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Units

As from 1 April 1978 NRPB adopted the International System of Units (SI).

Characterisation of exposure fields

Quantity	Unit
Frequency	hertz (Hz)
Wavelength	metre (m)
Electric field strength	volt per metre (V m^{-1})
Magnetic field strength*	ampere per metre (A m^{-1})
Magnetic flux density*	tesla (T)
Power flux density	watt per square metre (W m^{-2})

*A magnetic field strength of 1 A m^{-1} is equivalent to a magnetic flux density of $4\pi \times 10^{-7}$ T in non-magnetic media.

Dosimetric quantities

Quantity	Unit
Current density	ampere per square metre (A m^{-2})
Specific energy absorption rate (SAR)	watt per kilogram (W kg^{-1})

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Abstract

This review consists of three main parts. In the first the general features of electromagnetic fields and their interactions with the human body are described. It is pointed out that some evidence from biological experiments means that it is not *a priori* possible to conclude that there are no long-term effects on human health from exposure to levels of electric and magnetic fields below the thresholds for electric shock, burn and overheating.

The second part deals with the epidemiological evidence for effects on general health and birth outcome. It is considered that the bulk of the evidence suggests that there are no long-term effects on general health from the levels to which people are normally exposed from power frequencies, radiofrequencies or microwaves. As regards birth outcomes, the average female user of visual display units (VDUs) does not seem to be at risk of adverse birth outcome. There is a little evidence suggesting that intensive use is associated with an increase in spontaneous abortion, but this could be a result of higher job stress and postural problems rather than of exposure to the small electromagnetic fields.

The third part describes the epidemiological evidence from occupational and residential studies of a possible association between electromagnetic field exposures and cancer. Studies of electrical and electronic workers are suggestive of such a link, but are subject to the confounding factor of occupational class. If the association is real, the average excess risk appears to be small. Residential studies in both the USA and Sweden, but not in the UK, are suggestive of a strong link between living near to an electrical installation and childhood cancer. In the most comprehensive of the American studies there are correlations between living near high densities of electric power supply cables and traffic density, and also between traffic density and childhood cancer. As well as this confounding factor, there may also be other social-economic factors that may in part explain the observations. In all studies the association of cancer with domestic magnetic field levels is much weaker than with measures of proximity to electrical installations. While the evidence does not justify an excessive concern about domestic magnetic fields from electrical appliances or power supplies, neither is there any justification for complacency about the possible influence of electromagnetic fields on carcinogenesis. Further research to establish whether there is a real association should be carried out. Recommendations for such research are made in the report.

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1 Introduction

Along with the general increase in life expectancy in western countries in the last 50 years, which is associated with the elimination of major infectious diseases and the higher standards of living represented, for example, by better heating and refrigeration, there has emerged an increased awareness of the possible health hazards arising from many of the industrial processes and products responsible for these higher standards. One area in which this increased awareness has been evident is associated with the employment of electricity for the provision of power, communications, navigation and radiolocation. The use of electricity in these applications of necessity generates electric and magnetic fields. The extent to which the use and propagation of electromagnetic energy has increased in the UK since the early 1930s can be gauged from Table 1. This compares the increase in electric power generation and the transmission of radiofrequencies by the major broadcasting authorities with changes of life expectancy. Table 2 shows the statistics for infant mortality.

Following consultative documents published in 1982 and 1986 (NRPB, 1982, 1986), NRPB in 1989 published guidance on restricting exposures to electromagnetic fields of all frequencies up to 300 GHz (NRPB, 1989). The objective of this guidance was the prevention of acute effects due to electrical shock, burn and overheating. However, there is some evidence for effects from low level electromagnetic fields at all levels of biological organisation (NCRP, 1986; Ahlbom et al, 1987). The evidence for these and the acute effects is summarised in companion NRPB reports (Kowalczuk et al, 1991; Sienkiewicz et al, 1991; Saunders et al, 1991). The levels at which acute effects occur correspond approximately to those at which the induced electric potentials in cells exceed the intrinsic thermal-electrical noise levels. The available evidence suggests that effects are possible at levels which are 100 to 1000 times lower and possible mechanisms have been proposed to explain this evidence. Some of these are outlined in Section 1.2. It is not possible to say with certainty what implications much of this evidence has for human health. The data in Tables 1 and 2 might be taken to suggest that for the majority of the population any harmful effects are outweighed by the incidental benefits and the risks, if any, are rather small. However, this does not prove the absence of possibly high risks to some groups of the population or from certain types of exposure to electromagnetic fields. There are in any case many other factors involved in the improvements in health and life expectancy during the past half-century. It is the purpose of this report to review the available human data for evidence of any long-term risks to health from electromagnetic field exposures.

The risks considered are:

(a) of various categories of non-cancerous ill-health,

(b) of effects on birth outcome,

(c) of the induction or promotion of cancers.

Concerns about general ill-health began to be taken seriously in the late 1960s and early 1970s. In recent years these concerns, which are dealt with in Section 2, together with those about birth outcome, seem to have diminished and have been replaced by increased concern about the possibility of cancer induction and promotion. In order to help put this concern in perspective, some aspects of the general epidemiology of cancer are dealt with in Appendix A. The evidence for specific cancer risks in relation to electromagnetic fields is described in Section 3.

This report is meant for general readership rather than for the technically expert. Some

additional information to help understanding is therefore included below and some statistical and epidemiological terms used are explained in Appendix B.

1.1 Measures of electromagnetic fields

The two fundamental quantities associated with electromagnetic fields are frequency and wavelength. Conventionally, the frequency range is from zero (static) up to 300 gigahertz (GHz), Figure 1. Above 300 GHz the region of optical radiations begins with infrared radiations and progresses with increasing frequency and decreasing wavelength through the visible region to ultraviolet radiations and then to frequencies at which ionising radiations begin.

One way of describing electromagnetic fields is in terms of the radiation from the classical dipole aerial (or antenna) that is used for receiving and transmitting radio and television signals. An aerial of this type is shown in Figure 2. When a generator of alternating voltage and current is attached between the two arms of the dipole, electric charges of opposite polarities flow into and then out of the two arms. As shown in the figure, electric lines of force connect the charges in the two arms and constitute the electric field. As a result of the flow of electric current in the arms of the dipole, magnetic lines of force are produced around the arms and constitute a magnetic field at right angles to the electric field. In a similar way, electric and magnetic fields are produced about any cable carrying an electric current. The larger currents produce larger magnetic fields. An electric field, however, may exist even if there is only a very small or no current flow in a cable. The combined electric and magnetic fields propagate outwards from the aerial with the speed of light in a direction at right angles to both the electric and magnetic fields in the direction marked S in the diagram. The electric and magnetic fields are denoted by the letters E and H.

The measurement units of the electric field, E, are volts per metre ($V\ m^{-1}$) and those of the magnetic field, H, are amperes (amps) per metre ($A\ m^{-1}$). The quantity S is known as the Poynting vector and is the rate of transmission of electromagnetic energy, with units of watts per square metre ($W\ m^{-2}$). This rate of transmission of energy is usually referred to in radio technology as the power flux density and in optical technology as the irradiance.

At very large distances from an aerial, ie two or three wavelengths, there are simple relationships between the magnitudes of the power flux density and the strengths of the electric and magnetic fields. These are

$$\underset{(W\ m^{-2})}{\text{Power flux density}} = \underset{(V\ m^{-1})}{\text{Electric field strength}} \times \underset{(A\ m^{-1})}{\text{Magnetic field strength}}$$

$$\text{Power flux density } (W\ m^{-2}) = [\text{Electric field strength } (V\ m^{-1})]^2/377$$

$$\text{Power flux density } (W\ m^{-2}) = [\text{Magnetic field strength } (A\ m^{-1})]^2 \times 377$$

The factor of 377 is the electrical impedance of free space, in ohms. As a result, the following are equivalent:

$$100\ W\ m^{-2} \equiv 194\ V\ m^{-1} \equiv 0.515\ A\ m^{-1}$$

However, close to the source or aerial these relationships no longer hold, because of phase differences between the signal from different parts of the source. In particular, *they do not hold* for the electric and magnetic fields generated by cables and electrical equipment operating at the domestic power frequency of 50 Hz, since, as may be deduced from Figure 1, the distances that people are usually situated from these sources are very much less than the wavelength (6000 km).

In different materials the force exerted by the magnetic field on a magnet or on a moving electric charge is proportional to the magnetic flux density, B, which is related to the magnetic field strength, H, through the permeability, μ, of the material:

$$B = \mu H$$

The unit of measurement of the magnetic flux density is the tesla (T). The permeability of animal and human tissues is almost identical to that of free air (1.26×10^{-6} henries per metre). An older unit of magnetic flux density is sometimes encountered, the gauss (G). The relationships between these magnetic quantities may be summarised as

$$\begin{aligned} 1\ \text{T} &\equiv 10{,}000\ \text{G} \\ 1\ \text{A m}^{-1} &\equiv 1.26\ \mu\text{T} \equiv 12.6\ \text{mG} \\ 1\ \text{mG} &\equiv 0.10\ \mu\text{T} \end{aligned}$$

$$\begin{pmatrix} 1\ \text{mT (millitesla)} \equiv 1000\ \mu\text{T (microtesla)} \equiv 1{,}000{,}000\ \text{nT (nanotesla)} \\ \equiv 10^3\ \mu\text{T} \qquad\qquad \equiv 10^6\ \text{nT} \end{pmatrix}$$

Some examples of the electric and magnetic field strengths to which humans are exposed in different contexts are listed in Table 3.

1.2 Physical interaction of electromagnetic fields with the human body

As well as the magnetic permeability referred to above, there are two other properties of human and animal tissues which are important in the physical interaction of electric and magnetic fields with the body. These are the relative permittivity, ε, and the conductivity, σ. The permittivity represents the influence of the electric field on the electric flux density and electric charge in tissue; in a similar manner the permeability represents the influence of a magnetic field on the magnetic flux density. Both permittivity and permeability are measures of the storage of energy in tissue under the influence of electric and magnetic fields.

The electrons and atomic nuclei of which all matter is composed have associated electric charges and magnetic moments. In atoms and molecules these become aggregated and also in more complex molecular arrangements such as membranes, which are an essential part of the cellular structures that make up tissue, Figure 3. Electrically charged ions are present in the cellular fluids and the blood. Some molecules, which are otherwise electrically neutral, may have an apparent excess of positive charge at one end and a negative charge at the other, thus forming small electric dipoles. Atoms, molecules and chemical ions will tend to move and become aligned with the directions of electric and magnetic fields because of their electric charges and magnetic moments. If these fields change direction or oscillate, the atoms and molecules will attempt to change direction

and also oscillate. The energy associated with these alignments and oscillations in electric and magnetic fields of strengths normally encountered by people is about one-million times less than the energy associated with the random Brownian movement and vibration of the atoms and molecules due to body temperature (Fröhlich, 1982). Nevertheless, it is these alignments and their changes of direction which are largely responsible for the electrical properties of tissue and the absorption of energy by the body from electromagnetic fields.

Static electric and magnetic fields do not produce any movement of electric charge other than at the instant at which the fields are applied to the body. However, they do produce molecular alignments. Movement of the body or the blood flow in a static magnetic field may induce electric currents and potentials similar to the effects of movement of a conductor in the magnetic field of an electric dynamo.

The electrical properties of tissue have been investigated and described by many authors (Schwan, 1957; Grant et al, 1978; Schwan and Foster, 1980; Grant, 1983, Schwan, 1983a, b, c; Barnes, 1986a,b; Polk, 1986; Takashima, 1989). Living tissue is characterised by an extremely large electrical relative permittivity at low frequencies, Figure 4, which changes rapidly when the tissue dies. Below 10 kHz the ability to store electrical energy represented by this permittivity is thought to be due to the characteristics of the cells in the body and their outer membranes, Figure 3. These membranes consist of an electrically charged phospholipid layer containing large protein molecules (Fishman, 1984) which are capable of transporting a variety of other molecules into and out of the cell. Although all the details of the transport mechanisms are not completely understood, they involve enzyme reactions and sometimes, potassium, sodium or calcium ions. The thickness of the cell membranes is about 5 nm. As a result of the differences in the ion concentrations between the insides of the cells and the surrounding fluids, there is typically an electric potential of between 10 and 100 mV across the membrane. This corresponds to a static electric field of between 2×10^6 and 20×10^6 Vm^{-1}. This very high field possibly influences the shape of the protein molecules situated in and protruding from the membrane. It also results in a distribution of electrically charged ions (counterions) on both sides of the membrane. The counterions can move over the surface of the membrane under the influence of an applied electric field and the protein molecules can move within it. It is conceivable that such movements may influence the shape and function of the protein molecules affecting transport of substances across the membrane. It is the movement of the counterions (Schwarz, 1962), and possibly ion transport by the protein molecules (ionophores), that is thought to be responsible for the high permittivity of living tissue at low frequencies.

