It is also recognized that a person's ability to dissipate absorbed RF/MW energy will also depend on their physical activity, the ambient temperature, exposure to bright sunlight and health status. Hence a further safety factor of 5 , i.e. $0.08 \mathrm{~W} / \mathrm{kg}$ is used to avoid RF 'hot spots' (Repacholi (1993) p 185 and p 189) and avoid heating of the chronically ill, frail people on very hot, sunny humid days, (p 177) Repacholi (1993).

Jammet (1984), reporting the IRPA exposure guidelines remarks

> "The basic limit above 10 MHz ( $0.4 \mathrm{~W} / \mathrm{kg}$ for occupational exposure or 0.08 W/kg for the general public) protects against potential thermal hazards."

Hence it is made clear by both the WHO technical group (Repacholi (1993)) and the IRPA that the $0.08 \mathrm{~W} / \mathrm{kg}$ standard is a thermal protection standard and makes no attempt to protect from epidemiologically identified adverse health effects from chronic exposure to non-thermal levels of exposure to RF/MW radiation.

Michaelson (1971) also summarizes the animal experiments which were used to establish and validate the $10 \mathrm{~mW} / \mathrm{cm}^{2}$ "safe" exposure standard, which is openly and clearly related to the avoidance of heating effects.

A further indication of the thermal basis of the standard is the inverse " $U$ " shape of the allowable exposure as it changes with frequency, following the inverse of the whole body resonant absorption curve.

### 5.4 Australian/ New Zealand Standard:

The EMR standards committee is a committee of Standards Australia and it is a joint committee with New Zealand. Over the post war years EMR standards in the West have been the interest of the military and industry. The Australian and New Zealand RF/MW standards sub-committee reflects this. Its membership is described as "stakeholders". This is not universally accepted as the right way to go. People with a financial "stake" should not be involved in setting public health standards.

The U.K. air quality standards committee consists of 5 independent scientific experts with high standing in the community. They are not allowed to have any direct nor indirect financial involvement with industry. They are required to consult widely with industry, the community and with experts, and then to make recommendations based on epidemiology and toxicology, on public exposure levels which will not produce a detectable rise in public health risk.

In the U.K. the Government and the Public can have confidence that public health is being protected by these standards. They are reviewed regularly and have progressed downwards, with lower and lower allowable exposure levels to pollutants as epidemiological studies show adverse health effects at lower and lower levels of exposure. Contrast this with the membership and brief of the committee which originally set our radiofrequency public exposure guideline.

AS 2772 was set in 1990 by Committee TE/7 of the Australian Standards Association, comprising:

Australian Electronics Industry Association
Australian Radiation Protection Association
Civil Aviation Authority

Department of Defence<br>Department of Industrial Relations and Employment, NSW<br>Department of Transport and Communications<br>Institution of Radio and Electronics Engineers Australia<br>National Measurements Laboratory CSIRO<br>National Occupational Health and Safety Commission<br>Royal Adelaide Hospital<br>Telecom Australia<br>Wireless Institute of Australia

The membership illustrates the technical nature of the committee. One of the more benign looking members, Royal Adelaide Hospital, is represented by Dr Michael Repacholi, the then Chairman of the Committee, and a frequent consultant paid by industry. The committee has been expanded to include New Zealand representatives, including Telecom NZ (Mr Simon Cooke-Willis), NorthHealth (Dr Black), BCL Ltd, National Radiation Laboratory ( Dr Andrew McEwan), Ministry of Commerce, Local Body Association and a Public Representative (Prof Ivan Beale). Both Dr Black and Dr McEwan have appeared as paid consultants to industry.

The International Radiation Protection Association (IRPA) and the Australia/ New Zealand Standard are acknowledged to be thermally based, thermally derived and thermally expressed. They are based on the Specific Absorption Rate (SAR) of radiation by human bodies and the body's ability to dissipate heat and thus retain homeostasis (a constant Core Temperature of about $37^{\circ} \mathrm{C}$ ).

At the Waituna II workshop in Auckland, February 1998, and at a seminar sponsored by Telecom NZ on Monday 29th January 1998, Dr Miala Hietenan, Senior Lecturer in Nonlonizing Radiation from Finland, and a member of the International Commission on Nonlonizing Radiation Protection (ICNIRP) confirmed that the ICNIRP standard was based on "established" health effects and that the only established health effect of RF/MW was tissue heating. When asked if the standard was based in physics she said "yes", and when asked why it did not relate to physiology, biochemistry or epidemiology she stated that there was only weak evidence which did not establish health effects.

Hence, it is clearly shown and admitted by those involved that the international and national standards are based on established effects only, and the only such effect of RF/MW is heating. As such they have no standing in New Zealand where actual and potential effects must be taken into account.

### 5.5 Mind changing case study:

## Once established, this thermally-based mind-set is very hard to change.

The strength of the thermally-based position is very strongly held and very hard to change. Dr Michael Repacholi provides a good case study.

In November 1995 Dr Repacholi gave evidence in the MacIntyre Case in Christchurch to the effect that there are only thermal adverse effects and epidemiological studies, such as the Korean War Study showed no effects from radio and radar exposure of exposed service men.

A primary method used to dismiss adverse effects, the strong proof approach, is to require increasingly higher levels of proof, dismiss projects one at a time as insignificant, and to continue to recommend that more and more research is necessary to clarify uncertainties. In the light of the recommendations in the WHO review team's report which he edited (and was chair of the task group) that "research efforts be coordinated to clarify rather than increase the level of uncertainty" and the careful design and execution of the project, Dr Repacholi and his research team were at pains to play down the implications of the results that cancer prone mice exposed to cell phone signals for 1 hour a day were observed to double their cancer rate.

Mice were chosen because evidence from mice is indicative of probable effects in people. To claim that the effects can be ignored because the happen in mice is not consistent nor credible.

This is definitely an athermal, carcinogenic result of RF/MW exposure.
Several other mouse or rat studies carried out in the U.S. have found increases of cancer incidence in mice or rats exposed to radar signals. It was previously claimed that these other projects could not be applied to cell phones and so until an experiment was done with an actual cell phone signal we shouldn't worry about potential health effects. Now that this experiment has been carried out, and it was found that mobile phones double the lymphomas in B-cells of the immune system of these mice, there is little reason to assume that it couldn't happen in the B-cells of the human immune system. However, Dr Repacholi still claims there is no evidence to link cell phones to cancer in humans. This ignores seven epidemiological studies which show increases in brain tumours associated with RF/MW exposure. Dr Repacholi stated in a TV link from Geneva to Australia:
> "This is the first scientific study to show such an effect and as such in science it is necessary before you can really use this result for any health risk assessment, to repeat the study and extend it. By extending it I mean to look at other mouse models. Also to expose the mouse model to different levels of radiofrequency field so that we find out whether, first of all, this result was only a characteristic of these genetically engineered mice or whether it has wider implications for health. So we have a lot of research to do before the result can be used in the debate as to whether electromagnetic fields have an influence on cancer."

Hence, while the study was specifically and carefully designed to answer a particular question as to whether there were any health risks from cell phones, according to Dr Repacholi, this study has not answered the question nor has it decreased uncertainty at all.

The timing of the release of the results poses some serious questions about expert advice in court when the expert involved has a commercial contract requiring silence until 3 months after the contractor is told of the results and the results are published.

The Adelaide experiment is a very important result, as Dr Repacholi now acknowledges, showing an athermal effect. "I believe that this is the first study showing a true nonthermal effect", Dr Repacholi told Microwave News, May/June 1997.

The paper was received by Radiation Research on 8 July 1996. The paper was submitted to at least two other journals who declined to publish it, a process which could have taken at least six months. The writing up of the paper after the completion of the experiments would also take at least two or three months. Hence it is highly likely that final results were known by Dr Repacholi when he testified under oath in the Macintyre case, and at least the 15 month results would have been known.

On 21-22 November 1996 the WHO held a workshop on athermal effects in Munich (organized by Dr Repacholi). Dr Repacholi's mouse experiment results could not be presented here either. In fact Dr Repacholi circulated pre-prepared conclusions that evidence for nonthermal effects was weak at best. Papers presented identified the following athermal cellular responses to EMR exposure.

- release of calcium ions from chick brains, frog hearts, cat brains, rat brains and human cells.
- altered brain activity
- inhibition of T-lymphocyte cyclotoxicity
- decrease in non-cyclic AMP dependent kinase activity in lymphocytes
- transient increases in ornithine decarboxylase in various cell lines
- cell cycle alteration
- cell proliferation
- neoplastic formation
- biomolecular/biochemical changes
- membrane ion transport binding changes and altered single channel kinetics

Animal experiments involving athermal exposure reported showing effects including:

- Altered behaviour in cats, $147 \mathrm{MHz}, 0.001 \mathrm{~W} / \mathrm{kg}, \mathrm{AM}$ at $1-25 \mathrm{~Hz}$, Bawin and Adey.
- Structural rearrangement of DNA in mice exposed to 1 W/kg for 120-200 days, Sarker (1994).
- Single-strand DNA breakage, 0.6 and 1.2 W/kg for 2 hr , Lai and Singh (1994).
- Double-strand DNA breakage, 1.2 W/kg for 2 hr , Lai and Singh (1996).
- Increase in lung metastases in rats exposed to 2.45 GHz , Szmigielski (1982).
- Altered EEG rhythm in rabbits exposed to $1-10 \mathrm{MHz}, 0.001 \mathrm{~W} / \mathrm{kg}$ modulated at $14-16$ Hz . Takashima.
- Ocular damage in primates exposed to continuous and pulsed radiation 2.66-7.8 W/kg, Kues.

As reported in the E.P.A. review, the Cleary review and the WHO 1981 and 1993 reviews, this workshop confirms that there is a growing body of scientific research which shows that very low, non-thermal levels of RF/MW radiation alters the basic biochemistry of cells which have a potential to cause altered brain function, carcinogenesis and impaired immune system functioning. Animal experiments show that these effects occur in living animals, including DNA breakage, cancer at many body sites, behavioural alteration, EEG change and ocular damage. Many epidemiological studies show increased risks of cancer and many other health effects with increase RF/MW exposure.

## 6. Biophysics and Geophysics factors:

In order to understand the way in which EMR can influence people it is helpful to be aware of the physical absorption factors, biochemical changes at the cell level and how these have been shown to change with EMR exposure.

### 6.1 Physical Variables, Units and Formulas Used:

At any point in the EM wave the electric field ( E in $\mathrm{V} / \mathrm{m}$ ) and the magnetic field ( $H$ in $\mathrm{A} / \mathrm{m}$ ) are proportional to each other, such that $E / H=120 \pi=377 \Omega$, the free space impedance.

The flux of E-M energy which is radiated through space is called the Energy Flux (S), and when it impacts onto an object it is called the Exposure. Exposure relates to the electric field through:

$$
\begin{array}{ll}
S=E^{2} / 377 & {\left[\mathrm{~W} / \mathrm{m}^{2}\right]} \\
\mathrm{S}=377 \mathrm{H}^{2} & {\left[\mathrm{~W} / \mathrm{m}^{2}\right]} \tag{2}
\end{array}
$$

where $H$ is the magnetic field strength in Ampere/m ( $=\mathrm{A} / \mathrm{m}$ ) which is related to the Magnetic Flux Density $(B)$ through:

$$
\begin{equation*}
\mathrm{B}=\mu \mathrm{H} \quad \text { [Tesla] } \tag{3}
\end{equation*}
$$

where $\mu$ is the magnetic permeability, $\mu \approx \mu_{0}=1.257 \times 10^{-6} \mathrm{H} / \mathrm{m}$ ( $\mathrm{H}=$ Henry).
Exposure is frequently expressed in $\mu \mathrm{W} / \mathrm{cm}^{2}$.

$$
\begin{equation*}
1 \mathrm{~W} / \mathrm{m}^{2}=100 \mu \mathrm{~W} / \mathrm{cm}^{2} \tag{4}
\end{equation*}
$$

As the EM field propagates away from its radiating source (in the far field) the Energy Flux decreases as the square of the radius (r) and:

$$
\begin{equation*}
S=P / 4 \pi r^{2} \tag{5}
\end{equation*}
$$

where $P$ is the total radiated power,
and $r$ is the distance from the antenna.
Hence the Exposure of people and objects gets rapidly smaller as distance increases from a transmission facility, such as a radar, a cell site or a TV tower. For each doubling
of the distance the Exposure is reduced by one quarter. However, since $S$ varies as the square of electric field and magnetic field, E and H reduce linearly with distance from the antenna in the far field condition.

For an isotropic antenna, radiating equally in all directions, the exposure at any distance from the antenna can be calculated from Eq (5). Almost all commercial antennae have considerable directional focus to send their radiation more intensely in chosen directions so that TV, radio and cell site stations direct their signals towards potential receivers and not out to space. Hence for a given antenna power ( P ), the exposure at a given radius will be greater than that given by Eq. (5) by factor related to the Antenna Gain (G). The radiation pattern is further complicated by the existence of side-lobes in addition to the primary beam. Hence close to an antenna the exposure pattern is complicated. In the more distant field, outside the influence of the side lobes, the intensity decreases as the inverse square law of Eq.(5), but with a higher initial power determined by the antenna gain characteristics modified by the loss characteristic of the feeds etc. An overall gain factor of 6 to 7 is common for omni-directional antennae on Telecom cell sites.

The Energy Flux which impacts on a object is reflected, scattered and absorbed by the object. The proportion of the energy absorbed is a function of the wavelength of the wave compared to the linear dimension of the object. The most efficient energy absorption occurs when the wavelength of the EM wave is close to twice the size of the object. Larger objects have more efficient energy absorption at longer wavelengths, i.e. at lower frequencies, while smaller objects have higher absorption efficiencies at shorter wavelengths and higher frequencies. A 1.8 m man has a peak absorption rate at about 70 MHz , a monkey at about 300 MHz , an adult head at about 915 MHz and a mouse at about $2,450 \mathrm{MHz}$.

