 A \text{ simplified approach has been proposed by Liboff and McLeod (1985, 1986, 1988). They hypothesised, as a particular case of eq. (8), a frequency selective effect based only on the dc cyclotron resonance frequency of the ions. The ionic movements could be guided by helicoidal pathways created by the endogenous field present in the proteins' structure of channel walls, or by the quasi-cylindrical structure of a channel screens the ions from the thermal noise present on both sides of the membrane. Then, the ions could be accelerated by the field and cross the membrane channel.}

Halle (1988), referring to the Liboff and McLeod theory, criticised their proposed interaction mechanism. If the ionic trajectory is prescribed, the magnetic effect vanishes identically. Furthermore, Halle reiterates that, even if the motional constrains are "relaxed", dynamic friction ensures that the magnetic effect is utterly insignificant, if a viscosity value corresponding to bulk water is considered. Chiabrera et al. (1989) have proved that if the endogenous field is very large,
and changes rapidly in space, water molecules are expelled from the site, and the ion experiences very low effective viscosity. Nevertheless other unresolved problems, related to the endogenous force modelling, remains (Chiabrera et al., 1992), because of the nonlinearities involved. Furthermore the values of ion displacement and velocity due to the exogenous exposure alone (i.e. neglecting noise) are much smaller than their expectation values due to thermal noise alone (i.e. em exposure off). Then, time-averaging seems not to be sufficient to overcome thermal noise. We conclude that this classical approach seems not yet able to offer a physically plausible model that explains the experiments (Bianco and Chiabrera, 1992). The weakest point of the classical model so far discussed is the oversimplified expression of \( F_{\text{end}} \), which is a very large field near the binding site, dropping to zero a few angstroms or nanometers away from the site. Its values obtained from the Protein Data Banks point toward large non linear effects. Thus, a more rigorous quantum approach is needed.

Conclusions and Further research:

The basic idea behind me going through these research sites is to narrow down physically how genetic material could possibly be damaged by EM waves. I narrowed down my choice to analyzing the growing support for the Lorentz Effect as a cause for the Microwave Effect.

Further research should entail mainly getting specific statistics and facts of EM-related genetic defects so the reader will get an idea of how prevalent this could be.

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