As the frequency is increased above 10 kHz and up to about 10 MHz the permittivity is considered to be due to the cells appearing as small non-conducting bodies. This is because the electrical conductivity of the membranes is very low in comparison with that of the internal and external fluids. The effect is to shield the internal structure of the cell from the electric field. As the frequency is increased further the conductivity of the cell membrane increases and the electrical properties of tissue are associated with biomolecules and water, the latter either as water of hydration bound to larger molecules or as free water in the internal and external cellular fluids.

Advances in understanding of the electrical properties of tissue have been paralleled by an increased understanding of the interaction of electromagnetic fields with both cells and the human body as macroscopic objects (Lin et al, 1973; Bernhardt and Pauly, 1973; Deno 1974; Deno and

Zaffarella, 1975; Durney et al, 1978; Bernhardt, 1979; Pauly and Bernhardt, 1980; Schwan and Foster, 1980; Gandhi, 1980; Durney, 1980; Schwan, 1983b; Gabriel and Grant, 1985; Gandhi et al, 1985; Dimbylow, 1987, 1988; Bateman et al, 1990).

A first approach is to regard the human body as a homogeneous sphere or ellipsoidal body containing cells, which in turn are regarded as conducting spheres surrounded by insulating membranes. For the lower frequencies at which the wavelength of the electromagnetic field is much greater than the dimensions of the human body, ie for frequencies less than about 10 MHz, the average internal field, E_i, inside the body at a point where the internal radius is R, can be related to the average external electric field, E_0, and the average of magnetic field, B_0, by the equation (Lin et al, 1973; Bernhardt, 1979):

$$E_i = \frac{3\varepsilon_0 2\pi f E_0}{\sigma} + j2\pi f B_0 R \qquad (1)$$

In this equation ε_0 is the permittivity of free space (8.85×10^{-12} farad per metre), f is the frequency in hertz, and σ is the conductivity of tissue in siemens per metre. The symbol j represents the square root of minus one and is used to denote a difference of 90° in angle between the external magnetic field and the internal electric field. The average values of the fields are the root mean square (RMS) values of the amplitudes, which for sinusoidal fields oscillate between zero and positive and negative values that are 1.4 times these averages.

The products $2\pi f E_0$ and $2\pi f B_0$ in the equation represent in physical terms the average (RMS values) of the *rates of change* of sinusoidal fields. Some waveforms, particularly those which are rectangular or sawtoothed, may have rates of change that are higher than those present in sinusoidal waveforms. It is possible, therefore, that fields of nominally the same frequency and average field strength may differ in the internal field strengths they produce in tissue and their effectiveness in producing biological effects.

The conductivity of muscle tissue at different frequencies is given in Table 4 and the consequent ratios of the internal to external electric fields. It is obvious that at all frequencies where the formula is approximately correct (ie <10 MHz) the internal field is many orders of magnitude less than the external field. A physical explanation for this phenomenon is that the body can be regarded as a relatively good conductor of electricity covered by a slightly less conducting skin. Since conducting bodies cannot sustain electric fields within themselves, electric charge flows to distribute itself over the surface of the body in such a way that the electric field within the body becomes very small. However, one consequence is that the external electric field becomes enhanced at the surface of the head and can be up to 20 times the value of the unperturbed electric field (Deno, 1974; Deno and Zaffarella, 1975). These very high fields at the surface of the body can result in movement of the body hair and small perceptible spark discharges when the external field at 50 Hz exceeds about 2000 V m^{-1}.

The effects on cells within the body must now be considered. As a consequence of the very high resistance of the membrane, 10^5–10^7 ohm metre (Ω m), compared to that of the fluid of the cell and its surroundings (Bernhardt, 1986), at low frequencies, any applied external field to the cell becomes concentrated across the cell membrane. Bernhardt (1979) gives a formula which links the potential V_m, appearing across this membrane to the length of the cell, L, in the electric field direction, to the strength, E_i, of this field and to the frequency, f, ie

$$V_m = \frac{E_i L}{2\sqrt{1+(f/f_0)^2}} \quad (2)$$

At frequencies less than f_0, which is a function of the cell dimensions, capacitance and resistance, and is about 1 MHz for most cells in the human body, the equation simplifies to

$$V_m = \frac{E_i L}{2} \quad (3)$$

Cell dimensions are typically about 10–100 μm so that for a field external to the human body of 10,000 V m^{-1} per metre at the power frequencies of 50 Hz, the potential across the membrane of cells in the body will be less than one-hundredth of a microvolt. At 100 kHz with a field external to the body of 1000 V m^{-1} the potential across the membrane will be about one-tenth of a microvolt. These potentials are about 1000 times less than the natural cell membrane potential of 50–100 mV or the potential of about 10–40 mV required to trigger a nerve cell. It is perhaps worth noting that the ability of some fish to detect extremely weak electric fields is probably related to certain very large specialised cells in their bodies of some 10 cm in length (Schwan and Foster, 1980; Kalmijn, 1984), which will have potentials across their membranes that are 10,000 times that for a 10 μm cell in similar circumstances.

In considering the possible response of a cell to an electromagnetic field the magnitude of the signal that it receives from the field in relation to the electrical noise in the cell itself is important. This noise arises from the thermal agitation of the electrons in molecules and membranes and from the dynamic processes of the cell metabolism producing changes in electron distributions. Estimates of this noise in cell membranes give equivalent potentials in the range 5–50 μV (Barnes, 1986b; Weaver and Astumian, 1990). Table 4 gives the internal and external fields which would produce 'signal' potentials across the membranes of a cell of 100 μm dimensions equivalent to a noise level of 5 μV. A theoretical expectation would be that any biological effects would be unlikely at smaller field strengths. This appears to be the case for the acute effects such as electric shock and heating. However, biological effects in cells have been reported at field strengths that are 100 or more times less than these values in the frequency range 10–100 Hz or at higher frequencies when amplitude modulated at these frequencies (Adey, 1983a,b; McLeod et al, 1987; Blackman et al, 1988; Blackman et al, 1989). Fields of such low intensity can only be effective if some mechanisms exist in cells for averaging energy over times that are long compared with the inverse of the frequency (ie 20 ms at 50 Hz, 1 ms at 1 kHz, 1 μs at 1 MHz) or for interaction with enzyme-catalysed reactions dependent on molecular conformation in cell membranes (Weaver and Astumian, 1990). At the lower frequencies such mechanisms exist among those responsible for the high permittivity of tissue at these frequencies. The experiments imply also that cells can recognise the low frequency amplitude modulation of higher frequencies. Explanations based on such considerations are not so likely to account for the windows of intensity, as distinct from frequency, that some experimenters claim to have observed.

Frequency windows which have been observed in some experiments have been explained in terms of ion cyclotron resonance mechanisms involving the combined action of applied alternating fields and the earth's static magnetic field. Although this attractive explanation is superficially

plausible, considerations of the frequency of thermal collisions and energies suggests that these would prevent any effective transfer of energy by the mechanism implied by the simple theory (Liboff et al, 1990).

A variety of other, hypothetical, non-thermal mechanisms have been proposed that would lead to biological effects. They have been reviewed by Taylor (1981) and Postow and Swicord (1986). Possibly the mechanism most frequently cited is that by Fröhlich (1980). He pointed out that the elastic properties of the cell membranes could result in mechanical resonance at a frequency of about 50 GHz which, together with the electrical properties, could result in energy transfer from an electromagnetic field at the same frequency. This energy could then transfer further and concentrate in protein molecules. Cellular systems with slightly different resonance frequencies could interact to produce frequency softening, ie interaction at a frequency that corresponded to the difference in frequency between the two systems. In principle this could result in energy transfer at frequencies lower than 50–100 GHz. Fröhlich postulated that such energy transfer could interfere with cellular mechanisms at stages in the cell cycle which result in certain configurations of molecular energy states. There is very little direct evidence for this or any of the other hypothetical mechanisms, although they have been invoked to explain biological effects observed in cells at these very high frequencies and apparently with very sharp resonances (Gründler and Keilmann, 1978; Fröhlich, 1980). Some experimental evidence for resonance absorption of microwaves in DNA (Edwards et al, 1984) could not be confirmed on more careful investigations (Gabriel et al, 1987). It must, however, be pointed out that at these very high frequencies the penetration of the electromagnetic fields into the body is very limited. For example, at 10 GHz the electromagnetic field has been reduced by a factor of 10 after penetrating about 4 cm in tissue, but at 100 GHz it is reduced to this level after about 4 mm (Polk, 1986) in tissues with a high water content.

In any detailed investigation of the interaction of electromagnetic fields with the human body its shape and internal structure need to be considered. Internal electric currents induced by fields will be enhanced in the narrow sections of the body, particularly in the neck, wrists and ankles, where not only is the cross-section smaller but the comparatively non-conducting bone structures tend to confine currents to the softer tissues. In the frequency region from ten to a few hundred megahertz, the body becomes a much more effective absorber of electromagnetic radiation due to resonance between the wavelength of the radiation and the body dimensions. The exact frequency depends on the size of the person, whether he or she is sitting or standing and whether or not he or she is in good contact with the ground. At frequencies above a few hundred megahertz differences in electrical properties between the body tissues, ie skin, fat, muscle and bone, become of increasing importance and the radiation may be reflected at interfaces between these tissues to form standing wave patterns which increase the absorption in some places of the body relative to others. The curved nature of the body surfaces, particularly the head, may cause refraction and focusing of the radiation, very much in the way that visible light is refracted by curved glass surfaces. In some frequency ranges, mainly in the three gigahertz region, this could in principle cause the absorption of energy in some small regions of the head to be ten to twenty times the average value for the whole body. At higher frequencies again, above some tens of gigahertz, the absorption will be predominantly in the surface layers of the body, as it is for the infrared radiations in the optical range that begins at the highest frequency which, by convention, is included in the electromagnetic field range (300 GHz).

Although considerable progress has been made in the ability to calculate patterns of energy deposition in the human body (Dimbylow, 1987, 1988, 1991), further development and experimental

verification is still needed. Areas requiring attention are the details of areas of high absorption in the body, the effects of the temperature controlling mechanisms of the body, the interactions in situations close to antenna systems and the effects of pulsed and non-sinusoidal waveforms.

At low frequencies it is usual to use the induced electric current densities in tissue as an index of the degree of interaction as well, as to a lesser extent,the specific energy absorption rate (SAR) in units of watts per kilogram (W kg^{-1}). The latter quantity is used almost exclusively as the index of interaction at high frequencies. Both current density and SAR are proportional to the electric field strength in the tissue and as such are appropriate quantities whether thermal or athermal effects are being considered. They provide a basis for extrapolation of effects seen in cells and animals to man. However, it might be expected that the basis for extrapolation will be refined in future as interactions of electromagnetic fields with the body are better quantified and an understanding of the biological effects becomes more advanced.

2 Studies of effects on general health and birth outcomes

In general, neither workers nor members of the public are exposed to electromagnetic fields of sufficient intensity to cause even the milder forms of perception, ie vibration of body hairs in power frequency electric fields, with the possible exception of situations close to the vicinity of high voltage overhead power lines or in switch yards where the fields exceed 5 kV m^{-1}. However, it is open to speculation whether the slight and imperceptible accommodations and adaptations of the body in the presence of electromagnetic fields or the possible subtle effects of these fields on cell membranes might lead in the longer term to a deterioration in general well-being and an increased incidence of disease in those exposed. It has been suggested, for example, that the influence of magnetic fields could increase blood pressure (Easterly, 1982). Some slight transient changes in blood pressure and whole blood cell count have indeed been observed without an apparent effect on basic health (Marsh et al, 1982) in workers exposed to static magnetic fields of strengths up to 20 mT.

It is known that adaptation of the body to increases in ambient temperature involves short-term cardiovascular strain and changes in blood cell concentrations (Robinson, 1963). These might, therefore, be produced by the heating effect of microwave radiation absorption.

2.1 General effects on health

Early reports from the former USSR of adverse effects on health associated with exposures to electromagnetic fields at both low and microwave frequencies as well as static magnetic fields (Asanova and Rakov, 1966; Gordon, 1966; Vyalov, 1967) led to determined efforts in other countries to confirm these findings. Long-term exposures to power frequencies, microwaves or magnetic fields were alleged in the Russian reports to produce very similar non-specific symptoms, ie, tiredness, headaches, nausea, loss of sexual potency, cardiovascular effects, sleep disturbances, anxiety and changes in blood cell concentrations and blood chemistry. Cardiovascular changes and central nervous system effects were particularly emphasised (Gordon, 1966). Data in these reports were often sparse, the reports impressionistic, without proper control populations and the selection of the study population itself essentially *post hoc*, ie based upon *a prior* history of complaint by the workers concerned.