On a gross scale the energy absorption is expressed according to the incident absorbed energy's heating ability and it is expressed as a Specific Absorption Rate (SAR), which in terms of the incident electric field is (Gandhi (1990))

$$
\begin{equation*}
\mathrm{SAR}=\sigma \mathrm{E}^{2} / 2 \rho \quad[\mathrm{~W} / \mathrm{kg}] \tag{6}
\end{equation*}
$$

where $\sigma$ is the electrical conductivity of the tissue, in Siemens $/ \mathrm{m}^{2}$ or $\mathrm{S} / \mathrm{m}^{2}$, and $\rho$ is the density of the tissue, in $\mathrm{kg} / \mathrm{m}^{3}$.

| Table 5: Mid-range values of electrical conductivity ( $\sigma$ ) for biological tissue at $37^{\circ} \mathrm{C}$ |  |  |  |  |  |  |
| :--- | :--- | :--- | :--- | :--- | :--- | :--- |
| as a function of RF/MW frequency, in $\mathrm{S} / \mathrm{m}$, Stuchly and Stuchly (1990) |  |  |  |  |  |  |
| Tissue | 100 kHz | 1 MHz | 10 MHz | 100 MHz | 1 GHz | 10 GHz |
| Skeletal Muscle | 0.50 | 0.72 | 0.83 | 0.90 | 1.42 | 11.5 |
| Liver | 0.16 | 0.28 | 0.45 | 0.66 | 0.98 | 8.9 |
| Spleen | 0.62 | 0.63 | 0.67 | 0.89 | 1.2 | 10.1 |
| Kidney | 0.25 | 0.37 | 0.59 | 0.86 | 0.98 | 9.7 |
| Brain | 0.15 | 0.18 | 0.42 | 0.72 | 1.00 | 9.1 |
| Bone | 0.014 | 0.017 | 0.024 | 0.057 |  |  |

At ELF frequencies electrical conductivities are $0.1-0.35 \mathrm{~S} / \mathrm{m}$ for cardiac muscle, $0.1-0.3$ $\mathrm{S} / \mathrm{m}$ for nerve tissue, $0.2 \mathrm{~S} / \mathrm{m}$ for cerebral tissue and $0.25 \mathrm{~S} / \mathrm{m}$ for myocardial tissue, Repacholi (1993). Typical values for the electrical conductivity of tissue for MW are 0.05
$\mathrm{S} / \mathrm{m}$ for bone, $0.95 \mathrm{~S} / \mathrm{m}$ for muscle and $0.77 \mathrm{~S} / \mathrm{m}$ for visceral organs such as heart, liver, brain etc.

Density is close to $1000 \mathrm{~kg} / \mathrm{m}^{3}$ for most tissue because of the presence of water. However lung tissue is about $100 \mathrm{~kg} / \mathrm{m}^{3}$ since it contains pockets of air.

While in terms of the tissue heating rate it is:

$$
\begin{equation*}
S A R=C D T / d t \tag{7}
\end{equation*}
$$

where C is the specific heat of the tissue, in $\mathrm{J} / \mathrm{kg}-{ }^{\circ} \mathrm{C}$, and $\mathrm{DT} / \mathrm{dt}$ is the rate of increase in tissue temperature $\left({ }^{\circ} \mathrm{C} / \mathrm{s}\right)$

Combining Eq. (1) and Eq. (6),

$$
\begin{equation*}
S=(2 \rho / 3.77 \sigma) \text { SAR } \quad\left[\mu W / \mathrm{cm}^{2}\right] \tag{8}
\end{equation*}
$$

and using $\rho=1000 \mathrm{~kg} / \mathrm{m}^{3}$

$$
\begin{equation*}
S=530.5 / \sigma \text { SAR }\left[\mu W / \mathrm{cm}^{2}\right] \tag{9}
\end{equation*}
$$

Using the data in Table 1 a relationship between $\sigma$ and frequency has been derived, for example,
for brain tissue:

$$
\begin{equation*}
\sigma=0.27+0.0973 \ln (f) \tag{10}
\end{equation*}
$$

and for muscle tissue: $\quad \sigma=0.672+0.0877 \ln (f)$
where f is the frequency in MHz .
Hence, for example, an SAR of $0.00015 \mathrm{~W} / \mathrm{kg}$ for isolated frog hearts at 240 MHz ( $\sigma=$ $1.15 \mathrm{~S} / \mathrm{m}$ ) corresponds to exposure of $0.1 \mu \mathrm{~W} / \mathrm{cm}^{2}$ and for Von Klitzing's human brain EEG at $150 \mathrm{MHz}(\sigma=0.76 \mathrm{~S} / \mathrm{m})$ and $0.001 \mathrm{~W} / \mathrm{kg}$ to $0.7 \mu \mathrm{~W} / \mathrm{cm}^{2}$

The estimate of the SAR for a whole body or body part is a complex calculation because of different tissue densities and electrical conductivities of each tissue type, and the variable size of components of the body which influences the efficiency of absorption of the EMR, all of which varies with wavelength and frequency of the EMR.

Gandhi (1980) gives empirical formulae for the whole body averaged SAR for a 1 $\mathrm{mW} / \mathrm{cm}^{2}$ exposure as a function of the signal frequency, for when the electric field vector is parallel to the length dimension. Expressing the coefficient in Eq. 10 as $R(=2 \rho /(3.77 \sigma)$ and using the units for $R$ of $W / k g$ per $\mu W / \mathrm{cm}^{2}$, Gandhi's model is:

$$
\begin{aligned}
& \text { Resonant Frequency } \mathrm{f}_{\mathrm{r}}=114 / \mathrm{L} \mathrm{MHz} \\
& \qquad \mathrm{~S}_{\text {res }}=15.2 \sqrt{ }\left(\mathrm{~L}^{3} / \mathrm{m}\right)
\end{aligned}
$$

For the sub-resonant range: $0.5 \mathrm{f}_{\mathrm{r}}<\mathrm{f}<\mathrm{f}_{\mathrm{r}}$

$$
\begin{equation*}
R=5.2 \times 10^{-3} \mathrm{~L}^{2} / \mathrm{m}\left(\mathrm{f} / \mathrm{r}_{\mathrm{r}}\right)^{2.75} \tag{12}
\end{equation*}
$$

For the supra-resonant range: $\mathrm{f}_{\mathrm{r}}<\mathrm{f}<1.6 \mathrm{~S}_{\mathrm{res}} \mathrm{f}_{\mathrm{r}}$

$$
R=0.595 \mathrm{~L} /(\mathrm{mf})
$$

where $f$ is the frequency of the incident signal in MHz .
L is the long dimension in m , and
$m$ is the mass of the person in kg .
For example, for an incident signal of 300 MHz , an adult with $\mathrm{L}=1.8 \mathrm{~m}$ and $\mathrm{m}=80 \mathrm{~kg}, \mathrm{f}_{\mathrm{r}}=$ 63 MHz , it is in the supraresonant range and $\mathrm{R}=4.44 \times 10^{-5}$. For a child with $\mathrm{L}=0.9 \mathrm{~m}$ and $\mathrm{m}=25 \mathrm{~kg}, \mathrm{f}_{\mathrm{r}}=127 \mathrm{MHz}$ and $\mathrm{R}=7.14 \times 10^{-5}$. Hence an incident RF signal at 300 MHz and power density of $20 \mu \mathrm{~W} / \mathrm{cm}^{2}$ would produce an SAR for the adult of 0.0009 W/kg and the child of $0.00179 \mathrm{~W} / \mathrm{kg}$, two times higher for the child than the adult. This ratio remains the same for all frequencies since it is determined by the L/m ratio.

Figure 1 shows the calculated SAR levels produced by a content energy flux of $1 \mathrm{~mW} / \mathrm{cm}^{2}$ as a function of frequency, for various body sizes.

When an electric field is induced in a person by the incident RF signal, an electric current flows through the person to earth. Gandhi (1990) has shown that the electric current which is flowing through the feet of a grounded man ( $I_{n}$ in mA ) as the result of an incident electric field $E(V / m)$ is given by:

$$
\begin{equation*}
I_{h}=0.108 E h_{m}^{2} f \tag{13}
\end{equation*}
$$



Figure 1: The average SAR for 3 species exposed to $1 \mathrm{~mW} / \mathrm{cm}^{2}$ with the $E$ vector parallel to the long axis of the body, Durney et al. (1978).

For a 1.75 m person at 40 MHz , at the limit field exposure for the ANSI C95.1 safety standard, $63.2 \mathrm{~V} / \mathrm{m}\left(1 \mathrm{~mW} / \mathrm{cm}^{2}\right)$ this gives a current of 836 mA .

The localized SAR is a function of $\mathrm{I}_{\mathrm{h}}$, which flows down two legs and since $\operatorname{SAR}=J^{2} / \sigma \rho$,

$$
\begin{equation*}
S A R=\left(\ln _{h} / 2\right)^{2} /\left(A_{e}^{2} \sigma_{c} \rho\right) \tag{14}
\end{equation*}
$$

where $J$ is the current density $\left(A / m^{2}\right), A_{e}$ is the effective cross sectional area of the legs, at the ankles it is about $9.5 \mathrm{~cm}^{2}$ even though the physical cross section is about $40 \mathrm{~cm}^{2}$. Hence, assuming a density of $1000 \mathrm{~kg} / \mathrm{m}^{3}$ and $\sigma_{c}=0.77 \mathrm{~S} / \mathrm{m}$, this person has an SAR at their ankles of 251 W/kg. The ANSI standard is based on thermal protection and states that SAR should not exceed $8 \mathrm{~W} / \mathrm{kg}$ for any 1 g of tissue in occupational exposures and 1.6 W/kg for public exposures. Since the exposure $S$ is proportional to SAR, to protect a well grounded adult person from heat exceeding $1.6 \mathrm{~W} / \mathrm{kg}$ in the ankles the limit exposure near the resonant frequency would need to be reduced to $6.4 \mu \mathrm{~W} / \mathrm{cm}^{2}$. Insulating people with rubber soled shoes for example, reduces the induced current by 60 to $80 \%$. However, very young children frequently play with bare feet and so can often be in a well grounded state.

### 6.2 Thermal limits for young children:

An exposure of $1 \mathrm{~mW} / \mathrm{cm}^{2}$ (whole body mean SAR=0.4 W/kg), for a 10 yr old child ( $\mathrm{h}=$ $1.38 \mathrm{~m}, \mathrm{f}=50.7 \mathrm{MHz}, \mathrm{A}_{\mathrm{e}}=6.1 \mathrm{~cm}^{2}$ ) $\mathrm{SAR}=371 \mathrm{~W} / \mathrm{kg}$; for a 5 yr old child $(\mathrm{h}=1.12 \mathrm{~m}, \mathrm{f}=$ $62.5 \mathrm{MHz}, \mathrm{A}_{\mathrm{e}}=4.2 \mathrm{~cm}^{2}$ ) $\mathrm{SAR}=534 \mathrm{~W} / \mathrm{kg}$ (Gandhi (1990) and for a 2.5 yr old child (h= 0.9 $\mathrm{m}, \mathrm{f}=74 \mathrm{MHz}, A_{e}=3.0 \mathrm{~cm}^{2}$ ) SAR $=603 \mathrm{~W} / \mathrm{kg}$. Hence, using simple ratios since S is directly proportional to SAR, for children the allowable exposure to limit the heating of any 1 g of tissue to $1.6 \mathrm{~W} / \mathrm{kg}$ is $4.2 \mu \mathrm{~W} / \mathrm{cm}^{2}$ for a 10 yr old, $3.0 \mu \mathrm{~W} / \mathrm{cm}^{2}$ for a 5 year old and $2.65 \mu \mathrm{~W} / \mathrm{cm}^{2}$ for a 2.5 yr old.

Professor Gandhi notes that an empirical fit to heating rate as a function of SAR yields $0.0045 \times$ SAR $(\mathrm{W} / \mathrm{kg}){ }^{\circ} \mathrm{C} / \mathrm{min}$. If a 2.5 yr old toddler is exposed to $1 \mathrm{~mW} / \mathrm{cm}^{2}$, then the heating rate will be $2.73^{\circ} \mathrm{C} / \mathrm{min}$. Prof Gandhi concludes that for the ankle section "substantial rates of surface temperature elevation are anticipated."

### 6.3 Standards give inadequate protection even for thermal effects in children:

The Australian and New Zealand joint standard does not protect from these localized heating effects through its use of electric field intensity and related exposure limits based on whole body average SARs only, allowing 0.4 W/kg for occupational exposure and $0.08 \mathrm{~W} / \mathrm{kg}$ for public exposure.

Even at our lower public exposure level of $0.08 \mathrm{~W} / \mathrm{kg}$ maximum heating rates in toddler's ankles is about $0.55^{\circ} \mathrm{C} / \mathrm{min}$. This has serious implications when children are exposed to FM signals in the frequency band 20 to 150 MHz especially. Since the public exposure standard must be adequate to protect even the most vulnerable, if they are set to only deal with thermal effects, then in the sub- 150 MHz range they should be adequate to protect a baby from adverse heat levels. This would require the allowed maximum level to be set at $2 \mu \mathrm{~W} / \mathrm{cm}^{2}$ or less, simply to meet the $1.6 \mathrm{~W} / \mathrm{kg}$ limit.

These limits will be somewhat higher away from the optimal frequency for maximum induced current flow, but these figures illustrate the vulnerability of body parts to high levels of localized heating and to the greater vulnerability of young children.

While short-term exposures can give dangerous heating effects down to exposure levels far below the current "safety standard" when near the absorption frequency maximum, legitimate concerns exist about changes in our brains and at the cellular level the shortterm exposures might cause. There is also clear evidence about chronic exposures at far lower levels of exposure which have the potential to alter the ambient electromagnetic environment in ways which are potentially harmful through effects on reproductive processes, brain function and metabolism, sleep disruption, immune system suppression and EMR probably causes an increase in the risk of cancer.

### 6.4 Natural Electromagnetic Environment:

The earth's static magnetic field is about $30-50 \mu \mathrm{~T}$ and static electric field (in fair weather conditions) is about $150 \mathrm{~V} / \mathrm{m}$. Our bodies are well adapted to these static fields and to the radiation from the sun. We are now seeing how small changes to a minute part of the solar spectrum, UVA and UVB, are producing significant increases of skin cancer in nonblack skinned people. Legitimate concern can be raised about the significant increases in


#### Abstract

population exposures to other parts of the EM spectrum from ELFs to millimeter microwaves.




Figure 2: The signal of a typical cell site near the mast ( $2 \mu \mathrm{~W} / \mathrm{cm}^{2}$ ) against the thermal radiation background in the frequency range around that used by cell sites.

Our background thermal environment emits around $400 \mathrm{~W} / \mathrm{m}^{2}\left(40,000 \mu \mathrm{~W} / \mathrm{cm}^{2}\right)$ of "thermal" radiation, the vast majority of which is in the infrared. The part of this which forms the natural sources of oscillating EMR fields summed over the range up to 300 GHz is $0.3 \mu \mathrm{~W} / \mathrm{cm}^{2}$, Repacholi (1983). In the radiofrequency range it is less than $10 \mathrm{pW} / \mathrm{cm}^{2}$ because over $99.996 \%$ of the $0.3 \mu \mathrm{~W} / \mathrm{cm}^{2}$ comes from above 10 GHz , whereas most of our spectrum use for telecommunications, (radio, TV, RT and cell phones, satellite communication, radar) are in specific frequency bands below this frequency. Hence from the ELF to RF/MW parts of the EM spectrum we have massively increased our ambient exposure, especially following developments since the Second World War.