Attempts to verify the Russian observations have included surveys of exposed groups and carefully controlled laboratory experiments with volunteers. Although some reports have tended to support the early Russian work (Miro, 1962; Sazonova, 1967; Danilin et al, 1969; Deroche, 1971; Korobkova et al, 1972; Fole, 1973; Fole et al, 1974; Moscovici et al, 1974; Sadcikova, 1974; Vyalov, 1974; Baranski and Czerski, 1976), other and particularly more recent studies do not give such support (Barron and Banaff, 1958; Kouwenhoven et al, 1967; Strumza, 1970; Singewald et al, 1973; Siekierzynski et al, 1974a; Malboysson, 1976; Roberge, 1976; Robinette and Silverman, 1977; Lilienfeld et al, 1978; Stopps and Janischewsky, 1979; Knave et al, 1979; Robinette et al, 1980; Marsh et al, 1982; Silverman, 1985; Broadbent et al, 1985; Baroncelli et al, 1986). Strumza (1970), for instance, provides an account of a 'health events' survey made of male employees of the French power company Electricité de France. In total 144 such men, all living in rural areas, were included, together with their wives (129) and children (252). Medical and social security records covering almost 1900 man years in all were scanned, thus assuring an objective recording of health events. Employees and, in turn, wives and children were classified as 'exposed' if place of residence and workplace were closer than 25 m to high voltage (220–400 kV) cables, and 'not

exposed' if they lived and worked more than 125 m removed from such cables. All men of intermediate exposure status were excluded from the study at an earlier stage. For four separate measures of expressed demand for medical services and drugs, the results showed quite clearly no tendency for the exposed group to 'consume' more than the non-exposed group. This observation held for men, women and children. The analysis appears to have controlled for variation in neighbourhood type.

In a neighbourhood health status survey in California, USA, Haupt and Nolfi (1984), defined 'exposed' as living with 0.14 mile (200 m) of a 400 kV DC power line, and 'not exposed' if resident between 0.65 mile (1 km) and 0.85 mile (1.4 km) from it. The success rate in persuading individuals to participate in the survey was low (61.5%) and data were entirely subjective, confined to reported symptoms of the kind mentioned above in relation to the Russian studies, and visits to a doctor. No significant differences between the exposed and not-exposed groups were detected, even after statistical adjustments by regression analysis for possible confounding factors. However, the group sizes (245 exposed and 193 not exposed) were probably only sufficient to exclude gross inter-group differences.

The report by Broadbent et al (1985), is one of the most interesting in that it was a vigorous attempt to measure non-specific health effects in electric power transmission and distribution workers. This was achieved by using a schedule of derived health status indices obtained from both reported health events and a variant of the Middlesex Hospital Questionnaire (MHQ). This is a standardised instrument with the main advantages that it can be administered by non-medical staff and that a range of expected values is available for the various subsections of the questionnaire (Depression, Somatic Symptoms, Obsessional Symptoms, etc) based on past examples of its use. A small number of questions in addition to those of MHQ were asked, for instance, about visits to the general practitioner, intake of medicines and experience of headaches. All the health indices are thus based on subjective self-assessment. Unlike most other studies, objective measurements of exposure were made with personal dosemeters, although only of electric field exposure. These dosemeters were worn for periods of 2 weeks, providing data on 287 subjects. In addition, prior estimates of exposure were available as informed, but ultimately subjective, engineering judgements. These exposures were expressed in terms of the product of the electric field strength and the time spent in the field, ie in kilovolt hours per metre (kV h m^{-1}). It emerged at the end of the study period that the measured exposures were generally much lower than those estimated. This finding has implications for other studies in which exposures are based on subjective and surrogate indices of exposure.

The study was sufficiently sensitive to show clear differences in health between job categories and geographical districts in which the work took place. Adverse health effects correlated with lack of job satisfaction, personal domestic problems, working alone, working long hours, and recent changes in shift times. There were no adverse health effects associated with higher exposure levels. The implied annual occupational exposures averaged about 150 kV h m^{-1}, but ranged up to 500 kV h m^{-1}. These may be compared with the average domestic exposure of the general population in the USA of 70 kV h m^{-1} (Silva et al, 1985); the exposure of the general population in the UK will be rather similar (see Table 3). It is not possible to say what they represent in terms of magnetic field exposures, but the members of the study population will undoubtedly have been exposed to higher fields than experienced by the general population, possibly by factors of between 10 and 1000.

This absence of any adverse health effects related to power frequency electromagnetic field

exposure broadly confirms the findings of other health surveys of occupationally exposed persons undertaken in the last decade in Europe and North America (Singewald et al, 1973; Malboysson, 1976; Roberge, 1976; Stopps and Janischewsky, 1979; Knave et al, 1979; Baroncelli et al, 1986; Gamberale et al, 1989). Each of these surveys examined railway or power workers' health and found no significant excess of adverse effects in subgroups with higher electromagnetic field exposures by comparison with controls or unexposed populations.

There have been two major epidemiological studies which are relevant to the possibility of adverse health effects of exposure to radiofrequencies and microwaves. However, it should be remembered that persons particularly exposed to these higher frequencies are usually technicians working on the maintenance and repair of equipment that is itself usually driven by electric power at 50 or 60 Hz.

One of the studies (Robinette et al, 1980) was of 40,000 American Naval personnel, of whom 20,000 may have been rather more exposed (ie to levels of power flux density occasionally in excess of 100 W m^{-2}) to radar emissions during the 1950s (Korean War period) than the other 20,000 who formed the control or reference group. The exposures of the latter were considered not to have exceeded 10 W m^{-2}. The rather more exposed group was split into those who might be regarded as moderately exposed and those who might be regarded as highly exposed. However, the degrees of exposure could not be established with any certainty and the description is in fact a measure of potential rather than actual exposure.

Mortality in the groups was compared for the period, 1950–1974 (Table 5). The ratios given in this table are based on the combined experience of the total population of 40,000, standardised for year of birth. Confidence limits for the ratios are not reported. However, the authors remarked that the mortality ratios for the moderate exposure group are significantly different from the high exposure group at the 1% level for all diseases and 'other diseases'. Further breakdown indicates that most of the difference for 'other diseases' is due to diseases related to the consumption of alcohol. For both categories the ratio for the moderate exposure group is lower than that of the low exposure group. The question of elevated cancer risks among those occupationally exposed to electromagnetic fields is dealt with in Section 3, but it is interesting to note that the moderate and high exposure groups appear to have slightly higher mortality rates from cancers of the digestive organs, respiratory tract and the lymphatic and haematopoietic systems. None exhibits a significant trend with the level of presumed exposure, although that for the lymphatic and haematopoietic systems is marginally more so than the others.

As indices of general health, hospital admissions and compensation awards were also studied. The rates of admission to Naval hospitals are given in Table 6. These are uncorrected for age or time of entry into service and must be regarded with some reservations. The moderate exposure group has significantly lower admission rates for diseases of the ear, nose and throat (at the 1% level), acute respiratory infection (at the 1% level), other respiratory diseases (at the 2% level), diseases of the urinary and male genital organs (at the 5% level), and for accidents, poisonings and violence (at the 0.1% level) than the high exposure group. However, for these categories of disease and injury the moderate exposure group also has lower admissions than the low exposure group – significantly so at the 1% level for acute respiratory infection, at the 5% level for diseases of the urinary and male genital organs, and at the 0.1% level for accidents, poisonings and violence. A similar ranking of the moderate exposure group in relation to the low and high exposure groups is evident in respect of compensation claims (Table 7). The high exposure group has significantly greater claims than the moderate exposure group in respect of the musculoskeletal

system (at the 0.1% level), organs of the special senses (including cataracts) (at the 5% level), respiratory system (at the 1% level), cardiovascular system (at the 0.1% level) and mental disorders (at the 5% level). However, for none of these or any other category does the high exposure group significantly exceed the rates in the low exposure group.

Another major study was of the staff of the American Embassy in Moscow, who were exposed to very low levels (maximum 0.15 W m^{-2}) of microwave radiation in the period 1953–1976 (Lilienfeld et al, 1978). The initial impetus for this study was the fear among the embassy staff that the exposure was increasing the risks of cancer. The study covered 1827 employees at the embassy and 3000 of their dependants in comparison with 2561 employees with 5000 dependants at other American embassies not subject to microwave exposure. The study had only a limited power for the detection of small differences in risk. An exhaustive comparison of all symptoms, conditions, diseases and causes of death among the employees and their dependants was unable to establish differences in health status of any measure that could reasonably be attributed to the microwave exposure. The proportion of deaths due to cancer among the staff of the Moscow Embassy was slightly less than the proportion among the staff at other embassies, so that within the statistical uncertainties there was no indication that the microwave exposure had any effect on cancer rates. The death rates at both the Moscow and other embassies were less than for the general population of the USA, with that of the Moscow Embassy slightly less than that of the other embassies. Men employed in the Moscow Embassy were three times as likely to acquire protozoal infections, both men and women were more likely to suffer from a variety of common health problems, and mumps was more frequent among children of the embassy staff.

Two studies from eastern Europe are worthy of mention. The first by Djordjevic et al (1979) was of 322 Yugoslav workers aged between 25 and 40 years. All had been exposed to microwaves at intensities not exceeding 50 W m^{-2} for 5 to 10 years. They were subjected to a programme of clinical examination which included detailed internal, neurological, ophthalmological, otological, haematological and biochemical investigations. The investigations comprised analyses of urine, peripheral blood cells, blood sugar, cholesterol, lipids and bilirubin. The control group consisted of 220 persons who matched the exposed group in respect of age, character of working regime, social and living conditions. Although the exposed group had more subjective complaints of headache, fatigue and irritability, these were attributed to their generally rather uncomfortable working conditions. In all other respects there were no significant differences in health between the two groups. The second study by Siekierzynski et al (1974 a,b) was of 841 Polish men aged between 20 and 45 years exposed to microwaves. These were divided into two contrasted groups: those exposed to power densities between 2 and 60 W m^{-2} and those whose exposures did not exceed 2 W m^{-2}. Although both groups had an unusually high incidence of functional disturbances, there was nevertheless no statistically significant difference between the groups. Neither in the Yugoslav or Polish study could any evidence be found for an increase in lenticular opacities or cataracts in the more highly exposed persons.

More recently, a Swedish study (Nilsson et al, 1989) was made of 17 radar mechanics and engineers of average age 52 years with probable exposure to microwaves in excess of 10 W m^{-2} in comparison with 12 unexposed or minimally exposed men, all military technical personnel. The length of time of occupational exposure was not given, but appears to have been some decades. No indications of central nervous system effects associated with the microwave exposure could be found in extensive neurological, psychometric and neuropsychiatric examinations. However, the frequency of occurrence of an increased protein band with an isoelectric point of 4.5 in the cerebrospinal fluid

was higher in the exposed group. The nature and clinical significance of this finding is admitted by the authors to be unclear. The suggestion is made that it might be related to the pulsed nature of the radar emissions on the basis of similar observations in exposed monkeys. The authors noted that the men were also exposed to pulses of magnetic flux density with high rates of change of up to 350 teslas per second ($T\ s^{-1}$).

In a 2 year study of workers exposed daily to pulsed radar microwaves at intensities in the range 0.1–200 $W\ m^{-2}$, but not usually in excess of 50 $W\ m^{-2}$, Goldoni (1990), found indications of some lowering of both white and red blood cell concentrations. The data were insufficient to draw any definite conclusion, but the author claims to be encouraged to undertake a longer study.