Figure 2 shows how a cell site local exposure level of $2 \mu \mathrm{~W} / \mathrm{cm}^{2}$ stands out against the thermal background, by a factor or about $2 \times 10^{8}$.

The ionosphere has a net positive charge which creates a static electric field and the dynamics of thermal convection in the earth's core produce the earth's magnetic field. Thunderstorms create short-term localized field variations as they strongly alter the local electric field and lightning produces emissions broad spectrum emissions of RF/MW in bursts called sferics. These can be heard on radios as a burst of static.

Long-term natural ELF signals arise from thunderstorm sourced electromagnetic energy being ducted around the world in the cavity between the earth and the ionosphere. These are called the Schumann Oscillations. Their fundamental frequency is 7.8 Hz , with
harmonics at $14.1,20.3,26.4$ and 32.5 Hz . Their amplitude is about $0.2 \mathrm{mV} / \mathrm{m}$ for a 1 Hz bandwidth, Campbell (1967). Assuming each resonant frequency is associated with a 1 Hz bandwidth, the total field strength will be $1 \mathrm{mV} / \mathrm{m}$ or $0.1 \mathrm{~V} / \mathrm{cm}$. From Eq. 3 this is equivalent to an energy flux of $0.27 \mathrm{pW} / \mathrm{cm}^{2}$.

### 6.5 Modification of the natural environment:

While life on earth is well adapted to the static magnetic and electric fields and to minute intensities of naturally occurring ELF and RF/MW fields primarily from thunderstorms, biological tissues and organisms have, in this century, become exposed to ELF, RF/MW (VHF and UHF) fields at intensities which are thousands of times higher than they were at the turn of the century ( $<10 \mathrm{pW} / \mathrm{cm}^{2}$ ). The median public RF exposure measured by the U.S.E.P.A. in U.S. cities in 1979 was $0.005 \mu \mathrm{~W} / \mathrm{cm}^{2}$ or $5,000 \mathrm{pW} / \mathrm{cm}^{2}$, Tell and Mantiply (1980). About $1 \%$ of the population lived in more than $1 \mu \mathrm{~W} / \mathrm{cm}^{2}$ or 1 million $\mathrm{pW} / \mathrm{cm}^{2}$.

### 6.6 Healthy vs vulnerable people:

People who live or work close to transmission facilities have mean daily exposures of about 1 to $20 \mu \mathrm{~W} / \mathrm{cm}^{2}$, but when averaged over a year their mean is of the order of 0.2 to $5 \mu \mathrm{~W} / \mathrm{cm}^{2}$. In the work and military environment a "healthy worker" effect exists employment selection screens out the sick, young and elderly. Hence while occupational and military exposures can be somewhat greater than average in certain cases, studies on these workers tend to significantly underestimate the effect similar exposures would have on the vulnerable in society. It is the vulnerable groups in which overall sickness rates are higher. So when reviewing national morbidity and mortality statistics the vulnerable groups are over represented and there are large groups of "healthier than average" people who are under-represented in the health statistics.

The IRPA RF exposure standard is an acknowledged thermal standard based on an occupational exposure limit of $0.4 \mathrm{~W} / \mathrm{kg}$. Greater protection is offered to the general public because this includes the vulnerable. Hence the Public Exposure Limit of 0.08 W/kg, which will avoid burns, and slight heating effects in infants and the frail elderly on hot sunny days when they exercise, Repacholi (1993). However such standards do not deal well with hot spots, nor with potential nor actual health effects from chronic exposure.

## 7. Absorption Mechanisms:

Many epidemiological studies show statistically significant associations between EMR exposure and adverse health effects such as cancer, genetic and reproductive problems; animal experiments show increases in cancer, immune system impairment and reproductive problems; and in vivo cell experiments show many biological and biochemical changes with exposure to ELF and RF/MW at nonthermal levels of exposure. While these are sufficient to show highly probable effects, a large body of opininon which dominates the international and national standards setting bodies, holds that the above set of research is fundamentally suspect because there is no physically plausible mechanism through which ELF fields or RF/MW radiation can be absorbed in and alter the physical electromagnetic environment of molecules and cells.

While this physics dominated view is no real reason to ignore the implications and findings of biochemical, toxicological and epidemiological research. For example, the highly repeated result that ELF and ELF modulated RF/MW radiation causes and efflux
of calcium ions through the cell membrane. Many studies show broken DNA and damaged chromosomes with EMR exposure, providing evidence of genetic damage and cancer potential. We cannot ignore these simply because we cannot at this time identify and describe every step in the process. However, it would be scientifically significant and very satisfying to those which wish to complete the picture of they ways in which EMR interacts with biological systems, if satisfactory and identifiable physical mechanisms could be found.

### 7.1 Whole Body Absorption:

Just as a radio or television can detect and decode modulated RF/MW signals at RMS intensities at a minute fraction of the static electric and magnetic fields, using frequency banded tuned oscillators, so can biological organisms absorb and resonate in modulated RF/MW fields. Human and animals bodies act as antennae for whole body energy absorption and tissue cells act as resonant absorbers. The whole body absorption determines the energy absorbed, the fields induced and the currents which flow through the body. Many fundamental cellular processes involve electric fields and the flow of ions. Hence induced changes from imposed EM fields can and do alter the cellular processes and their ionic balance.

Molecular and cell level electric charges, and electric and magnetic fields play a fundamental role in these processes and in the organization of the complex cellular and macrocellular structures. With these understandings has come new insights. Gone from the outset are notions of isotropy. We see schemes of biochemical and biophysical organization of unparalleled complexity. Concepts of linear systems disappear and are replaced by non-linear, non-equilibrium thermodynamics. Observations and models are consistent with quantum processes involving long-range interactions between electrical charges on cell surface macromolecules, Adey (1992c).

It is at the level of the cell that non-thermal effects become very evident. It has been asserted by some that thermal noise, expressed in the term kT as a function of the Bolzmann constant and the absolute temperature, must remain a monolithic threshold below which no biological threshold can exist. Some biophysicists and others still hold this view despite the wealth of physiological evidence that sensory thresholds descend substantially below the floor of thermal noise, as happens for example in the auditory system of the ear, Adey (1993). Also, attention is now directed to newly defined roles for free radicals, that may also participate in highly cooperative detection of weak electromagnetic fields, "even at levels below the thermal (kT) noise, McLaughlan (1992), and Grundler et al. (1992).

### 7.2 Radical Pair Mechanism

Many reseachers have suggested that thermodynamical considerations do not necessarily impose a final limit with respect to the primary EMR interaction step, Walleczek (1994). Nonlinear biological processes provide a number of possibilities, one of which is the Radical Pair Mechanism (RPM). During such processes the magnetic field does not change the nature of biochemical reaction, only the product yield.

Free radicals are chemical species possessing one or more unpaired electron, which makes them generally very reactive. They are produced continuously in cells either as
the accidental by-products of metabolism or deliberately during, for example, phagocytosis. The most important reactants in free radical biochemistry in aerobic cells are oxygen and its radical derivatives, superperoxide and hydroxyl radical, hydrogen peroxide and transition metals.

The reactivity of free radical molecules is determined by the overall spin state of their outer shell electrons. For this reason, their chemical reactivity is spin-selective. Spinning electrons are associated with a magnetic component. The key to understanding the RPM is the fact that applied static or time varying magnetic fields can modify the electron spin states during free radical formation steps, and consequently, alter radical-dependent biochemical reaction rates. Numerous radical-dependent reactions are known to occur in cells, including reactions which may affect T-cell signal transduction steps.

### 7.3 Magnetic Resonance Mechanisms:

The search for mechanisms by which ELF signals could create biological resonances was first directed towards possible joint actions of static fields and oscillating ELF magnetic fields by Liboff et al. (1985) and Blackman et al. (1985). Blackman proposed a nuclear magnetic resonance model based on the oscillating field being perpendicular to the static field. Liboff proposed the cyclotron resonance model for situations where the fields are parallel. Lednev (1991), using a different theoretical approach, characterized field exposure parameters with sufficient precision to test possible cellular interations with specific combinations of oscillating and static magnetic fields. The Lebnev ion parametric resonance model describes protein-bound ions as spatial oscillators with a series of virbrational frequencies that depend on the bond energy, and the charge and mass of the ligand-bound ion. With the formation and breaking of coordination bonds between protein and a chain of ions, the ions oscillate around a mean energy level, due to random thermal motion. The energy level of the bound ions split into two sub-levels in the presence of a static magnetic field, and the splitting of the two levels occurs at a frequency equal to the cyclotron frequency (proposed by Liboff et al.)

Evidence for sensitivity of biological systems under cyclotron conditions has been extensive but so far inconclusive, with inconsistencies that suggest action of uncontrolled intercurrent factors, Liburdy (1995).

### 7.4 Cell-based resonant absorption of RF/MW:

Water is known to strongly absorb microwaves. This characteristic is used in microwave cooking. Living biological tissue is water-rich. It is reasonable then to expect that water in tissue will absorb microwave energy.

The surprise is the response to absorbed RF radiation. During the MacIntyre Case, the Court was not told about the research of Liu and Cleary (1995) in which they showed, using classical scattering theory, that both radiofrequency (RF) and microwave (MW) radiation is resonantly absorbed in the bound water layers on the cell membrane. This creates an electric field across the cell membrane in the case of RF and thermal fields in the case of MW, Figure 3.

This research provides vital link between the whole body absorption of RF/MW radiation by human beings and animals and the altered cellular biochemistry demonstrated in isolated cell lines in laboratory experiments. It is another direct rebuttal of the thermal view because the effect varies strongly with frequency between electric field and heating.

Liu and Cleary (1995) have shown, in a biophysical model of cells, that the bound water layer on each side of the cell membrane, resonantly absorbs radiofrequency and microwave radiation at the surface of the cell membrane.


Figure 3: Spatial distribution of the maximum induced E-field component ( $E_{x}$ ) and SAR (SAR ${ }_{x}$ ) on the $x$-axis of a five component mammalian cell model (extracellular medium, bound-water layer on exterior cell membrane surface, cell membrane, bound-water layer on inside surface of cell membrane surface, cytoplasm) exposed to 27 or 2450 MHz continuous plane-wave electromagnetic radiation. Incident field strength, $1 \mathrm{~V} / \mathrm{m}$. $\left(0.3 \mu \mathrm{~W} / \mathrm{cm}^{2}\right)$
 $(2.45 \mathrm{GHz})$ signal. For the 27 MHz signal the cell has a massive change in the electric
field in the membrane and very little heat absorption. For the 2.45 GHz microwave signal there is a very small change in the electric field from the outside to the inside of the cell, but there is a massive, resonant absorption of heat energy on the inside and outside surface of the cell due to the presence of the bound water layers. I have asked Professors Liu and Cleary to re-run the model for 915 MHz , a common mobile phone frequency. The biophysics suggests that this would show a lower heating response compared to 2450 MHz and a lower electric field difference than the 27 MHz signal.

This very significant paper shows that classical physics predicts resonance absorption of RF/MW radiation at the cell level. Hence there are energy, thermal and electric gradients which can alter the biochemical and chemical reactions at the cellular level. Professor Cleary pointed out to me that this paper also shows a difference in the resonant absorption on the various axes of the cell, depending on the polarization of the incident EM radiation. In a spherical model these differences are as great as a factor of 13 above average. That means, he explained, a mean SAR of $0.007 \mathrm{~W} / \mathrm{kg}$ could well have localized cellular based SARs or $0.10 \mathrm{~W} / \mathrm{kg}$. He was worried about the effects of cell phones on brain cells in the head near the antenna, in the light of this and his other work on cell-cycle timing changes and DNA changes in Chinese Hamster Ovary ( CHO ) cells, for example.

The absorption of the RF/MW energy at the cell membrane gives a basis to move on to the biochemistry of altered cellular processes, including the effect on signal transduction, cell cycle timing, on ODC in tumour development, on calcium ions and the immune system, and on melatonin in relation to free radical control and elimination. These in turn relate to adverse health effects such as cancer and immuno-competence, sleep disturbance, memory dysfunction and concentration disruption.

## 8. The Melatonin Mechanism:

On the macro-scale, human and animal circadian rhythms are driven by the day/night cycle with a phase-lock synchronization provided by environmental ELF fields ( $\mathrm{E}<0.3$ $\mathrm{pW} / \mathrm{cm}^{2}$ ). A fundamental physiological aspect of the circadian rhythm involves the pineal gland and the secretion of a neurohormone called melatonin. Light falling on the eye's retina produces signals which are biochemically amplified around a million times, to stimulate the pineal gland to reduce its melatonin output.

### 8.1 Pineal Melatonin - A plausible mechanism for EMR effects

Pineal and serum melatonin concentration drops during the day and rises overnight, Figure 4.

Melatonin production is very well understood. A schematic of the way in which the light induced signal passes from the retina of the eye, being amplified to release many molecules of the neurotransmitter, norephinephrine (NE), which is received by receptors on the surface of the pineal gland cell (Pinealocyte).


Figure 4: Blood melatonin levels for 4 adult males over a 24 h period, Reiter (1994).

Tryptophan is converted to serotonin which is then converted to Melatonin at a rate controlled by an enzyme NAT ( N -acetyltransferase) which has been activated or limited through protein synthesis from amino acids controlled by cyclic AMP, Figure 5.


Figure 5: The connection of the eyes (retina) to the pineal gland, represented by a single pinealocyte, and the synthesis of melatonin within the gland.

Tryptophan, an amino acid from the blood, is converted to the hormone melatonin, which is quickly released into the capillaries of the gland. The enzymes which catalyze the conversion of serotonin to melatonin include N -acetylifansferase (NAT) and hydroxyindole-O-methyltransferase (HIOMT). The pineal gland produces melatonin at night since the nerve endings which end in the pineal gland release the neurotransmitter norephinephrine (NE) which interacts with the b-and a-adrenergic receptors on the cell membrane; these interactions initiate the processes which control melatonin production. ATP, adenosine triphosphate; PVN paraventricular nuclei; SCN, suprachiasmatic nuclei; SCG, superior cervical ganglia. The melatonin easily passes through the cell wall into the blood stream to be dispersed throughout the body. The pineal gland is located near the centre of the brain. It is an endorcrine organ which produces most of the melatonin which is found in the blood, figure 6.


Figure 6: A mid-saggital section of the human brain showing the location of the pineal gland, Reiter (1994).

Once melatonin is produced it is the molecule's high ability to pass through the cell membrane which allows it to escape from the pinealocyte to the blood. Once in the blood melatonin has access to every cell in the body. It passes through the cell membranes where every nucleus has receptors for it. A few cell membranes have receptors. These may mediate the 24 h circadian rhythms of the endocrine system. In the nucleus melatonin plays a role in regulating the effects of the indole on gene expression. The ability of melatonin to enter all cells is also essential for one of the other important functions of melatonin, namely, its ability to scavenge the highly toxic hydroyl radical $(-\mathrm{OH})$.