A paper from China (Chiang, 1989) describes differences in visual reaction times, memory function scores and white blood cell phagocytosis in groups of children, soldiers and students exposed to microwaves at levels down to 4 $mW\ m^{-2}$. Virtually no details are given in the paper of the tests, on the selection of the exposed and control populations or the circumstances of the exposure. Since, however, the number of controls is approximately one-half of the number exposed, there would appear to have been very little attempt to match the groups. Examination of the data on the results of the visual reaction time test does not show any significant differences between the exposed and control groups, but significant differences between the children, soldiers and students. The memory function scores do show significant differences between exposed and control groups, but these are not so great as the differences between the children, soldiers and students. It seems likely that the observed differences reflect inherent differences between the exposed and control populations rather than the effects of microwaves. No details of the phagocytosis test are given in the paper, but in a private communication two test quantities are described in greater detail. The first consisted of the percentage of a sample of 100 neutrophils that engulfed staphylocci and is called the phagocytic activity. The second, the phagocytic index, was 100 times the number of staphylocci engulfed by active neutrophils, ie those actually engulfing staphylocci. Only the phagocytic index is reported in the publication. The ratios of the phagocytic activities of the exposed to control groups varied between 0.72 and 1.23 in a manner that did not depend markedly on the exposure levels. The actual phagocytic activity for the child controls was $65.8 \pm 18.7\%$ and for the soldier controls $84.0 \pm 11.7\%$ The respective phagocytic indices were said to be 4.45 ± 1.56 and 4.81 ± 1.10, respectively. The ratios of the phagocytic indices varied between 0.46 and 1.23 with a tendency to be lower in the most exposed group, ie the students. The variation is rather typical of that observed between different samples in such tests, the results of which depend on age, sex, diet, health and smoking habits. Changes have been seen in the cells of animals exposed to very much higher levels of microwave radiation (300 $W\ m^{-2}$, see Saunders et al, 1991). It is quite possible that the differences seen by the Chinese workers represent natural variation between populations or may be due to chance.

Workers with radiofrequency heat sealing and plastic welding machines, which operate in the frequency range 20–100 MHz, are possibly the occupational group most highly exposed to electromagnetic fields. The study by Kolmodin-Hedman et al (1988) of the health problems of a group of such workers is, therefore, of particular interest. The exposed workers consisted of 51 men and 62 women who worked with plastic welding machines. For controls, 23 female sewing machine workers were selected for the same age as the exposed women. The mechanical character of the work performed by the two groups is similar and highly repetitive. It requires a bent forward posture to control the work pieces. At most of the plastic welders' workplaces field intensities exceeding 100 $W\ m^{-2}$ could be found and intensities exceeding 250 $W\ m^{-2}$ in at least 50%. The

NRPB reference level for the frequency range 30–100 MHz is 10 W m^{-2} (NRPB, 1989). This does not necessarily imply that the basic restrictions advised by NRPB as regards the induced currents in the bodies of the workers are exceeded. However, that this was probably the case is indicated by the fact that 70% of the women and 60% of the men reported light radiofrequency burns at least once a year to their hands. Those burns were reported as being deep and taking a long time to heal. As far as general health was concerned there was little difference between the exposed and the unexposed groups. Both suffered more or less equally from muscular pain and tiredness. There was, however, a difference in neurothenic symptoms (nervous disability characterised by lassitude, headache, backache and indigestion) evaluated by a Swedish questionnaire. By use of this questionnaire, groups exposed to solvents have been found to have a higher frequency of symptoms (18%) compared to unexposed groups (6%). The incidence in the plastic welders was 19% compared to 9% in the control group. The plastic material gives off fumes when heated and this may have accounted for a fairly high incidence of complaints of eye irritation. Numbness of the hands was experienced by 40% of the exposed compared to 22% of the controls. The ability to discriminate between two closely spaced sharp points was diminished in 39 of the 113 exposed persons compared to 1 in the 23 controls. This difference is significant at the 5% level. The frequency of this lack of discrimination in the Swedish population is quoted by the authors as 10%, with double the frequency in women as in men. Although the effect can be produced by repetitive mechanical work with hands and fingers, since the incidence was much lower in the controls who did similar work in a mechanical sense, it seems possible that the electromagnetic field exposures were responsible.

As part of the same study the pregnancy outcome in 305 female plastic welders during 1974–1984 was found not to differ significantly from the Swedish average concerning malformations and prenatal mortality.

2.2 Effects on the eyes

There have been reports of apparent associations between cataract formation and exposure to microwaves. The frequencies concerned are above 3 GHz and the power densities in excess of 400 W m^{-2}, and possibly in excess of 3000 W m^{-2} (Hirsch and Parker, 1952; Shimkovich and Shilajev, 1969; Zaret et al, 1970; Zaret, 1973). In addition to these reports of cataract formation, there have been several reports of an increased incidence of minor defects of the eye lenses in microwave workers, particularly in older age groups, but usually having no effect on visual acuity (Cleary and Pasternack, 1966; Majewska, 1968; Appleton and McCrossan, 1972; Zydecki, 1974). A report has also been published indicating damage to the retinas of such workers (Tengroth and Aurell, 1974). Against these reports must be set others which indicate that there is no association between cataracts and lens defects when exposures are to power density levels less than about 100 W m^{-2} (Barron et al, 1955; Barron and Barraff, 1958; Cleary et al, 1965; Siekierzynski et al, 1974b; Shacklett et al, 1975; Hathaway et al, 1977; Djordjevic et al, 1979; Cleary, 1980). Very few of these studies have investigated elderly, retired workers in whom long-term effects might be apparent as they have been in the eyes of glass workers exposed to infrared radiation (Lydahl and Phillipson, 1984).

2.3 Suicide and depression

An early account of an apparent association between suicide and residence near overhead

and underground high voltage (>33 kV) power lines (Reichmanis et al, 1979) was followed by a further account (Perry et al, 1981), which was based on magnetic field measurements rather than estimates. These measurements were made outside front doors of the residences in which suicides had taken place. The median measurement was about 40 nT with a 90% range from less than 20 nT up to 140 nT. The account is of a case-control study covering parts of Shropshire and Staffordshire and including Wolverhampton, Walsall and Dudley. The cases consist of 598 suicides in the area between January 1969 and October 1976 who had resided at the address for more than 14 days prior to death. Control addresses were a random sample in the same area chosen from adults in the electoral registers for 1979.

Cases and control addresses were not matched for type of residence nor was this variable taken into account in the analysis. This may have confounded the result, particularly since suicide rates are linked to occupational class. It has also to be noted that there could have been 10 years between the measurement of magnetic field and the date of actual suicide, and the relevance of measurements external to the houses is not clear. In their analysis the authors state that at the 1% level there are significantly more suicide than control measurements above the median for the two groups. This median, although not given exactly, was quoted to be about 40 nT. However, analysis of the data given in the paper shows no significant difference in the proportion of suicide and control measurements above 40 nT. To examine this matter further, Table 8 gives the estimated odds ratios of committing suicide against the measured magnetic field strength, together with the 95% confidence interval. Although, as the authors point out, the distribution of the measurements differs significantly between the suicide and control groups, the odds of committing suicide do not vary in a consistent manner with measured field strength. In particular, while the estimated odds are similar for the 0–20, 40–60, 60–80 and 80–100 nT intervals, the odds over these ranges are significantly less than the odds for the 20–40 nT interval at the 0.1% level. This illustrates why the conclusions reached by the authors are extremely sensitive to the choice of a boundary in the region of 40 nT for the comparison between case and control addresses. As a consequence of these difficulties, no very great reliance can be placed upon the authors' conclusions. No other studies have been reported on this topic. An association between suicide and possible magnetic field exposure was not found in another UK study made with the primary objective of investigating an association with cancer (McDowall, 1986) (Section 3.2).

More recently, one of the authors of the earlier study of suicide has published a study of the distribution of different illnesses among hospital patients in Wolverhampton according to their place of residence in multistorey blocks (Perry and Pearl, 1988). The positions of flats of these patients was determined in relation to the main electric supply cable rising through the multistorey block. The mean magnetic fields at the entrances of those determined as being 'near' the cable was 32 (standard deviation, SD ± 29) nT and for those determined as 'distant', 16 (SD ± 11) nT. A preliminary study comprised 508 patients or 395, if those in hospital for childbirth are excluded, admitted to hospital in 1985. For this group of patients there were no significant differences in the distributions of disease between 'near' and 'distant' flats. An additional study comprised 95 patients with heart disease admitted between 1967 and 1984, 104 patients of drug overdose admitted between 1980 and 1985, and 75 patients with psychiatric disorders admitted between 1953 and 1986. There are discrepancies in the text between the numbers in these groups and the numbers claimed to have been analysed. Although the authors analyse these data to show that there are significantly greater numbers of persons from 'near' flats suffering from myocardial infarct, hypertension, ischaemic heart diseases and from depression at about the 5% level, this conclusion is compromised by the fact

that no estimate of the numbers at risk in the near and distant flats is presented. The authors say that they "feel that the numbers at risk were almost evenly divided between near and distant flats". The analysis would have been more convincing if a wider range of disease conditions had been included for the same, instead of different, admission periods. This could have provided some measure of those at risk in the two categories of flat.

It is important in epidemiological studies to make strenuous efforts to adjust for possible confounding factors, many – in particular the early Russian – studies did not do so. Such observational studies are influenced to a much greater extent than experimental studies by unconsidered, confounding factors.

2.4 Volunteer studies

Apart from some experimental studies on heat perception thresholds for microwave radiation described in a companion report (Saunders et al, 1991), reputable laboratory studies of the effects of electromagnetic fields on human subjects have been carried out almost entirely at low frequencies. These studies have indicated alterations in reaction time at frequencies below 12 Hz and electric fields of less than 4 V m^{-1} (Hamer, 1968) as well as alterations in the patterns of electroencephalogram (EEC) recording in 5 Hz, 6 kV m^{-1} fields. Such reports as well as the reports of adverse health effects on the general health of workers exposed to power frequencies stimulated a series of carefully controlled experiments (Hauf, 1974) whose results are summarised later (Hauf, 1982). In these experiments volunteers were exposed for periods of up to some hours to 50 Hz electric fields of strengths up to 20 kV m^{-1}. Although in some experiments there was a decrease in pulse rate during exposure which was ascribed to the long periods spent in a relaxed position during the experiments, there was no effect on the patterns of electroencephalogram (EEC) and electrocardiogram (ECG) records, blood pressure, blood chemistry and concentrations of biochemicals in urine. There was, however, a slight increase in the concentrations of white cells in the blood, but this was within the normal physiological range. Later studies have confirmed the absence of any detectable effects of exposure to electric fields at these strengths in the frequency range 50–60 Hz (Sander et al, 1982; Graham et al, 1985) and indicated a similar absence of any effects of magnetic fields at these power frequencies at strengths up to 5 mT. After exposure to magnetic field strengths high enough to give rise to phosphene stimulation (as sensation of light flicker) at about 60 mT at 50 Hz, volunteers complained of feeling unwell and of headache (Silny, 1985a,b). A similar effect was reported at lower frequencies when the threshold for phosphene stimulation was exceeded by direct current application to the head (Adrian, 1977).

In an attempt to determine whether exposure to electric fields has any effect on mental functions, electric currents were passed through volunteers from electrodes attached to their heads and bodies while they were subjected to a series of psychological tests (Bonnell et al, 1985; Stollery, 1986). The pattern of current was equivalent to that produced by exposure to a 36 kV m^{-1} field at 50 Hz. The psychological tests were for stress and arousal, verbal reasoning skills and attention skills. Subjects were tested twice, in one test no current was applied. One group of subjects appeared to show higher arousal in their second test when no current was applied and also improvements in syntactic reasoning. Another group who were sham exposed in the first test showed no differences. Both groups showed no differences associated with exposure to applied current in the majority of the tests. It seems rather doubtful that any weight should be given to the

finding that one group showed some increase in performance in a second test when no current was applied.

Possibly one of the most interesting investigations of the response of humans to electromagnetic fields is that of Wever (1973, 1974, 1985). In a study of human circadian rhythms it was found that these became desynchronised when people were kept for periods of about 30 days in a room shielded from the effects of the earth's static and time varying fields, and from all other environmental clues such as noise and light. This desynchronisation could be prevented by the presence of an extraordinarily low field of 2.5 V m^{-1} at a frequency of 10 Hz. This frequency was chosen because it corresponded to the electric oscillations present in the natural environment. It should be mentioned that the subjects in the shielded room had access to 50 Hz lighting, air conditioning and other electrical equipment and would have been exposed to some tens of volts per metre and some tens of nanoteslas at this frequency. The effects of the 10 Hz field were regarded as beneficial.

The observations of Wever have been linked with similar observations on animals exposed to rather higher electromagnetic fields and attributed to their influence on production of the hormones melatonin and serotonin by the pineal gland (Groh et al, 1990; Wilson and Anderson, 1990). This production is strongly influenced by light levels. Stevens et al (1990) speculate that, because of the suppressive influence of melatonin on the growth of some neoplasms, this may be a route by which electromagnetic fields could promote the growth of cancers. However, it should be noted that Wever's observations only support an effect at 10 Hz not 50 Hz.

2.5 Birth outcome

Much of the evidence linking parental exposure or exposure *in utero* to electromagnetic fields has been reviewed by Silverman (1980, 1985) in relation to radiofrequencies and microwaves by Savitz (1985) and Knave and Törnqvist (1985) in relation to low frequencies. Such reviews have concluded that there is no convincing evidence for an increase in adverse reproductive outcome due to the exposures. The weaknesses of most published studies are that they do not contain any well-based assessments of exposure and often fail to address potentially confounding variables.