The production of oxygen-based free radicals, such as $\bullet \mathrm{OH}$ is a consequence of the utilization of oxygen by organisms. About 1-2 \% of inspired oxygen ends up as toxic free radicals. It is generally considered the $\cdot \mathrm{OH}$, because of its high reactivity, is the most devastating to macromolecules such as DNA, proteins and lipids. The cellular damage produced by free radicals is generally referred to as oxidative stress, Reiter (1994).

Because of its action in removing free radicals, melatonin is probably the most efficient natural cell protection and oncostatic agent in our bodies. Every night, our pineal produces large quantities of melatonin which flood almost every cell in our body, cleaning out the free radicals and assisting cell division to take place with undamaged DNA. As we age our nocturnal peak melatonin production falls markedly, making elderly people much more prone to cancer. To test the cancer protecting properties of melatonin, Tan et al. (1993), injected rats with a chemical carcinogen, safrole. Safrole normally damages DNA because it induces the production of large numbers of free radicals. Rats injected with Safrole were found to have extensive DNA damage after 24 h . When melatonin was also injected, the DNA damage was reduced by $99 \%$. Since damaged DNA can undergo mutation it may result in the growth of a tumour. Thus melatonin is clearly a potent cellular protector against cancer initiation.

Three independent laboratories, Battelle PNL (Wilson), U.C. Riverside (Luben) and the U.S. EPA (Blackman), have shown that 60 Hz modulated magnetic fields in the 1 to 12 mG range, almost completely negate the oncostatic effect of melatonin in human breast cancer cells, with a dose-response relationship. Wilson et al. (1986) showed significant reductions in pineal melatonin in living rats when they were chronically exposed to 60 Hz modulated electric field at $1.7-1.9 \mathrm{kV} / \mathrm{m}$ for 20 h per day, for 30 days, Figure 7.


Figure 7: Pineal melatonin (top) and NAT activity (bottom) in groups of rats exposed to a modulated electric field for 1 to 4 weeks. The glands of the animals were collected at night. In the sham-exposed animals the pineal melatonin and NAT levels were always high. However, after both 3 and 4 weeks of exposure to the electric field, both parameters were depressed ( $p<0.001$ ).

The review paper by Professor Russell Reiter (Reiter (1994), was prompted by a number of epidemiological studies in which an increased incidence of cancer was reported in individuals living or working in an environment of higher than normal artificial electromagnetic fields. Because of the key role of melatonin is decreasing the likelihood of cancer because of its effect of removing free radicals, Prof. Reiter has been researching the effects of EM fields on melatonin production. He concludes:
"Reduction of melatonin at night, by any means, increases cell's
vulnerability to alteration by carcinogenic agents. Thus, if in fact artificial
electromagnetic field exposure increases the incidence of cancer in
humans, a plausible mechanism could involve a reduction in melatonin
which is a consequence of such exposures."

He also notes:
"Epidemiologists should look for other possible changes, including psychological depression, fatigue, sleep inefficiency, chronic feelings of jet lag, endocrine disturbances and other symptoms; all these may result from a chronically low melatonin rhythm."

Lerchl et al. (1988) samples for serotonin and its derivatives by periodically inverting the magnetic field at night. Figure 8.


Figure 8: Pineal serotonin (5-HT) and 5-hydroxyindole acetic acid (5-HIAA) levels in rats and mice (cross-hatched bars) with and without (clear bars) exposure to pulsed static MF at night. Both 5-HT and 5-HIAA levels increased as a result of the exposure; these changes are consistent with a reduced melatonin production. * $p<0.05$ and *** $p<0.001$ vs control; $+++p<0.05$ control male mice, from Lerchl et al. (1988).

Hence there exists a plausible mechanism for cancer and a host of disorders, most of which will be identified below as discovered in epidemiological studies. Often the papers or reports which identify statistically significant increases in cancer or other complains associated with above average exposure to EM fields, have rather weak conclusion, citing the lack of a plausible mechanism. In fact their conclusions can be much stronger because of the existence of the melatonin mechanism and several others which will be described.

Dr Reiter's review paper, quoted above, demonstrates the fundamental role of cells and the vast amount of cellular biochemistry which is known. It also documents biological mechanisms which are chemical and biochemical and are definitely not thermal.

### 8.2 Hypothesis for modulated RF/MW effects on melatonin:

Since it has been shown:

- That ELF electric fields do reduce melatonin production in living rats brains;
- That RF/MW signals produce tissue level electric fields about a million times higher than imposed ELF signals, Adey (1981);
- That RF/MW signals are resonantly absorbed at the cell membrane, Liu and Cleary (1995);
- That altering the electric and thermal fields on the surface of the cell membrane change the binding characteristics of $\mathrm{H}^{+}$and $\mathrm{Ca}^{++}$ions on the outer surface of the membrane;
- That modulated RF/MW has been shown to induce significant calcium ion efflux from cells;
- That it known that the cyclic AMP signal transduction pathway and the Calcium ion signal transduction pathway interact; and
- That in the pinealocyte cell the cAMP pathway assists in regulating the transformation of serotonin to melatonin;

The calcium ion mediated responses to neurotransmitters on the membrane of the pineal cells has been discussed by Wilson et al. (1989) in relation to ELF induced melatonin reduction. Thus it is highly probable that pinealocytes exposed to modulated RF/MW will experience an outflow of calcium ions, a reduction of the cAMP signal transduction activity and a reduction in the production of melatonin. This is a highly plausible mechanism to explain why RF/MW can reduce pineal melatonin production with consequent the adverse health effects.

### 8.3 Direct and Indirect evidence of EMR effects on melatonin:

Melatonin is associated with sleep quality, and sleep quality can be assessed by EEG measurements and poor sleep quality can be reflected in loss of energy, chronic fatigue symptoms, poor concentration and impaired learning and memory. Hence research association these symptoms with EMR exposure are relevant to identifying the potential or actual effects of EMR in reducing melatonin. Reduced nocturnal melatonin leads to reduced nocturnal serotonin because of the large nighttime rise in melatonin output from the pineal gland through conversion of serotonin. However the daytime levels of melatonin and serotonin are not so strongly related.

### 8.3.1 Biological Sensors of Environmental Fields:

Biological systems are sensitive to external EM fields for many functions. There is unequivocal experimental evidence that fields from ELF to UHF ( 10 Hz to 450 MHz ) interact directly with brain tissue, Adey (1981). Dr Adey cites bird navigation, bird circadian rhythms, monkeys' subjective time estimations and human circadian rhythms which are all related to tissue level gradients of about $10^{-7} \mathrm{~V} / \mathrm{cm}$. Weaver and Astumian (1990) use a mathematical model to calculate that membrane macromolecules can directly respond to $10^{-3} \mathrm{~V} / \mathrm{cm}$ while much smaller fields, about $10^{-7} \mathrm{~V} / \mathrm{cm}$, can be detected if signal averaging occurs through field-inducted variation in the catalytic activity of membrane associated enzymes.

The fact that intrinsic cell neuroelectric gradients are far higher than these observed tissue gradients, e.g. Membrane Potential $10^{5} \mathrm{~V} / \mathrm{cm}$, Synaptic Potential $10^{3} \mathrm{~V} / \mathrm{cm}$ and Electro-encephalogram 0.02 to $0.05 \mathrm{~V} / \mathrm{cm}$, attests to the vital role of modulation of EMR and the existence of amplification processes at the cellular level, Adey (1989).

### 8.3.2 Far greater tissue penetration of ELF modulated RF/MW than ELF alone:

Induction of electric fields in tissue at the cellular level varies with the intensity and the nature of the environmental field. Typical endogenous EM fields, with ELF modulation, induce fields in the order of $10^{-1}$ to $10^{-7} \mathrm{~V} / \mathrm{cm}$ in the pericellular fluid (fluid surrounding the cell). RF/MW fields penetrate the organ or body much more effectively than the ELF fields.

For example, when chick brains were exposed to an applied $56 \mathrm{~V} / \mathrm{m}$ field:

- An ELF field 1-32 Hz , induced a tissue gradient of $10^{-7} \mathrm{~V} / \mathrm{cm}$.
- An RF field, 147 MHz , ELF modulated, produced a tissue gradient of $10^{-1} \mathrm{~V} / \mathrm{cm}$.

Both of these signals significantly changed the calcium ion efflux from the chick brain tissue, Bawin and Adey (1976).

Thus the RF/MW field produced a cellular tissue gradient 1 million times higher than the ELF field of the same external field strength. This shows the highly penetrative nature of RF/MW fields compared to ELF fields. Since the energy flux relates to the square of the electric field gradient strength, Eq. 1, the energy imparted to the cell tissue by RF/MW modulated radiation is many orders of magnitude higher than the same external strength of ELF field.

### 8.3.3 The Max Planck Institute Identifies EMR Circadian Rhythm Effects:

Human biometeorology has a great deal to teach us about the effects of natural electromagnetic fields and biological reactions. There is a strong evidence, Wever (1974), that natural ELF signals such as the Schumann Oscillations, coupled with the earth's magnetic field, help to phase lock the 24 h circadian rhythm in people and animals.

Isolation experiments show that the dark/light cycle is insufficient to fully regulate the circadian rhythm but other environmental stimuli, called "Zeitgebers" by the German researchers, are also required to synchronize the rhythm. These must be globally available, naturally occurring signals since almost all terrestrial life is tuned to the 24 hr cycle The day/night light cycle is the primary driver of circadian rhythm for when people are deprived of this light cycle their daylength drifts, generally becoming longer. An extensive research program has been carried out by the Max Planck Institute over several years and involving over 200 subjects.

Two isolation rooms were used, one of which was also shielded from environmental electromagnetic fields (Room 2). In a simple "free running" experiment, it was found that the mean day period was 24.87 h for Room 1 and 25.26 h for Room 2. The difference between the light isolated (Room 1) and the light and electromagnetic field isolated subjects (Room 2) was significant at the $p<0.01$ level, Figure 8 . Those isolated from the extremely small environmental electromagnetic fields had mean daylengths that were significantly longer and more variable. The standard deviation of their variation in daylength was also significantly larger Room 2 and the number of internal desynchonizations was greater in Room 2 with $p<0.001$.

Depriving people of access to the natural electromagnetic fields made a very significant difference in their daily rhythm. Other experiments with a very low level artificial ELF signal were carried out. This $2.5 \mathrm{~V} / \mathrm{m}$ (peak-to-peak) 10 Hz signal (rms-amplitude of 1.77 $\mathrm{V} / \mathrm{m}$ giving $\mathrm{S}=0.83 \ldots \mathrm{~W} / \mathrm{cm}^{2}$ ) reduced the desynchronization significantly ( $p<0.001$ ). In many experiments, no case of internal desynchronization occurred as long as the 10 Hz field was in operation, Weber (1974), Figure 9. No effects were found with static electric fields.

Weber (1974) concludes that their research gives:

## "significant proof that electromagnetic fields in the ELF range influence the human circadian rhythms and therefore human beings."

People who were deprived of the light/dark cycle and natural electromagnetic fields with intensities of the order of $0.3 \mathrm{pW} / \mathrm{cm}^{2}$ showed significant shifts in circadian rhythm while an artificial ELF field of $0.8 \mu \mathrm{~W} / \mathrm{cm}^{2}$ significantly reduced the desynchronization, mean period and variance of the circadian rhythm.


Figure 9: Free-running circadian rhythm of a subject living under strict isolation from environmental time cues, during the first and third section protected from natural and artificial electromagnetic fields, during the second and fourth sections under the influence of a continuously operating 10 Hz electric field of $2.5 \mathrm{~V} / \mathrm{m}$, Wever (1974).

The biological mechanism involved in brain detection of extremely low intensity ELF signals is not discussed by Wever. This substantial project, carried out by a prestigious laboratory, establishes that human beings have the ability to sense and react to extremely small electromagnetic signals. The invoivement of the circadian rhythm points to a pineal gland involvement.

### 8.3.4 Human Research relating to EMR and melatonin reduction:

Clearly mammals can sense and react to extremely low levels of EMR but can and do human beings? The Max Planck Institute research shows a strong and repeatable effect on human circadian rhythm involving ELF signals. This implies a pineal gland reaction and is likely to involve melatonin. As set out in sections 8.3.1 and 8.3.2 above, greater tissue penetration of ELF signals carried by RF/MW radiation, strongly implies that effects on sleep, brain function, immune system, reproduction and cancer should be found in people since the pineal gland and melatonin is involved in each of these. for example:

- Lissoni et al. (1988) report: "It has been known for many years that the pineal gland is involved in regulating tumor growth". Through the "functional activity of the pineal gland in neoplastic diseases."
- Bullough et al. (1996) state: "Effects of light and electromagnetic fields (EMFs) on pineal function could have implications for long term risk of breast cancer, reproductive irregulatities, or depression."
- Sandyk et al. (1992) write "In the following communication, we propose that the deficient pineal melatonin functions in early pregnancy may be causally related to the development of spontaneous abortions in cases where chromosomal anomalies or structural abnormalities of the uterus have been excluded."
- Reiter (1994) concludes: "Epidemiologists should look for other possible changes including psychological depression, fatigue, sleep inefficiency, chronic feelings of jet lag, endocrine disturbances and other symptoms; all these may result from chronically low melatonin rhythm."

Few projects have attempted to directly measure the reduction of melatonin in human beings due to EMR exposure. Some which have, have only assessed daytime alterations. These are prone to great variability and little statitical significance because of individuals' large variations in melatonin from day to day and the low daytime levels of melatonin in all people. The Schwartenburg Study, Altpeter et al. (1995) for example, sample melatonin in people after they had woken up. The people exhibited a statistically significant doseresponse relationship for sleep difficulty, and indication of melatonin reduction, but their early morning melatonin levels showed no statitically significant reductions. This is not surprising because the melatonin levels which related to sleep are those of the evening and nighttime. The exposure involved a shortwave radio transmitter in Switzerland. The primary description of a combination of the symptoms reported was called "Chronic Fatigue Syndrome". Sleep disruption improved when the transmitter went off unexpectedly and unknowingly, showing a strong connection.

### 8.3.4 The Schwarzenburg Study

### 8.3.4.1 Introduction:

Following many years of reported health complains from the vicinity of a shortwave transmission mast, a study by Altpeter et al. (1995) was carried out and revealed statistically significant health and well-being effects which varied systematically with exposure zone. Follow up studies gave strong evidence of the involvement of melatonin as a biological mechanism in relation to sleep disturbance and the reported group of symptoms referred to a "Chronic Fatigue Syndrome".

### 8.3.4.2 Exposure levels associated with the radio tower:

Daily average exposures in the frequency range 3 to 30 MHz for each zone are given in Table 14.