Reports of excessive incidences of Down's syndrome and of club foot in children whose fathers were thought to have been exposed to radar were not confirmed on closer examination (Sigler et al, 1965; Cohen et al, 1977; Burdeshaw and Shaffer, 1977). In the first of these studies the most significant correlation appears to be with the exposure of the mothers of the Down's syndrome children to ionising radiations for medical reasons rather than with the exposure of the fathers to radar. An earlier study of radar workers did not indicate any effect on fertility (Barron and Baraff, 1958). One case-control study of male physiotherapists who may have used shortwave, microwave, infrared and acoustic equipment for therapeutic purposes indicated no significant excess of congenital abnormalities in their children, although the number of such abnormalities was higher in both study and control populations than the reported rates for the general population (Logue et al, 1985).

Olsham et al (1989) investigated associations between children with Down's syndrome and parental occupation. Although they found significant associations between the father's employment as janitors, farm workers and mechanics, the odds ratio for those employed as electricians or electrical workers was 0.86 (95% confidence interval 0.51–1.46)

An association of paternal employment in the Swedish power industry has been made with

a reduction in the number of male children (Knave et al, 1979). On close examination the lower number of children and particularly of male children in the exposed group of 53 workers in comparison with their matched controls could be identified in a period before the high voltage exposures began. It was more likely to have been associated with differences in educational achievement and other social factors which differed between the exposed and control groups. One other report of an alteration in the expected sex ratio among children of power workers indicated a deficit of girls rather than of boys (Roberge, 1976).

Nordström et al (1983) also studied employees of Swedish power plants with a substantial study population of 542. Three groups of workers were defined, based on work in 400 kV substations (switchyards) (331 men), work with 220–380 kV transmission lines (145 men) and work with less than 130 kV lines (66 men). In addition to these occupational categories, which may be some indication of exposure, the men were also categorised in terms of their work with different electric potentials, ie as less than 70 kV, 130–200 kV and 400 kV. A reference or control group consisted of pregnancies when the father was not employed by the power company or was not working with potentials in excess of 380 V. Adverse pregnancy outcome was defined to include spontaneous abortion, still-birth, perinatal death and congenital malformation. Apart from congenital abnormalities there was little difference between the occupational categories, and for all pregnancy outcomes apparently no dependence on the work with different potentials. For congenital malformations the incidence was 10.1% among the 400 kV substation workers compared to 1.7% in the other groups and 4.0% in the reference group. The authors claim that age of mother and her use of alcohol, which were clearly possible confounding factors, did not affect this observation. This difference was statistically significant. A greater proportion of the switchyard workers' wives (26%) were more than 30 years of age at the time of birth compared with 11.5% for the reference group. This may be relevant. The authors suggest that the result should be treated with caution. It would justify further studies.

There have been reports of adverse effects on fetuses exposed *in utero* to diathermy radiation (Rubin and Erdman, 1959; Cocozza et al, 1960). However, the first is a single observation and the second is a report of the pregnancy outcomes in four women given diathermy treatment for only one of which was there an adverse outcome. This was a spontaneous abortion. Since this woman later gave birth to a normal child, who was also exposed *in utero*, the abortion may have been connected with the reason for the treatment rather than with the treatment itself. Some few thousand women appear to have been exposed to microwave heating to relieve labour pains, but there has been no reported follow-up for adverse effects on the children and the effect on labour appears to have been beneficial (Daels, 1976). A case-control study of female physiotherapists who gave birth to children with malformations suggested that the number of such children was higher among those who had used shortwave and ultrasound equipment during pregnancy (Källen et al, 1982). More reassuring is the observation already mentioned (Kolmodin-Hedman et al, 1988) that there was no significant difference in birth outcome in 305 plastic welder operators who must be some of the women most highly exposed to radiofrequency electromagnetic fields.

Wertheimer and Leeper (1986) examined pregnancy outcome in relation to the use of electrically heated over-blankets and water beds in the Denver area of Colorado. According to these authors the use of the blankets is associated with exposure to 60 Hz magnetic fields of average strength about 1.5 μT and about 0.4 μT in the case of water beds. The data are based on 1256 published birth announcements in which the parents were asked by telephone only about their use of blankets and water beds. Further data were obtained from hospital birth records not only on the

1256 announced births but also on 692 earlier births to the same parents. There were no differences as regards the proportions of low birth weight children to users of electric blankets and water beds in comparison with non-users. However, gestational periods in excess of the median for age of mother and birth order were more common for those children conceived to users between September and June. Electric heating of both blankets and beds is less in July and August. The non-users did not show this seasonal variation. The authors also considered spontaneous abortions taking place in the year previous to the conception of a live child. This revealed that the users suffered abortions more frequently in the months of September to March than the non-users. The authors hypothesised that these observations indicate an influence of magnetic fields on the early stages of pregnancy soon after conception. They recognised that the effect might be due to excessive bed temperatures in the winter. It is possible that there were social differences between the users and non-users which confound the results, but the authors claim that there were no educational differences between them. In a later study the same authors have attempted to separate the possible effect of higher bed temperatures from that which might be attributed to the magnetic field (Wertheimer and Leeper, 1989). In this study spontaneous abortions to mothers living in homes in Oregon with electric ceiling cable heating were compared with those to mothers not living in such homes. The ceiling cables were said to produce ambient magnetic fields in the room of 1 μT and electric fields of 10–50 V m^{-1}. Overall there was no difference in the proportion of spontaneous abortions in the 863 pregnancies in homes with ceiling heating to that in the 1158 pregnancies in homes with other forms of heating. Social differences were evident in that in the homes with ceiling heating the mothers tended to be younger and better educated. Although there was no overall difference in the proportion of spontaneous abortions, the users of ceiling cable heating (like the users of electric over-blankets and water beds) showed a greater seasonal variation in the proportion of spontaneous abortions than the non-users. Compared to the virtual lack of seasonal variation in the latter, the users had a higher proportion from September to December and a lower proportion from May to July. The authors claim that this supports their hypothesis about the influence of magnetic fields in the early stages of pregnancy. This claim is worth further investigation.

Some of the greatest disquiet in respect of electromagnetic fields and adverse pregnancy outcome has been expressed about the use of visual display units, VDUs (or visual display terminals, VDTs) (Lee, 1985). Work with these items of equipment may involve job stress, ergonomic and ophthalmological problems (Bergqvist, 1989), and the first two could be confounding factors in the study of associated pregnancy outcomes. The main electromagnetic fields produced by VDUs based on cathode-ray tubes for display originate from the line transformer and deflection coils required to move the electron beam across the screen. The frequency is in the range 15–30 kHz, but harmonics with frequencies of up to 220 kHz may be present. The main field strengths at a position 0.3 m from the screen are about 15 V m^{-1} for the electric field and 0.17 A m^{-1} (210 nT) for the magnetic field (Marriott and Stuchly, 1986). There will also be electric and magnetic fields from the 50 Hz power supplies that will not be very different from the fields produced by domestic electrical appliances. Field strengths of up to 10 V m^{-1} and 0.56 A m^{-1} (710 nT) are reported (Marriott and Stuchly, 1986).

Given that there have been widespread reports of and consequent awareness of a possible connection between VDU usage and spontaneous abortion, there is the possibility of recall bias in studies based on questionnaires. As pointed out in a review by Blackwell and Chang (1988) there is evidence from a study of exposures to anaesthetic gas that miscarriage is under-reported in non-exposed groups but well reported in those exposed.

Studies published prior to 1986 have been reviewed by Bergqvist and Knave (1988) and by Blackwell and Chang (1988). The latter authors point out that in the USA and the UK about 10 million VDUs are in use. About 50% of these are possibly used by women of childbearing age. Blackwell and Chang estimate that there are therefore some 20,000 groups where at least 10 women could become pregnant in 1 year. Since the naturally occurring pregnancy failure rate is about 15%, they suggest that there is a chance of about 29 clusters each year in which more than half the pregnancies end in failure. If this is correct, it is not surprising that both reviews essentially come to the conclusion that there is no evidence or indication that work with VDUs results in an increased risk of miscarriage or malformation. This does not mean that there is no risk, but that any such risk is too small to be detected by the published studies and much less than indicated by reported clusters.

Since these reviews there have been two published papers that provide a slightly different perspective (McDonald et al, 1988a; Goldhaber et al, 1988). The study by McDonald et al was part of a wider study in Canada of the influence of occupational factors on pregnancy in the period 1982–1984 based on interviews with 51,885 women who had just delivered and 4,127 who were treated for spontaneous abortion. As part of this study (McDonald et al, 1988b), increased relative risks (RR) of spontaneous abortion were found for women exposed to high levels of physical stress such as lifting (RR 1.32, significant at 1% level), physical effort (RR 1.26, significant at 1% level), standing (RR 1.09, significant at 5% level), long hours (RR 1.13, significant at 5% level) and shift work (RR 1.17, significant at 5% level) and also work with plastics, particularly polystyrene (RR 1.58, 90% confidence interval (CI) 1.02–2.35) (McDonald et al, 1988c). Table 9 summarises the findings of the study in relation to VDU usage. The relative risks have been adjusted for age, gravidity, previous miscarriage, ethnic group, educational level, smoking and alcohol usage. In this summary the only significantly raised risk at the 10% level is for spontaneous abortion in the current pregnancy. This relative risk (1.19) is slightly smaller and is of less significance than for some of the other factors given above. The authors believe that this finding is suspect, firstly, because the analysis by occupational group did not give a higher risk for those occupational groups with high VDU usage and, secondly, because much of the evidence for this increased risk was obtained from women working in the clerical sector for whom as a whole the spontaneous abortion risk was close to that for all working women (RR 1.01, 90% CI 0.95–1.08), and for whom there was evidence of under-reporting of VDU usage in those pregnancies that came to term. They did note an excess risk of congenital renal urinary defects which they thought may have been due to chance.

Although McDonald et al (1988a) express doubts about their finding of excess spontaneous abortion, they did find in an analysis by individual VDU usage that there appears to be a trend for the risk to increase with the level of this usage (Table 10). This was also found in the case-control study of Goldhaber (1988) of 1583 pregnancies. This shows a clear trend not only in relation to spontaneous abortion, but also as regards birth defects (Table 11) against VDU usage in the first trimester of pregnancy. The wide confidence interval on the odds ratios should be noted. As in the McDonald et al study, the odds ratios have been corrected, in this case for age, previous miscarriage or birth defect, race, education, smoking, alcohol use, occupation and gestational age at pregnancy diagnosis (for risk of miscarriage) and for hospital in which delivery took place (for birth defects). Goldhaber et al remark that their findings could reflect a recall bias. As already mentioned, McDonald et al are inclined to doubt the finding of a trend on the basis of a more detailed analysis of their data. In this analysis the observed to expected rates of spontaneous abortion were examined for 60 occupations divided into 8 groups according to the percentage of pregnancies in which VDUs

were used for more than 15 hours a week. These range from 21 occupations with 0% usage to 1 occupation with 60% usage. There was no trend within this grouping for the ratios to increase with the frequency of use above 15 hours a week. The overall relative risk across this grouping was 1.06 (90% CI 0.8–1.4), as shown in Table 9.

It may be concluded that the two later studies do not alter the conclusions drawn from earlier studies. There may be a slightly higher risk of spontaneous abortion associated with intensive use of VDUs during pregnancy, but this could be a result of higher job stress and postural problems rather than the exposure to the small electromagnetic fields. Rather higher risks are associated with physical stress in pregnancy. The absence of any indication of risk to women with much higher exposures to electromagnetic fields (Kolmodin-Hedman et al, 1988) tends to reinforce this conclusion.

Further reinforcement is provided by the study of Bryant and Love (1989) of 333 cases of spontaneous abortion in Calgary during the period from January 1984 to December 1985. Controls were postnatal women who were delivered of a healthy infant and prenatal women. No association between VDT usage and spontaneous abortion could be found in this careful study. It did provide evidence for recall bias in other studies in that the postnatal controls appeared to have less VDT usage than the prenatal controls. The study also identified a social difference between women who used VDTs and those who did not, although this did not affect the negative finding.