The background exposure level in the frequency range 3 to 30 MHz , was measured during a 3-day period when the transmitter was turned off. Levels averaged $0.083 \mathrm{~mA} / \mathrm{m}$ $\left(0.00183 \mu \mathrm{~W} / \mathrm{cm}^{2}\right)$, with a nocturnal peak of $0.169 \mathrm{~mA} / \mathrm{m}\left(0.0076 \mu \mathrm{~W} / \mathrm{cm}^{2}\right)$ and an afternoon minimum at about 1 pm to 2 pm of $0.041 \mathrm{~mA} / \mathrm{m}\left(0.00045 \mu \mathrm{~W} / \mathrm{cm}^{2}\right)$. This diurnal variation is likely to be related to changes in atmospheric conditions such as those which give better shortwave reception at night than during the day.

Globally this relates to the height of the reflecting electron concentration in the ionosphere, which is higher at night and lower during the day. Locally it relates to atmospheric stratification and ducting of high frequency RF signals, such as radios and radars, as has been observed in Canterbury during nor'westers. This suggests the
probability of nocturnal ducting of the radio signals from the tower, increasing the nocturnal strength in Zones $A, B$, and $C$, and hence making them all prone to sleep disturbance effects (if such is the result of RF exposure as is strongly suggested by Table 18).

The exposure range, mean and median exposures measured in each zone during the transmitter's operation are set out in Table 9.

| Exposure Zone | Exposure Range | Median | Mean ( $\mu \mathrm{W} / \mathrm{cm}^{2}$ ) |
| :---: | :---: | :---: | :---: |
| Zone A : High | $0.031-9.1$ | 0.10 | 0.24 |
| Zone B: Medium | $0.0034-0.074$ | 0.024 | 0.024 |
| Zone C: Low | 0.00046-0.0074 | 0.0004 | 0.0004 |

The median exposure gives the most likely exposure for the population in each zone. For the high exposure (Zone A) this is $0.1 \mu \mathrm{~W} / \mathrm{cm}^{2}$. The maximum exposure is about $1 \%$ of the allowable public exposure level for SW transmissions, and the Zone A median is about $0.01 \%$ of the current standard.

Zone C has readings in the range of frequencies produced by the tower from above and below the mean background level. The higher than background levels are likely to be more prevalent at nighttime due to ducting phenomena except during Föhn conditions.

### 8.3.4.3 Effects associated with the RF Exposure:

The statistically elevated symptoms in the high and medium exposure groups, compared to the low exposure group, include Nervosity and restlessness, Disturbances in falling asleep and difficulty in maintaining sleep, Joint pains, Psychovegetative Index changes, Disturbances of Concentration, General Weakness and Tiredness, Constipation, Diarrhea and Lower back pain, all significant at $p<0.02$ except the first for which $p=0.034$ which is less than the usual significance level of $p<0.05$.

An increased exposure from $1 \mathrm{~mA} / \mathrm{m}$ to $10 \mathrm{~mA} / \mathrm{m}\left(0.038 \mu \mathrm{~W} / \mathrm{cm}^{2}\right.$ to $\left.3.8 \mu \mathrm{~W} / \mathrm{cm}^{2}\right)$ had on Odds Ratio for insomnia of 1.13 (Cl: 1.04-1.23) and from $0.1 \mathrm{~mA} / \mathrm{m}$ to $1 \mathrm{~mA} / \mathrm{m}$ $\left(0.00038 \mu \mathrm{~W} / \mathrm{cm}^{2}\right.$ to $\left.0.038 \mu \mathrm{~W} / \mathrm{cm}^{2}\right), \quad \mathrm{OR}=2.1$ (Cl: $0.95-4.57$ ). Table 10 presents the adjusted Odds Ratios for the primary effects found, which show significant dose response relationships and a highly statistically significant increase with mean exposure increase.

Table 10: Odds Ratios for an increase in 24-hour average exposure from 1 $\mathrm{mA} / \mathrm{m}\left(0.04 \mu \mathrm{~W} / \mathrm{cm}^{2}\right)$ to $10 \mathrm{~mA} / \mathrm{m}\left(3.8 \mu \mathrm{~W} / \mathrm{cm}^{2}\right)$ adjusted for age, sex, attribution, and duration of time lived at the same place.

| Symptoms | OR | 95\% Conf. Interval |
| :--- | :---: | :---: |
|  |  |  |
| Nervosity, Restlessness | 2.77 | $1.62-4.74$ |
| Diff. in falling asleep | 3.35 | $1.86-6.03$ |
| Diff. in maintaining sleep | 3.19 | $1.84-5.52$ |
| Joint Pain | 2.46 | $1.37-4.43$ |
| Limb Pain | 2.51 | $1.15-5.50$ |
| Cough and Sputum | 2.80 | $1.18-6.64$ |



Figure 10: Frequency of different psychovegetative disorders by Zone $\mathrm{A}, \mathrm{B}$, and C . The light bars concern persons aged over 45 years, the dark ones aged 45 or less, Altpeter et al. (1995).

It is important to note that the Odds Ratio in the range from 2.46 to 3.35 is much more significant in this study than the incidence of cancers since the occurrence of symptoms in the low group in this study is around $10 \%$ compared to about $0.007 \%$ in total leukaemia incidence in the Polish Military Study.

Figure 10 and Table 11 show that several complaints, especially for the over 45 agegroup, have statistically significant dose-response curves which Bradford-Hill viewpoint (5) Biological Gradient ascribes a strong weight of evidence. When linked to the melatonin mechanism ((6) Biological Plausibility) this is strong evidence of a probable cause-andeffect. Symptoms which reach this standard ( $p \leq 0.05$ ) include Nervocity/restlesness and genral weakness and tiredness (all ages), and difficulties in falling asleep and maintaining sleep, joint pain, psychovegetative index, constipation and diarrhea (>45).

| Symptom | Zone A (\%) |  | Zone B (\%) |  | Zone | (\%) | P-value |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Nervosity restlessness (<45) | $12 / 55$ | (21.6) | 5/61 | (24.6) | $8 / 85$ | (9.4) | 0.034 |
| ( $>45$ ) | $14 / 50$ | (28.0) | 7158 | (12.1) | $4 / 94$ | (4.3) | <0.001 |
| Difficulties in maintaining sleep (<45) | 7/55 | (12.7) | $4 / 61$ | (6.6) | $4 / 86$ | 4.7)0.194 |  |
| ( $\geqslant 45$ ) | 27/50 | (54.0) | $18 / 58$ | (31.0) | $12 / 94$ | (12.8) | $<0.001$ |
| Limb pain (<45) | $1 / 55$ | (1.8) | $3 / 61$ | (4.9) | 186 | .2) 0.329 |  |
| $\ldots$.. $>45$ ) | $14 / 50$ | (28.0) | 5/58 | (8.5) | $5 / 94$ | 5.3)<0.001 |  |
| Difficulties in falling asleep (-45) | $8 / 55$ | (14.5) | $6 / 61$ | (9.8) | $3 / 86$ | 3.5) 0.062 |  |
| ... (>45) | 16/50 | (32.0) | $15 / 58$ | (25.9) | 9/94 | 9.6) 0.002 |  |
| Joint pain ( $\leqslant 45$ ) | 5.55 | (9.1) | 2.61 | (3.3) | 41866 | .7) 0.353 |  |
| ( $>45$ ) | $19 / 50$ | (38.0) | $10 / 58$ | (17.2) | $14 / 94$ | (14.9) | 0.003 |
| Psychovegetative index ( $<45$ ) | 1.54 | (1.9) | 1/60 | (1.7) | 1/84 | (1.2) | 0.947 |
| (>45) | 12/50 | (24.0) | 5/55 | (9.1) | $5 / 90$ | (5.6) | 0.004 |
| Disturbances of concentration (<45) | 1/55 | (1.8) | $2 / 61$ | (3.3) | $2 / 86$ | (2.3) | 0.874 |
| ( $>45$ ) | $7 / 50$ | 14.0) | 1/57 | (1.8) | 3794 | (3.2) | 0.009 |
| General weakness, tiredness (<45) | 10/54 | 18.5) | 9/61 | (14.3) | 3186 | (3.5) | 0.01 |
| (>45) | 13/50( | 26.0) | 758 | (12.1) | 8194 | (8.5) | 0.014 |
| Constipation (<45) | $2 / 55$ | (3.6) | 5.61 | (8.2) | $2 / 86$ | (2.3) | 0.222 |
| ... (>46) | $6 / 50$ | (12.0) | 3158 | (5.2) | 1/94 | (1.1) | 0.016 |
| Diarhora (<45) | $1 / 55$ | (1.8) | $1 / 61$ | (1.6) | $2 / 86$ | (2.3) | 0.952 |
| ... (>45) | $6 / 50$ | (12.0) | $3 / 58$ | (5.2) | $1 / 94$ | (1.1) | 0.016 |
| Stomach trouble (<45) | 3/55 | (5.5) | 2161 | (3.3) | $4 / 86$ | (4.7) | 0.845 |
| ... (>45) | 7150 | (14.0) | $5 / 58$ | (8.6) | $3 / 94$ | (3.2) | 0.057 |
| Lower back pain (<45) | $5 / 55$ | (9.1) | 9/61 | (14.8) | $11 / 86$ | (12.8) | 0.644 |
| $\ldots$.. $>45$ ) | 15/50 | (30.0) | $12 / 58$ | (20.7) | 14/94 | (14.9) | 0.100 |
| Feelings of excitement on body (<45) | 3/55 | (5.5) | $3 / 61$ | (4.9) | 0185 | (0.0) | 0.034 |
| ... (>45) | $5 / 50$ | (10.0) | $4 / 58$ | (6.9) | 2194 | (2.1) | 0.102 |
| Irregular heartbeat (<45) | $2 / 22$ | (3.7) | 0.61 | (0.0) | $0 / 86$ | (0.0) | 0.067 |
| .... $>45$ | 0.50 | 0.01 | 0/58 | (0.0) | $3 / 93$ | (3,2) | 0.170 |

The insomnia is related to a disturbance of nocturnal melatonin and leads to a general debilitation and lack of mental alertness. This is therefore a very important symptom which warranted extra study. Follow up studies of people's melatonin detected no significant change. However readings were taken after people awoke in the morning and so they did not and would peak which is reached about 2 hours after falling asleep. This problem, and several others, is acknowledged by the authors.

### 8.3.4.4 Unexpected and unaware transmitter breakdown:

A fortuitous event is very revealing. For three days during the study, the transmitter broke down and emissions ceased, and the people did not know about it. During these three days reported sleep quality was markedly improved from the first night in the high exposure group and from the second night in the middle and low exposure group. Averaging over 3 day intervals for each zone, for before, during and after, starting on day 2 gives the following percentage awakenings, Table 12.
"Before" shows the level of awakenings increasing with exposure, "After" shows a rate of recovery which is slow and smallest in the high exposure group and is quick and highest in the Low exposure group.

| Table12: Sleep disturbance rates (\%) for |
| :--- |
| before, during and after the transmitter went |
| off, the brackets show the $\%$ difference from |
| the previous 3-day period. |
| Exposure Before During After <br> Zone A (High) 70 $64(-6)$ $61(-3)$ <br> Zone B (Middle) 68 $52(-16)$ $63(+11)$ <br> Zone C (Low) 61 $37(-24)$ $54(+17)$ |


#### Abstract

"During" shows a significant reduction in awakenings in Zones B and C, which is greatest and most significant in the Low exposure group, and they show the fastest recovery. It appears that the High exposure group takes many days to react to the shut down. This is fully consistent with the study on ELF exposed rats by Wilson et al. (1986) whose melatonin dropped slowing over a period of weeks.

Hence the High exposure group, which is shown to be most strongly affected by sleep difficulties, is very slow in their reaction time, which might limit the level of relief shown by a three day shut down. The Low exposure group gets the greatest relief and the fastest reactions. This strongly suggests that even in Zone C the effects of the transmissions on sleep quality is significant. Hence the Odds Ratios of "exposed" to "unexposed" are significantly under estimated since the group which is assumed to be unexposed clearly is reacting to the exposure they are getting, even though it is in the range 0.00046 $0.0074 \mu \mathrm{~W} / \mathrm{cm}^{2}$.


### 8.3.4.5 Nocturnal Melatonin measured in cows:

Mean measurements of bovine salival melatonin showed that exposed cows had lower mean melatonin, $17.7 \mathrm{pg} / \mathrm{ml}$ compared to $19.0 \mathrm{pg} / \mathrm{ml}$, but this was not statistically significant. It must again be pointed out that it is not the mean melatonin levels which relates to sleep but it is the nocturnal melatonin peak.

The Bovine melatonin study shows that when the transmission was off the cows in the exposed group had strong nocturnal peaks not seen as that pronounced and high at any time when the transmission was on. Since it is the nocturnal peak and not the daily mean nor daytime levels of melatonin which are implicated in sleep and other probable health effects, this data is important ion showing that the noturnal peak in the exposed group is constantly higher than the unexposed group, while the transmitter is off but it is frequently, and on average, lower when the transmitter is on.

Stark et al. (1997) report further analysis of this bovine data stating that while there was a statistically significant 21 -fold higher exposure on the exposed farm compared to the control farm, the lack of statistical significant of the reduce levels of salivary melatonin between the groups suggested that a chronic melatonin effect was unlikely. Figure 11 illustrates one of the fundamentals of melatonin production and that is great variability. The short observational period is also significant in the result. If the reduction between the exposed group compared to the unexposed group had been evident in observations continued for another 10 days then the result is likely to have reached statistical significance.

The authors actual did find a significant observation: "However, on the first night of reexposure after the transmitter had been off for 3 days, the difference in salviary melatonin concentration between the two farms ( $3.89 \mathrm{pg} / \mathrm{ml}, \mathrm{Cl}: 2.04-7.41$ ) was statistically significant, indicating a two- to seven-fold increase in melatonin concentration. This a delayed acute effect effect of EMF on melatonin cannot be completely excluded."


Figure 11: Melatonin concentration measured in the saliva of EMF-exposed (continuous line) and unexposed (dashed line) cows: acute effect of EMF exposure. The lines at $100 \mathrm{pg} / \mathrm{ml}$ indicate the dark phases when melatonin concentration was measured, black box is the period of no EMF exposure of both groups. (Altpeter et al. (1995))

The rate of response and the possiblity of a degree of adaption should be considered when interpreting this data. The human sleep alteration data indicate this, with the lowest and lowest response in the highly exposed group and the greated and quickest response from the lowest exposure group, Table 12.

These combined observations point strongly to the effect of the RF EMR in decreasing the nocturnal peak of melatonin with an accompanying significant degradation in the quality and maintenance of sleep.

There appears to be little reason to assume that human nocturnal peak melatonin levels were not affected in the same way that the bovine melatonin levels were. Sleep disturbance was a highly reported effect, nocturnal peak melatonin relates to sleep quality, bovine exposed melatonin levels were reduced, bovine nocturnal peak melatonin levels improved when the transmitter was off, sleep improved when the transmitter was off. Hence human nocturnal melatonin reduction this is a reasonable and likely
mechanism to explain the reported sleep disturbance and the Chronic Fatigue Syndrome condition, as projected by Reiter (1994), Section 4.1.

Reduced nocturnal melatonin also has significant health implications relating to cancers, such as breast cancer, which has been related to melatonin reduction in laboratory studies, Liburdy et al. (1993) and related to ELF exposure in epidemiological studies, Demers et al (1991) and Section 3.1. This study implicates RF exposure.

### 8.3.4.6 Heart and Blood Pressure:

Concerns about heart problems were investigated with a blood pressure and hypertension survey. Normal blood pressure was reported in $55 \%$ in Zone A, $56 \%$ in Zone B and 74 \% in Zone C. Arterial hypertension was reported in $14 \%$ in Zone A, $8.4 \%$ in Zone B and $7.9 \%$ in Zone C. An extensive review of reporting and measurements revealed that there was an Odds ratio of 1.4 (CI: $0.75-2.52$ ) for blood pressure difference in Zone A compared with Zones B and C combined. Hence blood pressure was slightly elevated with exposure to EMR, but not significantly so.

### 8.3.4.7 Effect of school children:

Sleep and concentration disruption also relates to the performance of school children. Children from a school in Zone A were compared with children from a school which was unexposed, in terms of rate of promotion from primary to secondary school. They found a significantly slower promotion rate in the exposed school. They conclude that even though the association is weakened by a small sample size, "an adverse effect (from the transmitter) cannot be excluded." (p130)

The children in Zone $A$ are in the range of exposures ( $0.031-9.06 \mu \mathrm{~W} / \mathrm{cm}^{2}$ ) which Von Klitzing (1995) shows that significant changes in human EEG occur in $70 \%$ of subjects $\left(0.7 \mu \mathrm{~W} / \mathrm{cm}^{2}\right)$. Reduced nocturnal melatonin and sleep disturbance would also be contributory factors. Mann and Roschke (1996) found that sleeping near to a cell phone caused an EEG monitored adverse change in REM sleep, accompanied by learning and memory problems. Hence a slowing of promotion was found in the Swiss study and scientifically identified mechanisms also exist to reinforce this observations link to EMR. Learning difficulties associated with RF/MW exposure was also found by Latvian researchers, Kolodynski and Kolodynska (1996), and Chinese researchers, Chiang et al. (1989).

Hence very strong evidence exists, with identified mechanisms involving noctural melatonin reduction, for RF/MW radiation significantly reducing the intellectual and memory functions of children and adults, at exposure levels which correspond to those found within many hundreds of metres of cell sites.

## 9. Alterations in brain chemistry, CNS and EEG:

The brain and its accompanying organs such as the pineal body, the pituitary gland and the hypothalmus play fundamental roles in human mood, sleep, immune system, health and well-being. Considerable data exists to suggest that EMR acts in a similar way to sunlight in altering the hormone relationships between the pineal and the pituitary. Visible light exposure modulates the pituitary and pineal gland which leads to neuroendocrine changes, Roberts (1995). Melatonin, norepinephrine and acetylchorine decrease with
light activation while cortisol, serotonin, GABA and dopamine increase. All of these neuroendocrine changes can lead to alterations in mood, circadian rhythm and immune modulation, Roberts (1995).

### 9.1 Calcium ion balance in brain cells:

One of the most repeated effects of ELF modulated RF/MW is the calcium ion efflux from brain cells and muscle cells. Bawin et al. (1976) summarise some of the effects known up to that time.

- Weak extracellular voltage gradients ( $1-5 \mathrm{mV} / \mathrm{mm}$ ) have been shown to significantly affect the excitability or firing thresholds of the spinal neurons of cats.
- Nelson (1966) pointed out that the complex structural organization of brain tissues, as seen in the cerebrum, should be highly favourable for multiple electric field interactions, both in the intricate rate of overiapping dendritic trees and between macromolecules on the extracellular space and the glycoprotiens of the out cell membrane.
- Weak pulsed electric currents ( $2-5 \mathrm{mV} / \mathrm{mm}, 200$ pulses $/ \mathrm{sec}$ ) applied across the cat cortex were able to trigger the release of previously bound radioactive calcium $\left({ }^{45} \mathrm{Ca}^{2+}\right.$ ).
- Intercranial injection of $\mathrm{Ca} 2+$ or $\mathrm{Mg} 2+$ in chronically implanted neonatal chicks resulted in an almost immediate synchronization of the hyperstriatal EEG, accompanied by behavioural depression, Bawin et al. (1984). During successive testing days, the animals appeared to recover behaviourally but never showed any sustained EEG arousal. By contrast animals treated with sodium chloride recovered completely within the first hour after injection.

Because of the high sensitivity of the chick forebrain to small perturbations of the extracellular divalent cations, this was chosen for investigating in vitro, possible interactions of weak voltage gradients induced by VHF radiation and ionic movements in cerebral tissue. The experiment showed that weak VHF fields ( $147 \mathrm{MHz}, 1 \mathrm{~mW} / \mathrm{cm}^{2}$ ), amplitude modulated at brain wave frequencies ( 6 Hz and 16 Hz ) are able to increase the calcium efflux from the isolated brain of the neonatal chick. This result has been repeated by totally independent laboratories, and extended to a wide range of modulation frequencies up to 510 Hz , Blackman et al. (1988), and down to extremely low exposures. These include $10 \mu \mathrm{~W} / \mathrm{cm}^{2}$ (SAR $=0.0075 \mathrm{~W} / \mathrm{kg}$ ), Shandala et al.(1979) and an SAR of $0.00015 \mathrm{~W} / \mathrm{kg}$ ( $S=0.5 \mu \mathrm{~W} / \mathrm{cm}^{2}$ ), Schwartz et al. (1990).

Hence Calcium ion efflux is shown to alter mammal EEG and behaviour.
Professor Adey, and others, have been able to show that imposed oscillating electromagnetic fields can produce significant and repeatable changes in the behaviour of advanced mammals (cats and monkeys) in the laboratory, Adey et al. (1979). They used 450 MHz microwave signal at $0.8 \mathrm{~mW} / \mathrm{cm}^{2}$, modulated at 10 Hz , which produced an EEG level voltage gradient in the cat's brain of $0.1 \mathrm{~V} / \mathrm{cm}$ and no detectable heating.

Wever (1974), section 8 above, showed changes in human subjects isolated from environmental stimuli including ELF fields, which resulted in altered circadian rhythms
which were corrected by applying a $10 \mathrm{~Hz}, 2.5 \mathrm{~V} / \mathrm{m}$ field, which produces about $10^{-7} \mathrm{~V} / \mathrm{cm}$ in tissue. The experiment was repeated using birds, with similar results, of lengthened circadian rhythms.
> "RF fields that are sinusoidally amplitude modulated at ELF frequencies produce a wide range of biological interactions. Induced electric gradients can be substantially higher than those produced by simple ELF electric fields, and at levels of $10-100 \mathrm{mV} / \mathrm{cm}$, are the same range as intrinsic oscillations generated biologically, such as the electroencephalogram (EEG).", Adey (1990)

How does the brain cells sense these EMR fields? The cell membrane outer surface is charged and the alpha-helix glycoprotein stands outside ends are highly charged. Calcium and hydrogen ions interact with the strands and its receptors, which is the first and most sensitive transductive coupling in brain tissue.

Many studies have shown significant efflux of calcium ions from cells exposed to ELF modulated RF and ELF fields. Since calcium ions $\left(\mathrm{Ca}^{2+}\right)$ are known to stimulate specific glutamate binding to the synaptic membrane it is of value to determine whether modulated RF/MW alters glutamate binding.

An efflux has been recorded for the amino acid neurotransmitter gamma-aminobutyric acid (GABA), Kolomytkin et al. (1994), in association with microwaves modulated at 16 Hz . This is very significant since GABA and glutamateric synapses make up about $60 \%$ of the CNS and calcium ions appear to hold the key to every aspect of cell-surface transduction, Adey (1979). Kolomytkin et al. (1994) showed that at 915 MHz microwave signal, modulated at 16 Hz , altered the binding of 3 H -glutamate and 3 H -muscimol in rats brains, at power densities below $50 \mu \mathrm{~W} / \mathrm{cm}^{2}$, which are statistically significantly different from controls to below $10 \mu \mathrm{~W} / \mathrm{cm}^{2}$, Figure 12.

Kolomytkin et al. (1994) link these changes to $\mathrm{Ca}^{2+}$ ions which have been shown to stimulate specific glutamate binding to synaptic membranes due to the activation of a calcium-dependent protease and resulting proteolysis (splitting into fragments) of cytoskeletal proteins.

Since it is shown that modulated microwaves increase the glutamate uptake by synaptomes, Kolomytkin et al. pose the question as to whether microwaves directly affect the synaptosomes or does their sensitivity require some other brain system ? They determined that it was the synaptosomes which were sensitive to the microwaves. They then investigated whether it was a simple heating effect. Heating the samples to produce the same mean SAR did not produce the result. Hence they proposed the mechanism of localized microheating at the cell membrane. This membrane heating in the presence of microwaves has been demonstrated now by Liu and Cleary (1996).


Figure 12: Altered the binding of 3H-glutamate and 3H-muscimol in rats brains versus microwave power density ( 915 MHz , modulated at 16 Hz ), Kolomytkin et al. (1994).

Kolomytkin et al. (1994) conclude that:
"Our findings can be directly related to and complement the findings of Frey and Wesler (1990) and Kavakiers and Ossenkopp (1992). frey found that dopamine and opiate systems of the brain were influenced by exposure to low intensity electromagnetic fields. Kavaliers has shown that electromagnetic fields can influence the functioning of multiple endogenous opoid systems and that the effects depend on the modulation of the field. Considering the great importance of GABA and glutamatergic systems in both normal and pathological brain processes, the finding of low intensity microwaves on these receptor systems is of significance."

Dumanskiy and Shandala (1974) and their colleagues reported altered conditioned reflex in rabbits and rats chronically exposed to extremely low levels of VHF and microwave fields. They used either 50 MHz or 2.5 GHz CW fields or $10 \mathrm{GHz} 1 \mu$ s pulses at 1,000 or 20 Hz , with $10-12 \mathrm{~h}$ daily exposure with 50 MHz and 8 h with microwave fields. They found statistically significant effects with field intensities between 1.9 and $2.0 \mu \mathrm{~W} / \mathrm{cm}^{2}$.

In each experiment the animals were irradiated for 120 days, with a 60 day follow-up. For the first 10 days the animals were "somewhat excited" and reacted to the onset of exposure. Thereafter responses to conditioned stimuli has a longer latency, with weaker
responses to positive stimuli and more numerous missed responses, leading to "pathologic stagnation and inertia".

Clifford et al. (1989), in an effort to duplicate research carried out in the Soviet Union. The U.S. group found significantly less $\mathrm{Na}^{+}, \mathrm{K}+$ and ATPase activity in microwave exposed animals compared to sham exposed animals. Both groups found incidences of statitically significant effects in the power spectrum analysis of EEG frequency, but not at the same frequency.

Shandala et al. (1979) found statistically significant changes in the EEG and brain biochemistry of rats and rabbits exposed to 2.375 GHz microwaves at 10,50 and 500 $\mu \mathrm{W} / \mathrm{cm}^{2}$, for 7 hours/day over 30 days. The $10 \mu \mathrm{~W} / \mathrm{cm}^{2}$ and $50 \mu \mathrm{~W} / \mathrm{cm}^{2}$ initially stimulate brain activity, while $500 \mu \mathrm{~W} / \mathrm{cm}^{2}$ causes suppression as seen from the increase in slow, high amplitude $\Delta$-waves. After 1 month of exposure to a power density of $10 \mu \mathrm{~W} / \mathrm{cm}^{2}$ (for $7 \mathrm{hr} /$ day, i.e. averaging $2.9 \mu \mathrm{~W} / \mathrm{cm}^{2}$ ) a reliable ( $p<0.05$ ) increase was observed in the alpha-rhythm in the sensory-motor and visual cortex due to a suppression of the slow EEG components.

These interactions included entrainment of brain EEG rhythms at the same frequencies as the ELF components of the imposed fields, conditioned EEG responses to imposed fields, and modulation of brain and behavioural states, Bawin et al (1973); and in nonnervous tissues, strong effects on cell membrane functions, including modulation of intercellular communication through gap junctions mechanisms, Fletcher et al. (1986), reduction of cell mediated cytolytic immune responses, Lyle et al. (1983), and mediation of intracellular enzymes that are markers of signals arising at cell membranes and couple to the cell interior, Byus et al. $(1984,1988)$.

Vorobyov et al. (1997) studied short-term alterations of EEG in mice exposed to ELF fields carried on a 945 Mhz microwave carrier with exposures in the range 100 to $200 \mu \mathrm{~W} / \mathrm{cm}^{2}$. They found an induced asymmetry in the EEG on each side of the brain of an ongoing EEG power decrease in the $1.5-3 \mathrm{~Hz}$ range in the left hemisphere and a power increase in the $10-14 \mathrm{~Hz}$ and $20-30 \mathrm{~Hz}$ ranges in the right hemisphere. Significant elevations of EEG asymmetry in the $10-14 \mathrm{~Hz}$ range were observed after the first 20 s after five onsets of the microwave field, when averaged spectra were obtained for every 10 s . In their conclusions they comment that :
"One of the possible key links in this effect can be calcium jon exchange in brain tissue (Adey (1981)). Indeed it was found that the intracellular calmodulin level was changed by modualted microwave fields, Katkov et al. (1992), This change, as is known, can cause the change in receptor sensitiveity to mediators, because in neural tissues both the transmitterreceptor mechanism and the second messenger are $\mathrm{Ca}^{2+}$ dependent."

### 9.2 EMR induced EEG changes in humans:

Are these effects found in humans?
Two papers known to the author show EMR alteration of the human EEG. The first, von Klitzing (1995) shows dominantly EEG delta to alpha rhythm change when exposed to GSM signal. The second shows sleep and EEG change with GSM phone exposure.

### 9.2.1 Human EEG delta to alpha when GSM exposed:

Von Klitzing (1995) shows the same result, alpha enhancement and slow wave suppression in human subjects exposed to a GSM cell-phone like signal with an SAR of $0.001 \mathrm{~W} / \mathrm{kg}\left(\mathrm{S}=0.7 \mu \mathrm{~W} / \mathrm{cm}^{2}\right)$, (from Eq. 8 using $\sigma=0.77 \mathrm{~S} / \mathrm{m}$ ) and a pulse frequency of 217 Hz . The power spectrum of one of the subjects is shown in Figure 13. Von Klitzing's paper presents an example of the 45 experiments from 17 students tested. Around $70 \%$ of the students showed significant alteration in their EEG at these very low exposure levels.