More recently, a well-conducted prospective study (Schnorr et al, 1991) investigated the risk of spontaneous abortion in a cohort of female telephone operators using VDUs during the first trimester of pregnancy and exposed to both extremely low frequency (ELF) and very low frequency (VLF) electromagnetic fields compared to telephone operators using equipment containing light-emitting diodes or neon glow tubes and exposed mostly to power frequency electromagnetic fields. The study group comprised 2430 women who were interviewed in detail; there were a total of 366 pregnancies in 323 operators using VDUs at any time during the first trimester of pregnancy during the study period compared to a total of 516 pregnancies in 407 operators using the other telephone equipment. Pregnancy outcome was classed as spontaneous abortion (fetal loss at 28 weeks gestation or earlier, stillbirth (fetal loss after 28 weeks gestation) or live birth. The abdominal region of the VDU operators was exposed to VLF (around 15 kHz) magnetic flux densities of about 5–20 nT; compared to non-VDU users exposure to background levels of around 2 nT, the abdomens of the non-VDU-users were also exposed to a wider range of power frequency magnetic flux densities (about 40–80 nT) compared to the VDU users (about 70–80 nT) exposed to 45–60 Hz fields.

The authors found no increase in the risk of spontaneous abortion associated with the occupational use of VDUs; in addition; the lack of excess risk was unaffected by the number of hours worked per week. Both groups of operators were well matched for a number of ergonomic factors related to the work and were similar in mean age, mean lifetime number of pregnancies, race, education, percentage currently employed at a company included in the study and mean years employed by a company included in the study. The authors also took into account in their analysis the possible confounding effect of recall differences in subject-reported data. Multiple logistic regression analysis was used to assess the effect of VDU use on the incidence of spontaneous abortion while controlling for the effects of other variables.

Although no effect of VDU use was found, the study did report an altered risk of spontaneous abortion for previously reported risk factors: alcohol consumption, smoking, the presence of a thyroid disorder and a history of spontaneous abortion. However, the study did not

address an association between spontaneous abortions and physical or psychological stress; these factors may have been similar in both groups of operators. In addition, both groups were exposed to similar levels of extremely low frequency magnetic fields which were in the lower part of the range of power frequency magnetic flux densities found in the home environment.

2.6 Cot deaths

There have been allegations in certain British newspapers that electromagnetic field exposures are involved in sudden infant deaths (cot deaths). These allegations appear to have been based on a hypothesis described in the *Journal of Alternative and Complementary Medicine* of October 1988 and attributed to R Coghill. This hypothesis of *cerebral morphogenetic radiation* postulates that electromagnetic radiation is produced by electrical oscillations between the left and right hemispheres of the brain and communicates with the DNA of the cells in the body, thus governing development. Interference with the communication by external electric fields is said to account for outbreaks of influenza, AIDs, and cot deaths. Attenuation of the communication by the skull is said to account for male pattern baldness. In terms of current biological and physical knowledge this hypothesis is extremely implausible.

There was an earlier suggestion that sudden infant deaths might be associated with electromagnetic fields (Eckert, 1976). This German report was based on 1 family in Philadelphia, USA, reported to have had 9 successive sudden infant deaths and who lived near a series of electric railway lines, and the distribution of 294 sudden infant deaths in Hamburg between 1961 and 1967. Much was made of the facts that there was a high incidence of deaths in public welfare residential camps, most of which were sited near to electric railways, and that the incidence appeared to be higher in the lower floors of multistorey apartment buildings. No data were given to show that these situations were in fact associated with higher electromagnetic fields nor was any attempt made to address the social factors which were likely to have been involved.

There appear to be many factors involved in sudden infant deaths (Brown, 1989; Milner and Ruggins, 1989) – it may be that exposure to electromagnetic fields is one of these factors, but there is no evidence for this possibility and it is at present pure speculation.

2.7 Allergies and electrophobia

There is some variation between individuals in their ability to perceive small electric currents and discharges. Typically, the perception levels of the most sensitive are about one-third of those to which the least sensitive react (Dalziel and Mansfield, 1950; Larkin et al, 1986). Whether there are individuals whose sensitivity is such that they suffer 'allergic' reactions to very small electromagnetic fields is for most scientists an open question. Certainly, there are individuals who believe that they suffer from such allergies and there are those who support them in their beliefs. However, the treatments described for such 'allergies' (Smith and Best, 1989) are such that one might suspect that their efficacy depends on the degree of belief of the patient and the charisma of the practitioner.

2.8 Summary and conclusions

Exposure to electromagnetic fields at levels below those advised by NRPB (NRPB, 1989) does not appear to have any effects on the general health of exposed persons or on their physical and mental performance. This conclusion is valid for extremely low frequency, radiofrequency and

microwave exposure. However, Nilsson et al (1989) observed changes in cerebrospinal fluid protein levels i dar mechanics and suggested that this might be due to the pulsed nature of their exposures. The one or two reports suggestive of changes in blood cell concentrations or function in those exposed to microwave radiations might be worth further study. It must also be remarked that although studies have not shown any excess of cataracts among workers exposed to comparatively high levels of microwave radiation, these studies do not appear to have included the elderly and retired. The possibility of an effect that has a long latency cannot be excluded.

Workers with radiofrequency heat sealers appear to be at risk for changes in the touch sensitivity of their hands, particularly when radiofrequency burns are a common feature of the work practice. They may also be subject to some general nervous disability.

One study has reported that there was no increase of adverse birth outcomes among women using radiofrequency heating for plastic welding. However, this is worth further study both because these women will be among the most highly exposed to electromagnetic fields and because of the observations made in relation to the use of electric over-blankets, electrically heated water beds, electric cable ceiling heating and VDUs. Although the evidence from electric blanket, water bed and ceiling heating usage has not shown an absolute increased risk of spontaneous abortion, there is an unexplained seasonal variation that did not apparently occur among non-users in the reported studies. The authors of the reports interpret this to mean that electromagnetic fields may influence the early stages of pregnancy. This may be relevant to a little evidence pointing to an influence of high usage of VDUs in early pregnancy on spontaneous abortion. However, high VDU usage may be associated with job stress and postural problems. Physical stress due to occupation seems to carry a greater risk to pregnancy outcome than either VDU usage or the use of electric over-blankets, water beds or electric cable ceiling heaters. If there is in fact a risk from the use of these appliances, which may or may not be due to the associated electromagnetic fields, it is almost certainly smaller than and outweighed by other environmental and work-related factors that increase the risk of spontaneous abortion. Although any link between pregnancy outcome and paternal exposure seems unlikely, the slight indication of such a link is, as those for maternal and *in-utero* exposure, worth further investigation in highly exposed groups.

3 Electromagnetic field exposure and cancer

There have been two main threads to reports of associations between cancer and electromagnetic field exposures. One thread comes from studies of cancer incidence in workers in occupations with electrical or electronic connections. The first in a series of such studies was by Milham (1982) and concerned a 30 year set of death registrations in Washington State, although Wertheimer and Leeper (1979) had noted earlier that the cancer rate appeared to be elevated among men who might be exposed to power frequency magnetic fields. Leukaemia mortality appeared to be raised across a range of such occupational groups and most prominently increased for the acute forms of leukaemia. The other thread comes from studies which rely upon the place of residence as the indicator of exposure. The first studies with this as the basis were those of Wertheimer and Leeper (1979, 1982), who found an association between domestic electric power lines near homes and childhood cancer. There have been a number of such studies since those of Milham and of Wertheimer and Leeper, with the majority concentrating on the occupational associations. There have been almost as many reviews of the studies as there are studies. Possibly some of the most comprehensive reviews are those of Knave and Törnqvist, 1985; Sheikh, 1986; Michaelson, 1987; Savitz and Calle, 1987; Coleman and Beral, 1988; Brown and Chattopadhyay, 1988; Repacholi, 1988; Creasey and Goldberg, 1988. These reviews all come to essentially the same conclusion, which is that the evidence for an association between electromagnetic fields and cancer is too tenuous to be conclusive but too consistent to be ignored.

Epidemiological findings are often criticised because they produce associations that are not necessarily causal. This is because in contrast to the majority of laboratory experiments there may be unrecognised or uncontrolled agents which are the true causal factors. However, the causal nature of observed associations is strengthened when the relative risk is large and statistically significant and, most particularly, when the relative risk increases with the level of exposure. As has been pointed out by Creasey and Goldberg (1988), some of the biological effects for which significant claims have been made are alleged to occur only at specific combinations of frequency and intensity. They are attributed to the magnetic rather than the electric component of the fields, possibly in an interaction with the earth's static geomagnetic field. If these are real effects and have any connection with carcinogenesis, there could be a complex dose relationship. For example, a much higher risk could be associated with a limited subset of exposure conditions and exposures at high intensities might carry a lower risk than those at lower intensities.

In general, it must be remarked that many of those in electrical or electronic occupations will not be exposed to any greater extent to electromagnetic fields than workers in other occupations. Many are employed in the assembly of components and others work on the repair of essentially 'dead' equipment. The derived risks, if genuine, from populations including the non-exposed will be underestimates of those to the genuinely exposed. Competing risks due to other possible agents such as solvents and metal fumes will also result in underestimates of any real risks from electromagnetic fields to these people.

3.1 Occupational studies

One of the most common approaches in the investigation of the possible links between cancer and occupational exposure to electromagnetic fields is to analyse national or regional statistics of cause of death or cancer registration. Usually this results in estimates of proportional mortality ratios, PMRs, or proportional incidence ratios, PIRs, whose interpretation is subject to

certain disadvantages (outlined in Appendix A) due to the confounding factor of occupational class. Where standard mortality ratios, SMRs, are given they rely upon the occupation descriptions recorded on death certificates, which are often the last occupation of the deceased, to provide the numerators, and census returns, to provide the denominators. (SMRs and PMRs are described in Appendix B.) The errors produced by this procedure in regard to British statistics have been described by Alderson (1972) and are pertinent to the later discussion in Section 3.1.4. An investigation of the occupation recorded at death by interviews with close family or friends disclosed a major discrepancy between the recorded and final occupation in about 10% of cases. Moreover, in about 28% of deaths there was a major discrepancy between the death certificate occupation and the principal occupation during life. Rather discouragingly Alderson remarks "... a search of the literature and discussion with colleagues failed to identify a single major hazard that has been discovered where the initial hunch for the enquiry was derived from the published occupational mortality data".

Another approach to the investigation of occupational exposures is the case-control study. In this type of study the distribution of occupations among those dying from a specific cause are compared with the distribution among a randomly chosen set of those dying from other causes. Alternatively, the distribution of occupations among hospital patients with the disease under investigation will be compared with that among patients with other diseases. However, the incidence of disease is likely to vary between the occupational classes in the same way as the mortality rates illustrated by Table A9 of Appendix A. Case-control studies, therefore, will reflect proportional ratios rather than the absolute rates of death and disease among occupations.

3.1.1 Leukaemia

Studies which have identified increased occupational risks to leukaemia among electronic and electrical workers have been reviewed by both Savitz and Calle (1987) and by Coleman and Beral (1988) as well as by others. The studies considered by these reviews and some additions are listed in Table 12. Both of the two reviews mentioned came to the same conclusion about the magnitude of the apparent risk. Using a weighted average of all the studies, Savitz and Calle determined the relative mortality risk to electrical and electronic workers from all leukaemias as 1.2 (95% confidence interval (CI) 1.1–1.3) and for acute myeloid leukaemia as 1.5 (95% CI 1.2–1.8). These are not of course separate risks, the all leukaemias risk includes that from acute myeloid leukaemia. Coleman and Beral obtained relative risks of 1.18 (95% CI 1.09–1.29) and 1.45 (95% CI 1.27–1.68) for all leukaemias and for acute myeloid leukaemia, respectively.

Two of the studies in Table 12 that are not included in the two reviews, are those of OPCS (1986) and Milham (1988). Their inclusion does not alter the weighted average risk values given above. The OPCS data are discussed in greater detail in Section 3.1.4. The study by Milham of amateur radio operators is of particular interest, since it avoids some of the doubts raised earlier about national and regional mortality statistics. By the use of records of licensed amateurs, which give dates of birth and addresses, Milham was able to derive standardised mortality ratios for those living on the west coast of the USA in a comparison with the mortality rates for the whole population. The SMR for all causes of death was 71 (95% CI 69–74). For all leukaemias it was 124 (95% CI 87–172), although this is raised it is not statistically significant. However, statistically significantly raised SMRs were obtained for acute myeloid leukaemia, 176 (95% CI 103–285), and for the related neoplasms of the lymphoid tissues (8th ICD code 202) and multiple myeloma (8th ICD code 203). Taken together, the SMR for these latter neoplasms was 162 (95% CI 117–218).