The human subjects react much more quickly than the rat and rabbit subjects. Not all human being show this sensitivity. The author underwent the exposure and EEG test and showed no significant difference between the exposure and unexposed periods. He therefore joins the $30 \%$ who show no effects.

### 9.2.2 Cell phone signal alters sleep EEG:

Healthy people sleeping with a digital GSM cell phone on next to the bed, exposing their heads to about $50 \mu \mathrm{~W} / \mathrm{cm}^{2}$ while their brain EEG was being monitored, Mann and Roschke (1996). This revealed a statistically significant distruption of alpha EEG frequency range and REM sleep. REM sleep decreased from $17.07 \%$ to $13.91 \%$, which is significant at $p<0.05$. In addition subjects went to sleep faster, a hypnotic effect also reported by Reite et al. (1994) who used a signal of 27.12 MHz modulated at 42.7 Hz .

Mann and Roschke (1996) exposed 14 healthy, non-smoking, non-drinking, 21-34 year old male volunteers to 900 MHz , pulsed at 217 Hz with a pulse width of $580 \mu \mathrm{~s}$, digital GSM signal with a resultant average power density at the head of $50 \mu \mathrm{~W} / \mathrm{cm}^{2}$. They concluded that:
"Besides a hypnotic effect with shortening of sleep onset latency, a REM suppressive effect with reduction of duration and percentage of REM sleep was found. Moreover, spectral analysis revealed quantitative alterations of the EEG signal during REM sleep with an increased spectral power density. Knowing the relevance of REM sleep for adequate information processing in the brain, especially concerning the mnestic functions [Memory functions] and learning processes, the results emphasize the necessity to carry out further investigations on the action of this type of electromagnetic fields and the human organism."

The results are summarized in Figure 14.


Figure 13: Human Alpha-EEG (O2-position) is altered by pulsed electromagnetic fields following first exposure, von Klitzing (1995).


Figure 14: Mean power density (dB) of the averages of different sleep stages in 12 subjects averaged, left columns without field and right columns with GSM digital field of $50 \mu \mathrm{~W} / \mathrm{cm}^{2}$.

Reite et al. (1994) also found an hypnotic effect when a 27.12 MHz signal, modulated at 42.7 Hz as applied over a 15 min period. Exposed subjects reached a deeper state of sleep than sham exposed subjects.

The GSM exposed subjects also reported having fewer "bad dreams". This is consistent with reduced melatonin. Post sleep subjective surveys found non-significant changes with GSM exposure such as reduced sleep quality, number of wakings. Post waking increased calmness and alterness, along side decreased concentration and increased anxiety. These latter two are frequenty associated with increased daytime serotonin. The authors relate REM sleep impairment to memory and learning processes. Recently large numbers of cell phone users have been reporting headache, loss of concentration and memory impairment. This is consistent with these results.

### 9.3 Studies showing learning difficulties with EMR exposure:

Sound REM sleep is necessary for learning, memory and wellbeing. Any studies associating learning difficulties with EMR exposure would strengthen this association and the evidence of likely melatonin reduction and sleep disruption.

Three published papers or reports identify such effects:

- Chiang et al. (1989) found that visual reaction time, a measure of the function of the visual receptor and the central nervous system, varied with microwave exposure of children up to $4 \mu \mathrm{~W} / \mathrm{cm}^{2}$. Children exposed to over $10 \mu \mathrm{~W} / \mathrm{cm}^{2}$ had lower scores in the memory function test. They concluded "the data indicate that chronic exposure to EMFs are associated with significant changes in some physiological parameters."
- Altpeter et al. (1995) showed a statistically significant delay in promotion from primary to secondary school in the more highly exposed school compared to a low exposure school, $\mathrm{OR}=0.63,95 \% \mathrm{Cl}: 0.43-0.85, \mathrm{p}<0.005$. This involved shortwave radio exposure. The daily mean exposures in the highly exposed group were in the range 0.031 to $9.1 \mu \mathrm{~W} / \mathrm{cm}^{2}$, median $0.1 \mu \mathrm{~W} / \mathrm{cm}^{2}$ and mean $0.24 \mu \mathrm{~W} / \mathrm{cm}^{2}$.
- Kolodynski and Kolodynska (1996) investigated the effects of a RLS radar in Latvia, radiating at $154-162 \mathrm{MHz}$ and pulsed at 24.4 Hz , on the performance of school children living several km in front of the radar compared to children living behind the radar. They concluded that "Motor function, memory and attention significantly differed between exposed and control groups, children living in front of the RLS had less developed memory and attention, their reaction time was slower and their neuromuscular apparatus endurance was decreased." Assuming that the closest child lived 2 km in front of the radar, the maximum mean measured exposure is in the $0.16 \mu W / \mathrm{cm}^{2}$.

Hence there is evidence from a wide range of RF/MW frequencies, at public exposure levels of around $0.1 \mu \mathrm{~W} / \mathrm{cm}^{2}$ and less, of learning, memory, sleep and physical performance of children; sleep distruption, aches, pains and chronic fatigue in adults. All of these symptoms are consistent with the hypothesis that RF/MW reduces nocturnal melatonin with consequent psychological and physical impairment.

### 9.4 The relationship between EEG brain states and moods:

Typical electroencephalographs (EEG) are given below, from Dorland 28, p535. As noted above, cells have electric charges on their surfaces. By placing sensor electrodes on the surface of the skull electrical signals are detected. Voltages are detected which have been produces by currents emanating from nerve cells in the brain. The dominant frequency of these signals in about 8 to 10 Hz with an amplitude of 10 to $100 \mu \mathrm{~V}$.

EEGs are characterized by frequency bands which are associated with various brain states, Dorland 28:

## Alpha Rhythm: $8-13 \mathrm{~Hz}$

Are typical of the normal person awake and in a quite resting state, and principally in the occipital region. Alpha amplitude increases with joy and anger and decreases with fear and sorrow.

Beta Rhythm: $18-30 \mathrm{~Hz}$.
Are typical during periods of intense activity of the nervous system, occurring principally in the parietal and frontal regions.

Delta Rhythm: $<3.5 \mathrm{~Hz}$.

Typically occurs in deep sleep, in infancy and in serious brain disorders.

## Theta Rhythm: $4-7 \mathrm{~Hz}$.

Occurs mainly in children but also in adults during periods of emotional stress.


Figure 15: EEG recordings made while the subject was excited, relaxed and in various stages of sleep. During excitement the brain waves are rapid and small amplitude, whereas in sleep they are much slower and of greater amplitude.

Adey (1979) notes that the electric process between dendrites (in the brain) is one of slow waves, not pulses. The integral of the slow wave activity of the dendrites constitutes the electroencephalogram (EEG). When the brain is awake the electro-encephalogram is fast, as are the waves outside the cell.

In classical axodendritic synapse, Figure 16, (a) synaptic vesicles in the axon of one neuron release neurotransmitter toward the receptors on the dendrite of a target neuron. It is also possible for a dendrite to pass a message to another dendrite by way of dendrodenritic synapses. In a reciprocal dendrodenritic synapses (b) each dendrite passes messages to the other by way of a separate synapse. In some synapses, called axoaxonic synapses (c), the axon of one neuron passes a message through the axon of another neuron to the dendrite of a third neuron. In synaptic glomerulus (d) the axon of one neuron passes messages to dendrites of two others; the dendrites may pass messages to each other as well. Snyder (1985) [p138]

The electroencephalogram is produced by the leakage of these big waves from inside the dendrites into the fluid around the cell. The electroencephalogram recorded over the dimensions of the cell is a few microvolts. The neuronal wave inside the cell is of the order of 5 to 15 mV . Thus the difference in amplitudes is about 200 to 1 .


Figure 16: Communication between neurons takes place across gaps called synapses.
It is very evident that brain activity changes a great deal with rest and activity, with health and illness, and with stress and emotion. During these wide ranging changes, significant changes in neurotransmitters such as serotonin and adrenaline have also been monitored. These biochemical changes send neurohormones throughout the body to change heart beat, vasodilation etc.. They also change the brain cell behaviour in such a way that electrical signals in regions of the brain show altered, coordinated and repeatable changes in oscillating voltages which are indicative of coordinated electrical communication between large groups of brain cells.

A key scientific question is: is the EEG simply a product of the changing electrical environment within the brain, or does it provide the oppoitunity for external oscillating electrical fields to superimpose changes in the electrical behaviour of the brain which would then produce imposed changes in neurotransmitters and neurohormone production? Is the brain, or parts of the brain, sensors which can pick up external
electrical signals which can change the psychological and/or physiological state of the brain and body?

### 9.5 Conclusions - EEG and EMR:

These recent studies show unequivocal evidence that low level modulated and pulsed RF/MW signals, including GSM digital signals, alter the human EEG and affect the state of sleep in ways which interfere with information processing and learning. This confirms a neurological basis for the observed impairment of children's learning in Switzerland, Latvia and China.

Hence, far from being an isolated example, as this data was considered by the Planning Tribunal, the von Klitzing results for human beings is consistent with research on people and rabbits, Dumanskiy and Shandala (1974) and Shandala et al. (1979); and in cats, Bawin et al. (1973). Studies on altered reaction times and circadian rhythms in humans and animals are linked to EEG changes, Adey (1981).

Adey (1991) goes a considerable way towards describing the mechanisms which underlie these changes in the brains of higher animals, including people. Dendridic cells in the brain, high levels of entrainment of ELF signals from RF/MW ELF modulated radiation, associated with changes in calcium ion concentrations and altered release and binding of neurohormones and neurotransmitters, such as GABA, serotonin and melatonin, have all been described and linked to EMR exposure.

Hence Dr von Klitzing's results are consistent with animal experiments, have a basis in Neurophysiology and therefore stand as a serious concern about the impact of very low intensity modulated RF/MW signals on the fundamental processing of information by our brains. The intensities of exposure which show effects are all non-thermal and reach levels well below the current public exposure standard ( $200 \mu \mathrm{~W} / \mathrm{cm}^{2}$ ), at 50 , about 1 and $0.7 \mu \mathrm{~W} / \mathrm{cm}^{2}$. With isolation from natural environment's $0.3 \mathrm{pW} / \mathrm{cm}^{2}$ Schumann Oscillations also having measurable effects of circadian rhythms.

## 10. Cellular Biology:

### 10.1 Introduction:

The fundamental basis of biologic activity is the cell. Cell biology and biochemistry has advanced our understanding of cell behaviour and cellular processes to highly advanced levels. The structure of cells is well described, the processes which regulate cell growth and development, the genetic basis of cell reproduction and the amino acid typing of complex molecules, including RNA and DNA is being advanced daily. This shows the importance of understanding and appreciating cellular characteristics and processes in order to understand the interactions of EMR with living tissue and potential health hazards from EMR exposure.

In cellular aggregates that form tissues of higher animals, cells are separated by narrow fluid channels that take on a special importance in signaling from cell to cell. These channels act as windows on the electrochemical world surrounding each cell. Hormones, antibodies, neurotransmitters and chemical cancer promoters, for example, move along then to reach binding cells on the cell membrane receptors.

### 10.2 Biochemistry and cell biology:

During the MacIntyre Case, Associate Professor Richard Luben presented evidence of biochemical mechanisms which are observed to change under exposure to modulate RF fields, Luben (1995). This included changes in calcium ion efflux and Ornithine Decarboxylase (ODC), both of which are involved in the signal transduction aspects of control of the growth and development of cells in the human body and other animals.

Two of Dr Luben's key statements are worth recalling. He stated that laboratory studies had shown the similarities and parallels in the biological effects of ELF and RF modulated by ELFs. For example, calcium ion efflux and ODC are observed to vary in similar ways in ELF fields and in RF fields modulated at ELF frequencies, Byus (1994), Giuliana, et al. (1996).

Dr Luben also stated that the electromagnetic radiation did not need to enter the cell in order to change its behaviour, it just needs to be absorbed by the cell surface, and then the altered signal transduction process changes the cell behaviour.

A more detailed and updated review of biophysics and biochemistry reveals clear means of EMR altering the biochemical behaviour at the cellular level with a great deal of detailed existing understanding about these processes. These form a set of plausible mechanisms to explain the way in which EMR can change cell behaviour and hence can cause adverse health effects. Where these are matched by appropriate epidemiology an extremely strong association is established. Where there is a pattern of epidemiology which is consistent with animal experiments and for which there are detailed biophysical and biochemical mechanisms, the evidence approaches the level required to establish cause and effect.

The research here reviews our current understanding of cell structure and processes, including resonant absorption of radiofrequency and microwave radiation at the cell membrane, gap junction communication between cells, signal transduction processes from regulating cell growth and behaviour, the vital role of calcium ions, the implications for changes in ODC, the formation and effects of free radicals, the dendridic structure of the brain which relates to neurotransmitters and EEG, and the role of neurotransmitters such as serotonin and adrenaline, and neurohormones such as melatonin.

Evidence will then be presented showing cellular and molecular changes which occur with exposure to electromagnetic radiation which are directly or indirectly related to known biophysical biochemical characteristics of cells. The implications of this for public health will be discussed. The identified biophysical and biochemical mechanisms will then be related to epidemiological research and public health implications will be discussed and exposure standards will be recommended where supportable by sound research.

### 10.3 Cell Biochemistry and Neurophysiology

The last 10 to 15 years has seen an exciting and challenging revelation of the complex biochemistry at the cellular and molecular level in living systems. As laboratory techniques have advanced we have progressed from the study of organs, to tissues, to cells and to molecules. The behaviour and organisation of cells and molecules in living tissue relies on sequences of reactions which use and transform energy in the continual creation and re-creation of molecular complexes including material to form cell walls (membrane), cell nuclei, inside to outside cell communications, cell to cell
communications, enzymes to stimulate and slow cell division and cell growth, and the RNA and DNA molecules which code the genetic structure of the host of different types of cell, and cell and tissue structures which make up highly organized living systems.

### 10.4 The Cell:

The cell is an identifiable entity of all living organisms and is recognized as the fundamental unit of biologic activity. Cells consist of a nucleus which is surrounded by cytoplasm, which contains various organelles, and is enclosed in a cell or plasma membrane, Figure 17.

The nucleus contains the hereditary material of DNA and chromosomes, along with the proteins and enzymes which are necessary for the sustenance of the nucleus and the processes of chromosome separation during mitosis.

The cytoplasm is the protoplasm of a cell exclusive of that nucleus. It consists of a continuous aqueous solution (cytosol) and the organelles and inclusions suspended in it. It is the site of most biochemical activities of the cell.

The cell membrane is a bimolecular layer of lipids which encloses the cytoplasm and nucleus. It had is permeable to some substances and contains protein structures which pass through the membrane, providing for processes such as signal transduction, see Figure 13 below.

Cells form many shapes and have a host of different functions. Some cells are bound together to form tissue such as in skin and muscles, some are near spherical and float in fluid, such as T-cells, and others are dendridic, with long dendrite structures extending to several times the diameter of the central cell body, such as many brain and central nervous system cells.

### 10.5 The Cell cycle:

A cell is a cooperative of molecules which is capable of reproducing itself. Cells are discrete entities that grow and divide. Most cells must complete four tasks during the cell cycle. They must grow, replicate their DNA, segregate their chromosomes into two identical sets and divide. To do this a cell needs between 2000 and 5000 different enzymes and structural proteins.


Figure 17: A typical animal cell (Dorland 28, p285)

The cell cycle is divided into four discrete phases. Rapidly dividing human cells have a cell cycle which lasts about 24 hours.

Some of the molecules, like ribosomal proteins and RNAs are present in the millions per cell, while DNA are present as only one or two. Cells contain many different types of proteins, each specialized for a particular role in the life of the cell. Important classes include enzymes that produce the building blocks for the synthesis of DNA, RNA and proteins, and the enzymes which build these blocks to replicate DNA, transcribe DNA into RNA, and translate mRNA into protein. The form and function of cells depend on the structural proteins that form the cytoskeleton and on the motor proteins that move objects along elements of the cytoskeleton, such as chromosomes. Mammals are estimated to have as many as 200 different cell types.

The cell cycle is divided into two fundamental parts: interphase, which occupies the majority of the cell cycle, and mitosis, which lasts about 30 minutes, ending with the division of the cell. During interphase DNA is diffusely distributed throughout the nucleus, and individual chromosomes cannot be distinguished. Little activity can be seen in the microscope but two important classes of process are occurring, continuous processes (referred to collectively as 'growth') and stepwise processes which occur once per cycle.

For example, chromosome replication is restricted to a specific part of interphase called $S$ phase (for DNA synthesis). S phase occurs in the middle of interphase, preceded by a gap called G1 and followed by a gap called G2, Figures 11 a and 11b. After each chromosome has been replicated, the two daughter chromosomes remain attached to each other at multiple points along their length and are referred to as sister chromatids.

In a typical animal cell cycle, G1 lasts 12 hours, S phase 6 hours, G2 6 hours and mitosis (M) about 30 minutes.


Figure 18: (a) Cell growth and DNA content during the cell cycle. Mass increases continuously throughout the cell cycle while DNA content is constant for most of the cycle, increasing during the $S$ phase as DNA replicates, then falling dramatically during chromosome segregation.
(b) Stages of the cell cycle, which in adult vertebrates rapid cell cycles take about 24 hours. Mitosis (M) represents about $5 \%$ of the cycle, and there is a substantial gap (G1) between mitosis and DNA synthesis (S), as well as a gap ( $G 2$ ) between replication and mitosis.

Source: The Cell Cycle, by Murray and Hunt (1993).

The interaction mechanisms of radiation and some chemicals which have strong adverse health effects occur through alteration or interference with the cell cycle. X-ray radiation is known to damage cells and their DNA, while caffeine is known to accelerate mitosis. Rowley (1990) reported on studies into the repair of radiation-induced chromatid aberrations: relationship to G 2 arrest in CHO cells. The literature suggests that the function of radiation-induced G2 arrest is to allow repair of potentially lethal damage before cell-entry into, and damage expression in, mitosis. The nature of the damage repaired is not known, but chromosome aberrations have been considered.

To examine this possibility in G2 cells, Rowley (1990) compared the rate of repair of chromatid aberrations in CHO cells progressing to or arrested in G2, with the rate of repair of the damage which gives rise to G 2 arrest. To measure aberration repair rates, exponentially growing CHO cells arrested in G2 with $1.5,2.5$ or 3.5 Gy of X -rays were released into mitosis by treatment with 5 mM caffeine immediately or 1,2 or 3 h after irradiation. Aberration frequencies in these cells were then related to the caffeine-free (repair) interval. To measure the rate of repair of arrest-causing damage a split-dose procedure was used. The half-times for aberration repair were approximately 1 h for
achromatic gaps and 1.5 h for breaks, intrachanges and interchanges. The half-time for arrest damage repair varied with radiation dose. This result suggests that chromatid aberrations are not a primary cause of radiation-induced G2 arrest.

While Rowley (1990) has shown arrest during G2 of the cell cycle under X-ray irradiation, DeFrank et al (1996) show that UV radiation arrests the cell cycle in G1, slowing the transit of cells into the S-phase, which is reduced by the application of caffeine. DeFrank et al. investigate the role of the p53 tumour suppresser protein whose function is inactivated in malignant cells. They find that p53-null cells are more sensitive to UV light, only in the presence of caffeine, implicating caffeine in processes which reduce cell repair and enhance cell damage under UV exposure.

The role of caffeine in accelerating mitosis before DNA repair can take place implicates caffeine in enhancing chromosome aberrations. This shows the quite complex interactions of diet, environmental exposure and other factors such as familial genetics, in the susceptibility of people to adverse health effects.

### 10.6 EMR alteration of the cell cycle time:

Brulfert et al. (1985) studied the growth of plant roots in 2 day exposure to a strong ( 430 $\mathrm{V} / \mathrm{m}$ ) ELF ( 60 Hz ) electric field in vivo. They found that exposed roots were shorter because cell elongation was reduced in exposed roots compared to controls. Heller and Teixeira-Pinto (1959) showed that a strong pulsed RF (27 MHz) field caused chromosome breaks which probably occurs in the replication of DNA in the S-phase. These pose the question as to whether animal cells are similarly affected.

Levin and Ernst (1995) report that 60 Hz fields (3.4-8.8 mT) and magnetic fields over the range $\mathrm{DC}-600 \mathrm{kHz}(2.5-6.5 \mathrm{mT}$ ) can alter the early embryonic sea urchin embryos by inducing alterations in the timing of the cell cycle. Their results, as for the cellular studies above, were dose-dependent and biphasic as a function of frequency, duration and timing of the exposure. Low frequencies advanced mitosis and higher frequencies delayed mitosis. Stein and Lian (1992) point out the importance of cell cycle perturbations since the loss of growth control in transformed and tumour cells is accompanied by an abrogation of developmental regulatory mechanisms that are functionally coupled to proliferation.

Do the differences of these sea urchin cells and human cells mean that people will not experience alteration of their cell cycle in exposure to EMR ?

Conti et al. (1983) investigated the effects of extremely low frequency EMR on immature human peripheral blood lymphocytes which were also exposed to substances which participate in the mitosis of the cells. They found that a frequency window ( $3-50 \mathrm{~Hz}$ ) significantly inhibited the conA-induced blastogenesis, while the pokeweed mitogen (PWM) was significantly affected only at 3 Hz . Conti et al. explored the mechanisms which EMR might have and excluded a direct effect on thymidine incorporation. They focus on the flux of calcium ions. A reduction of calcium ions upon exposure to EMR (through the outward flow through the cell membrane - calcium ion efflux). The effect on lymphocytes of calcium loss represents a decrease in the rate of DNA synthesis in all cells and/or a reduction in the number of cells undergoing DNA replication.

Hence they conclude: " $\mathrm{Ca}^{2+}$ ions are involved in the control of lymphocyte proliferation. In fact, mitogenic lectins produce a rapid, initial calcium influx and calcium is required for

DNA synthesis some 18-72 h after the mitogenic stimulus." Considering the theoretical (and observed) effects of EMR on cellular efflux of calcium ions "we think that an alteration of calcium fluxes by EMF may be the most realistic hypothesis to explain the observed inhibitory effect on human lymphocyte blasticgenesis."

Human lymphocytes are the primary agents in the immune system, in the form of T cells, B-cells and NK-cells (natural killer). The calcium ion efflux is now well documented to increase with ELF modulated RF signals and hence these signals, at levels down to SAR of $0.00015 \mathrm{~W} / \mathrm{kg}$, Schwartz et al. (1988) have the effect of reducing the protection to infection offered by the immune system cells (white blood cells), Walleczek (1992). This corresponds to an energy flux of about $0.04 \mu \mathrm{~W} / \mathrm{cm}^{2}$ for isolated frog hearts or about $0.4 \mu \mathrm{~W} / \mathrm{cm}^{2}$ for a human body.

Professor Stephen Cleary's group has been studying the effects of cell cycle changes when exposed to RF ( 27 MHz ) and MW ( 2.45 GHz ) radiation. Clearly et al. (1990a) exposed human blood to these EMR frequencies under isothermal conditions ( $37 \pm 0.2^{\circ} \mathrm{C}$ ) for 2 h and observed a statistically significant biphasic, dose-response dependent effects of the radiation on human lymphocyte proliferation, both with and without a mitogenic stimulation.

Cleary et al. (1990b) carried out a similar exposure experiment on human glioma cells. They found alterations of the cell proliferation which were not caused by RF-induced cell heating. The dose-response for both frequencies was biphasic. Lower exposures enhanced proliferation while exposures over $50 \mathrm{~W} / \mathrm{kg}$ suppressed DNA and RNA synthesis. Statistically significant time-dependent alterations were detected up to 5 days postexposure, suggesting a kinetic cellular response to RF radiation and the possibility of cumulative effects of cell proliferation. Cleary et al. (1992) concluded that there is direct, nonthermal cellular effects of RF radiation which included effects on the mitotic cell cycle but no mechanisms had been identified.

Cleary et al. (1996) exposed Chinese hamster ovary ( CHO ) cells to within $37+/-0.1^{\circ} \mathrm{C}$, to 5 W/kg and $25 \mathrm{~W} / \mathrm{kg}$ signals of 27 MHz and 2.45 GHz radiation. They studied the effects at each phase of the cell cycle, including DNA distributions. They found that a 2 hr exposure induced significant time-dependent cell cycle alterations for up to about 4 days. These effects were generally reversible over 96 hours and were twice as great for the 2.45 GHz microwave signal as they were for the 27 MHz radiofrequency signal. They considered this to be a real effect of relatively low magnitude and in "agreement with predictions of a theoretical analysis", referring to Liu and Cleary 1995.

### 10.7 Cell Structure and EMR Alterations Conclusion:

RF and MW electromagnetic radiation has frequency and intensity effects on the alteration of cell cycles which are not heat induced effects. Highly probable mechanism involves the change in cell cycle with effects on DNA efficacy and altered membrane permeability to key agents. For example, the efflux of calcium ions which alters the synthesis of DNA and other aspects of the cell cycle in plant, animal and human cells, including cells of the central nervous system, immune system and cardiac system.

## 11. Cellular Control Factors

### 11.1 Gap-Junction Communication:

Cell-to-cell communication takes place through signals transmitted through the intercellular fluid, and through direct cell-to-cell contact through two apposed epithelial cells made of two hexagonal studs embedded in the membrane layer, called a Gap Junction, Figure 19.

Through this structure, ions, amino acids, sugars, nucleotides and other molecules which are smaller than $20 \AA$ in diameter pass, but proteins, nucleic acids and larger molecules cannot, from Bretscher (1985).


Figure 19: Gap Junction between adjacent cells (Schematic) from Bretscher (1985).

### 11.2 Gap Junction Alteration by EMR:

A 2 mT 50 Hz field induced a $160 \%$ flow of cAMP through gap junctions in a monolayer of mouse fibroblast cells, measured immediately after a 5 minute exposure, Schimmelpfeng et al. (1995). Cyclic AMP is a primary "second messenger" of the cell developmental biochemistry along side calcium ions. Cooper (1995) calculated that millisecond applications of electric fields on the range 0.01 to $0.1 \mathrm{~V} / \mathrm{cm}$ can result in significant hyperpolarizations and depolarizations across the gap junction. Cell-to-cell is a vital biological function. Disruption of the gap junction communication is associated with unregulated cell growth, Adey (1989).

Fletcher et al. (1987) noted that the blockage of the entry of natural cytolytic substances, alpha-lymphotoxin (LT) and recombinant tumour necrosis factor (TNF), into Chinese hamster ovary cells depends on their ability to form gap junctions, a function which varies between different strains of these cells. Fletcher found that the phorbol ester cancer promoter (TPA) opens gap-junctions to permit the entry of LT, leading to cell death (lysis) in a dose-dependent fashion.

Weak RF fields ( $450 \mathrm{MHz}, 1-1.5 \mathrm{~mW} / \mathrm{cm}^{2}$ incident energy) with 16 Hz sinusoidal modulation, enhanced this ability of TPA to impair gap-junction communication. The effect did not occur without modulation.

Oncogenes may also interrupt gap-junction communication. Hence, EMR modifies gapjunction communication in ways which are potentially adverse to the health of tissue, either through cell death or through disrupted growth control which leads to cancer cells.

### 11.3 Extra-cellular environment:

In cellular aggregates that form tissues of higher animals, cells are separated by narrow fluid channels that take on a special importance in signaling from cell to cell. These channels act as windows on the electrochemical world surrounding each cell. Hormones, antibodies, neurotransmitters and chemical cancer promoters, for example, move along them to reach binding sites on cell membrane receptors, Adey (1992a). These narrow fluid "gutters", typically not more than $150 \AA$ wide, are also preferred pathways for intrinsic and environmental electromagnetic (EM) fields since they offer a much lower electrical impedance than cell membranes. Although this intercellular space (ICS) forms only $10 \%$ of the conducting cross section of typical tissue, it carries at least $90 \%$ of any imposed or intrinsic current, directing it along cell membrane surfaces.

### 11.4 Signal Transduction:

The division of labour among the cells of a multicellular organism requires that each cell population be able to call on the services of some cell populations and respond to the requirements of others. Much of this is accomplished with chemical and electrical signals. Yet most of the arriving signals never invade the privacy of the cell. They are picked up on the surface of the cell by molecular antennae called receptors. This initiates the communication into the cell in a process termed "signal transduction".
> "Signal Transduction refers to reactions by which the cell receives and acts upon regulatory information from outside the cell. Information-containing signals may include neural messages, hormones, growth regulatory factors, chemical substances, physical forces, and electromagnetic variables such as heat, light, and internal currents from bones and muscles. Signal Transduction is very specific and sensitive. Only particular cells respond to signals and some signal transduction systems can amplify the incoming signal by many orders of magnitude, for example a single photon of light in the eye can induce the synthesis of millions of molecules of neurotransmitters in the nerves leading from the eye to the brain (Stryer, 1986)."

### 11.5 Signal Transduction structures:

Numerous stranded protein molecules protrude from within the cell into this narrow ICS. Their glycoprotein tips form the glycocalyx which senses the chemical and electrical signals in the surrounding fluid. Their highly negatively charges tips form receptor sites for hormones, antibodies, neurotransmitters and for many metabolic agents, including cancer promoters.

These charged terminals form an anatomical substrate for the first detection of weak electrochemical oscillations in the pericellular fluid, including the field potentials arising