It is tempting to connect these observations with the possible exposure of these people to radiofrequencies in the course of their hobby, but it is notable that between 30% and 40% of them had electrical or electronics occupations. It is possible that these occupations and their hobby may involve exposure not only to power frequency electromagnetic fields, but also to solvents, polychlorinated biphenols and metal fumes, particularly when the assembly or construction of equipment is involved. The connection with assembly or repair work is seen also in the studies by Juutilainen et al (1988), Pearce et al (1989) and Gallagher et al (1990). In these studies the highest risks were associated with those workers involved with some sort of assembly and the lowest with general electricians and welders. This connection will be raised again in the discussion of brain tumours in Section 3.1.2.

One study (Barregård et al, 1985) found no increased risk of mortality or cancer incidence among men exposed to strong static magnetic fields ranging from 4 to 29 mT in a chloralkali plant in which they had worked for periods of more than 1 or 5 years when compared to men in the same Swedish county.

A study of Polish military personnel which was first reported in the March 1985 issue of *Microwave News* and later with slightly more detail (Szmigielski et al, 1988) indicated an excess risk of cancer to men exposed to microwaves. Neoplastic morbidity for the period 1971–1980 in these personnel was reported, of whom 3% were exposed to microwave and radiofrequency emissions. The actual numbers of deaths were not given, only the mortality rates, neither was the size of the studied population, but it was probably in total about 150,000 persons, both exposed and unexposed. The typical exposure was described as 4–8 hours per day at levels below 2 W m^{-2} with several minutes in the range 2–10 W m^{-2}. Occasional exposures at levels in the range 100–200 W m^{-2} are also reported. From this description an average annual integrated exposure of about 125 W m^{-2} days might be inferred. The risks of all forms of cancer, with the possible exception of lung cancer, seem to have been elevated in the exposed group . Particularly elevated risks were reported for lymphosarcomas and lymphomas, for acute myeloid leukaemia and for chronic lymphoid leukaemia. The relative risks for all forms of cancer by age and length of exposure that can be obtained from the data given are shown in Table 13, which is probably based on not more than about 30–40 deaths in the exposed group. It is possible to derive a coefficient for the increase in relative risk of about 0.001 per W m^{-2} day of exposure with an assumption of a multiplicative risk model and no latent period. If this were correct, it would imply that the acceptable exposure levels should be several orders of magnitude less than at present considered. However, there must be considerable statistical uncertainties attached to the relative risks given in Table 13 and the assumptions used in deriving such a coefficient are questionable. Indeed, Szmigielski et al express considerable caution about the results.

Exposures to organic solvents, and particularly to benzene, have been identified as leading to increased risks of all forms of leukaemia (Brandt, 1985, 1988). One of the most comprehensive reviews relating to benzene exposures is that of Austin et al (1988); exposure to this solvent has been specifically linked to an increase in acute myeloid leukaemia. For the studies quoted in the review the statistically significant relative risks at the 5% level range from 1.5 to 3.5 with SMRs for exposed persons in the range from 560 to 6637. The involvement of solvents in raised leukaemia risks is identified also in a study by Lindqvist et al (1987), who obtained relative risks of 4.9 (95% CI 2.2–12.1) for any exposure to solvents and 13.0 (95% CI 2.0–554) for exposure as a painter. A proportional mortality ratio for leukaemia of 178 (95% CI 81–392) for automobile mechanics (Schwartz, 1987) was also attributed to benzene and other organic solvents. Elevated

leukaemia risks have been described for abattoir workers (Pearce et al, 1988), farmers (Milham, 1971; Blair et al, 1985; Brownson and Rief, 1988), wood and paper mill workers (Schwartz, 1988; Flodin et al, 1988), and cooks, metal working machine operators, truck drivers and motor vehicle manufacturers (Howe and Lindsey, 1983). Exposures to DDT and fresh wood were particularly associated with an increased risk of chronic lymphatic leukaemia (Flodin et al, 1988).

In a published study of 5351 leukaemia cases occurring in a 19-year follow-up of Swedish men, who were employed in 1960, specific incidence rates (SIRs) for the different forms of leukaemia were significantly elevated at the 5% level for several occupations (Linet and Cartwright, 1988). The elevated SIRs were 500 in woodworkers for acute lymphatic leukaemia, 300 in cloth and pattern workers and 190 in electrical line workers for chronic lymphatic leukaemia, 210 in circular saw and plane operators for acute non-lymphocytic leukaemia (which includes acute myeloid leukaemia), and 150 in motor mechanics for chronic myeloid leukaemia. So many occupations are tested in this study that some of the elevated values must be due to chance.

3.1.2 Brain tumours

The first report which drew attention to a possible link between brain tumours and employment in electrical and electronic occupations was that of Lin et al (1985). In their report mention is made of earlier reports which had also indicated such a linkage, but which did not receive much attention (Milham, 1979; Robinette et al, 1980; Wrensch, 1984). Milham's study was of aluminium reduction plant workers based on the occupational mortality records of Washington State for 1950–1977. The primary concern was clearly with the effects of chemical fumes and dusts in the plants, although these workers are exposed to comparatively high magnetic fields. The SMR for neoplasms of the brain (7th ICD code 193) was unremarkable at 99 (95% CI 30–324), but Milham notes that there were five deaths due to benign neoplasms of the brain against 1.3 expected, giving an SMR of 391 (95% CI 157–972). The study of Robinette et al has already been discussed in Section 2 and is of American Navy personnel exposed to microwaves in the 1950s. Lin et al point out that 7.9% (16 of 202) of the deaths due to neoplasms were of the eye, brain and nervous system (8th ICD codes 190–192) compared with 3.8% expected from the statistics for all American males. The apparent relative proportional mortality ratio is therefore 208 (95% CI 126–344). Breakdown between the different exposure groups identified in Table 5 give relative PMRs for the low exposure group of 223 (95% CI 110–455), for the medium exposure group 202 (95% CI 83–494) and for the high exposure group 184 (95% CI 58–115). The loss of statistical significance due to the small numbers prevents any conclusion being drawn from these values. As noted in Section 2.1, there were no significant trends for cancer mortality to increase with the presumed level of exposure, although those for lymphatic and haematopoietic systems exhibited a marginally more positive trend than the others (Table 5). The case-control study of Wrensch was of 90 cases treated for brain tumours at the University of California. The cases were more likely than the controls to have had jobs as drivers, heavy machine operators or to have worked outdoors. Both cases and controls were equally likely to have had jobs involving exposure to extremely low frequency electromagnetic fields, but for the cases these jobs were more often the lifetime or predominating job.

The study of Lin et al was principally a case-control study based on death certificates from the state of Maryland in the period 1969–1982 of 951 cases distributed among the 8th ICD codes 191, 192.9, 225.0 and 238.1 and the 9th ICD codes 191, 225 and 239.6. Of the cases, 519 were gliomas or astrocytomas and the remaining 432 were non-specified brain tumours. For both these

categories the observed cases appeared to be in excess of expectation for electrical and electronic engineers and technicians, and for electric and telephone line and servicemen. (In the USA power supply lines and telephone lines are often mounted on the same poles.) However, only for gliomas and astrocytomas did there appear to be a significant trend of association with the attributed level of exposure (Table 14).

In the same year that Lin et al published their paper, Milham (1985b) published a further study of the mortality of workers exposed to electromagnetic fields in Washington State which covered the period 1950–1982. It seems likely that this included deaths from his earlier publications already mentioned. In addition to the significantly raised PMRs for the leukaemias (Table 12), he also obtained a PMR for malignant neoplasms of the brain of 123 (95% CI 100–151) for workers occupationally exposed to electromagnetic fields. Significantly elevated PMRs were also obtained for neoplasms of the pancreas, lung and for lymphomas. In Table 15, these are distributed among those occupations which were believed to involve only exposure to electromagnetic fields and those which also involved exposures to other agents, particularly metal and chemical vapours and fumes. In the case of cancer of the brain, the elevated PMR appears to be confined to those whose exposures included other agents.

Another American study which found an association between electromagnetic fields and brain tumours was that of Speers et al (1988). This was based on an area of Texas in which the SMRs for neoplasms of the brain (8th ICD code 191) seemed unduly elevated. Using the same groupings of attributable exposure as Lin et al (1985), a significant trend was observed (Table 16), although the overall odds ratio quoted for electrical and electronic workers was not significantly raised at 2.11 (95% CI 0.77–5.81). Higher and significant odds ratios were obtained for utility workers of 13.10 (95% CI 1.33–128.87) and employees in the trucking industry of 6.65 (95% CI 1.05–42.19), for whom no particular association with electromagnetic fields was claimed.

Two reports from Sweden did not find any excess of brain tumours in electrical workers. The most comprehensive of these studies was by McLaughlin et al (1987), which linked cancer incidence in the period 1961–1979 with the 1960 Swedish census information on occupation. Significantly raised standard incidence rates, SIRs, at the 5% level were observed for professional, technical and related workers (110), service workers (110), agricultural scientists (270), dentists (210), welders and metal cutters (140), and unspecified glass, porcelain or ceramic workers (220). For electrical and electronic workers the SIR was 90. Specific industries which had statistically significant high SIRs were cellulose plants (160), other petroleum and coal industries (260), brick and tile making (180), engineering and architects offices (180), and coffee shops (320). The report by Törnqvist et al (1986) covered the same period, and presumably the same population, but was specific to workers in the electric power industry. They could find no elevated risks of leukaemia or brain tumours in power linemen or power station operators, although there was a tendency towards an excess risk of cancer of the kidney and other organs.

Thomas et al (1987) made a detailed study of the brain tumour risk among men with electrical and electronics jobs, with particular emphasis on their possible exposures to microwaves and radiofrequencies. However, occupations considered to involve exposures to these radiations included those of electrical linemen, aluminium production workers, welders and motion picture projectionists, whose exposure to electromagnetic fields is more likely to be of extra low frequency electric power fields. The study population consisted of men in the north-eastern part of the USA dying of brain and other central nervous system tumours in 1979, 1980 and 1981. Controls were men dying of other causes and matched for age, year of death and place of residence. It is claimed

that the calculated relative risks are adjusted for the confounding influence of educational attainment. Table 17 gives these relative risks according to whether or not the exposure occurred in an electrical or electronics job. As may be seen, the excess risk appears to be confined to those who were exposed in one of these occupations. A further breakdown showed a statistically significant trend for the risk to increase with the duration of exposure. The relative risk was higher for men involved in the manufacture and repair of electronics equipment (3.9, 95% CI 1.6–9.9) than for electrical tradesmen (1.9, 95% CI 0.9–3.8). These observations led the authors to the conclusion that simple exposure to microwaves and radiofrequencies was not responsible for the excess risks and that additional exposures to soldering fumes and solvents might be involved.

Excess risks of brain tumours have been associated with other occupations, in particular with farming (Blair et al, 1985) and the chemical and petroleum industries. Howe and Lindsey (1983) found excess risks for Canadian men in highway and bridge maintenance and the manufacture of household furniture. In a conference on brain tumours in the chemical industry (Selikoff and Hammond, 1982) during which brain tumours were associated with exposure to vinyl chloride and possibly to petroleum products, electrical occupations were mentioned in two papers. The first of these by Mancuso (1982) highlighted excess brain tumour rates during 1939 in men employed in the rubber industry and in the manufacture of electrical machinery, and in the manufacture of paper and allied products. At that time rubber was used extensively for electrical insulation. Mancuso, himself, suggested that the association was strongest with exposures to rubber production occurring mainly in the period from 1914 to 1935 and possibly with exposure to carbon disulphide and benzene in the production process. In the second paper by Bond et al (1982) it was indicated that brain tumours among employees of Dow Corning in Texas were associated with work in a magnesium production plant which involved electromagnetic field exposures. The authors go on to suggest that this indication was not supported by the absence of any latency effect. In the same conference Alderson and Rushton (1982) pointed to an absence of any excess brain tumour risk among employees of UK oil refineries.

The studies associating brain tumours with electrical and electronics occupations are listed in Table 18. The weighted average relative risk over all these studies is 1.33 (95% CI 1.19–1.49).

3.1.3 Malignant melanoma and other cancers

In 1983 Vågerö and Olin published a study of cancer incidence in the Swedish electronics industry based on cancer cases reported to the Swedish Cancer Environment Register in the period 1961–1973. They identified slightly raised relative risks for all cancers (men, 1.15 95% CI 1.10–1.20; women, 1.08 95% CI 1.01–1.15) and rather more elevated risks of pharyngeal cancer (7th ICD code 145) and lung cancer (7th ICD code 162) of 2.30 (95% CI 1.11–4.97) and 1.52 (95% CI 1.35–1.72), respectively. Although not specifically identified in their text, the risk of malignant melanoma (7th ICD code 190) was given as 1.35 (95% CI 1.05–1.76).

In a subsequent study (Vågerö et al, 1985) the cancer incidence in one Swedish company producing telecommunications equipment was studied for the period 1958–1979. For male employees statistically significantly elevated SMRs were found for malignant melanoma and Brill-Symmon's disease of 250 (95% CI 40–490) and 1760 (95% CI 210–6380). For female employees the only elevated SMR was for neoplasms of the small intestine (1600, 95% CI 180–5780). The malignant melanoma risk appeared to be rather more associated with work that involved soldering which gave an SMR of 390 (95% CI 140–150) compared with 280 (95% CI 130–510) for any type of work.

The same group of authors also studied the mortality for the period 1930–1979 of Swedish electrical engineers who graduated between 1930 and 1959. The only elevated SMR for this group of 1254 men was for malignant melanoma of 320 (95% CI 70–940); but the small numbers prevent this value from reaching statistical significance. It must be presumed that the three Swedish studies mentioned all relate to some extent to the same set of data and cannot be regarded as entirely independent.

A raised risk of malignant melanoma was also found among the male workers of a Canadian telecommunications company. During the period 1976–1983, 10 cases occurred when 3.68 would have been expected. Of these 10 cases, 3 were categorised as office workers, 1 was a salesman and 1 a computer specialist. It is unlikely that these had any remarkable exposure to electromagnetic fields. The highest risks appeared to be associated with first employment by the company within 20 years of contracting the disease.

Swerdlow (1983) drew attention to the high proportional registration rates (PRR) for eye cancer in electrical and electronics workers. These were based on data for England and Wales for the period 1962–1977 for persons over the age of 15 years. Equally high rates occurred in the occupational orders of administrators and managers and in professional, technical workers and artists. These two occupational orders were thought to reflect the general tendency for higher PRR values in the higher occupational classes to which the electrical and electronics workers were not considered to belong.

Elevated risks of cancers of the respiratory tract and lung have been indicated in three of the reports already mentioned (Robinette et al, 1980; Vågerö and Olin, 1983; Milham, 1985). In the first of these the higher risk was for the more highly exposed of the three groups (see Table 5). It is noticeable that this group was the one consisting of those American Navy personnel concerned with the repair and servicing of equipment. In Milham's study the higher lung cancer risk was also associated with those workers for whom there was exposure to solvents and metal fumes in addition to the electromagnetic field exposure (Table 15). The Swedish study provides no information on the possibility of other exposures. If the elevated cancer risks in electrical and electronic occupations are related to exposures to solvents and metal fumes, it would not be surprising to observe some elevation of the lung cancer risks among workers in these occupations. Smoking is a notorious confounding factor for this cancer, however.

For other cancers there have been only isolated reports of elevated risks for electrical and electronic workers. Neoplasms of the pancreas and lymphomas have been identified by Milham (1985b) Table 15, and pharyngeal cancer by Vågerö and Olin (1983). In view of the close connection with the leukaemias it is surprising that other cancers of the immunological system such as multiple myeloma and the lymphomas do not appear to have been reported as elevated among electrical and electronics workers apart from the reports of Milham (1985b, 1988) which may contain some overlap of data. One study of 435,425 American Navy enlisted men in the period 1974–1983 (Garland et al, 1988), in which 68 cases of non-Hodgkin's lymphomas occurred, did not indicate any generally excess risk among electrical and electronics occupations. Although based on only two cases, a standardised incidence ratio of 1.7 (95% CI 0.2–6.1) was observed for internal communications electricians.

It has been hypothesised that the increase in mortality rates in recent decades from breast cancer (Appendix A, Table A4), among women over 55 years is linked to electric power use (Stevens, 1987). This hypothesis is based on some evidence from animals that exposure to electric fields decreases melatonin production and this is linked in the hypothesis to oestrogen and prolactin

production which increases cell turnover in the breast and breast cancer risks. (See also Section 2.4.) However, melatonin production is strongly influenced by exposure to light, and is reduced when light is incident on the retina. Since there is an obvious association between the use of artificial light and the use of electric power this is a confounding factor which could exert a stronger influence than any effect of electromagnetic fields.

3.1.4 Cancer mortality among British electrical and electronics workers

Two reports of increased leukaemia risks have been based on data derived from the decennial publications on occupational mortality issued by the British Office of Population Censuses and Surveys (McDowall, 1983; OPCS, 1986). These are listed in Table 12. The OPCS publications give SMR and PMR values for the age groups 20–64 years and PMRs for the age groups 65–74 years by occupation. The numerators are derived from death certificates and the denominators for SMRs from the decennial census data for the United Kingdom. Unfortunately, as remarked earlier, it is not always certain that the two sources of data apply to exactly the same occupational populations. In particular, a rather wide range of work experience and educational attainment might be covered by the description 'engineer'. The OPCS publications specifically identify some occupations including electrical where the SMR values are doubtful, but indicate that it is not certain that the data for other occupations are any more reliable.

McDowall's study considered mortality data for England and Wales for the period 1970–1972 (OPCS, 1978) and a 10% sample of the population from the 1971 census. For a range of electrical and electronic occupations, PMR for all leukaemias was 98 (95% CI 79–21) for the ages 15–74 years, and for acute myeloid leukaemia 104 (95% CI 73–148). For specific occupations a high PMR of 249 (95% CI 110–565) was derived for telegraph and radio operators for all leukaemias. High PMRs for acute myeloid leukaemia were obtained for electrical engineers (so described) (231, 95% CI 118–451), telegraph and radio operators (241, 95% CI 58–996), electrical engineers (197, 95% CI 48–809), and electronic engineers (professional) (305, 95% CI 111–837). Only the first and last of these are statistically significant. McDowall also carried out a case-control study based on acute myeloid leukaemia deaths reported in 1973 using as controls deaths from other causes randomly selected, but matched for age to the leukaemia deaths. This does not avoid the confounding factor of occupational class. The derived relative risk for all occupations connected with the electrical or telecommunications industries was 2.3 (95% CI 1.4–3.7) and for those connected only with electrical industries 2.1 (95% CI 1.3–3.6). There is essentially no difference between these two relative risks.

The 1986 OPCS report in a commentary on its data drew attention to an elevated SMR of 202 for electrical and electronic engineers for all leukaemias that was statistically significant at the 1% level for the age group 20–64 years, and an elevated SMR for acute myeloid leukaemia of 155 for electricians, fitters, plant operators, etc, that was statistically significant at the 5% level for the same age group. Unlike the earlier OPCS reports, the 1986 report covered the whole of Great Britain instead of only England and Wales. Mortality data were for the years 1979–1980 and 1982–1983 and the occupational data from the 1981 census, which sought rather more detail about occupation than earlier censuses.

Data from the 1986 OPCS report are analysed further here. The occupational groups for men who may have some extra exposure to electromagnetic fields have been separated into those who may in addition be exposed to solvents and metal fumes that are particularly connected with the manufacture, repair and assembly of electrical and electronic equipment and those who are less

likely to have been exposed to t[illegible]her agents, although they may have been exposed to other solvents and fumes. The two groups are:

Group I Occupations with possible electromagnetic field exposures and other exposures associated with manufacture, repair and assembly of electronic equipment

Code 027	Electrical and electronics engineers
Code 120	Production fitters, electricians, electricity power plant operators, switchboard attendants
Code 122	Telephone fitters, cable joiners, linesmen
Code 123	Radio, TV and electronic maintenance fitters and mechanics

Group II Occupations with possible electromagnetic field exposures, but unlikely to have been exposed to the solvents and metal fumes associated with the manufacture, repair and assembly of electrical and electronic equipment (they may have been exposed to other solvents and fumes)

Code 022.02	Sound and vision equipment operators
Code 051	Telephonists, radio and telegraph operators
Code 110.03	Electroplaters
Code 128	Welders

Apart from the difference as regards other exposures, these groups may differ in other ways. Group I workers on the whole might be expected to have a higher level of training and education. For comparison another group was selected with a comparable background of technical training and occupation, but with no obvious electromagnetic field exposures, composed as follows.

Group III Engineers and technologists with no specific connection with electromagnetic fields

Code 025	Civil, structural, municipal, mining and quarrying engineers
Code 026	Mechanical and aeronautical engineers
Code 028	Engineers and technologists not elsewhere classified
Code 030	Laboratory and engineering technicians, technician engineers

The SMRs for these groups are presented in Table 19 for males aged 20–64 years for the major causes of death, ie all neoplasms and diseases of the circulatory system, and for the specific neoplastic diseases of malignant melanoma, of the brain and for all leukaemias. In addition, the SMRs for the main leukaemias of adults, acute myeloid leukaemia and chronic lymphatic leukaemia, are given. Similar data are given in Table 20 for males aged 65–74 years – for this age group only PMRs are supplied by OPCS.

As may be seen, the SMR values for all the disease categories other than chronic lymphatic leukaemia listed are statistically elevated at the 5% level for the 20–64 years age group of Group I workers, the PMR values for the 65–74 years age group are unremarkable. In contrast, for the Group III workers the major causes of death have significantly low SMR values in the 20–64 years age group, although the PMR for all neoplasms is significantly high for the 65–74 years age group. Group II workers are similar to Group I in that the SMRs for the two major causes of death are elevated to a statistically significant degree; the other causes have unremarkable values. Aside from the possibility that the mortality rates for workers in the Group I and II occupations are elevated for all the major causes of death, the patterns of SMRs and PMRs in Table 19 and 20, when compared with the patterns for the different occupational classes shown in Table A9 of Appendix A, suggest

that there may well be problems with the numerators and denominators from which these values are derived. To further illuminate these data the relative standardised mortality ratios (RSMRs) have been calculated and are shown in the tables. These RSMR values are defined as

$$RSMR = (R1/R2) \times 100$$

where $$R1 = \frac{\text{Observed deaths from the specific cause}}{\text{Expected deaths from national statistics}}$$

and $$R2 = \frac{\text{Observed deaths from all neoplasms and diseases of the circulatory system less deaths from the other diseases listed}}{\text{Expected deaths from all neoplasms and diseases of the circulatory system less expected deaths from other diseases}}$$

In this comparison the RSMR values for the Group III workers are all significantly elevated at the 5% level for the 20–64 years age group, and none is significantly elevated for Groups I and II. It seems possible, therefore, that the apparently elevated SMR and PMR values for electrical and electronic workers for leukaemias, neoplasms of the brain and malignant melanoma are an artefact of the mortality data for Great Britain. In view of the data for Group III and those for the occupational classes in Table A9 of Appendix A, it is also likely that occupational class is a strong confounding factor in the elevated risks for the workers derived by studies in other countries, and that to some extent they reflect a lower mortality from other diseases rather than higher mortalities for the specific diseases for which elevated risks have been claimed.

Because of the suggestion that if the elevated risks to electrical and electronics workers are real then exposures to solvents and metal fumes may also be implicated, similar data to those in Tables 19 and 20 are given in Table 21 for plumbing, heating and ventilating fitters, and gas fitters who might be exposed to lead fumes from soldering, and in Table 22 for chemical, gas and petroleum plant operators, rubber and plastics workers who might be exposed to solvents.

It is difficult to draw any firm conclusions from these data because of the lack of statistical significance for the relative standardised mortality ratios, other than those for the Group III workers of general engineers and technicians in the 20–64 years age group. On the whole for this age group, however, and in particular for acute myeloid leukaemia, the values tend to be lowest for the Group II category of workers not concerned with assembly or repair of equipment and for the plumbers, heating and ventilating fitters, and gas fitters. Highest values occur in Groups I and III, with the chemical, petroleum, rubber and plastics workers in an intermediate position. In the older 65–74 years age group, for which chronic lymphatic leukaemia is more significant, the highest values tend to occur among the plumbing, heating and ventilating fitters, gas fitters and Group I workers; the lowest values tend to occur among chemical, petroleum, rubber and plastics workers and Group II workers. *Thus, Group II electrical and electronic workers tend to have the lowest relative standardised mortality ratios for the neoplasms in question at all ages and Group I workers tend towards the highest.*

Of the observed leukaemia deaths in Group II, 73% are of welders. Welders as an occupational group are exposed to the highest levels of low frequency magnetic fields of up to 100–200 μT (Stern, 1987; Stuchley and Lecuer, 1989). In a world-wide survey Stern (1987) found their relative risk of all leukaemias to be 0.86 (95% CI 0.67–1.06) and for acute leukaemias 0.85

(95% CI 0.55–1.15). This reinforces the above findings. Stern did find that welders had an elevated risk of lung cancer that is attributed to welding fumes.

These observations would suggest that if there is any risk associated with exposure to electromagnetic fields it arises only in association with other agents. The data for the plumbing, heating and ventilating fitters, and gas fitters suggest that if lead soldering fumes are implicated then there is a long latency. It could be hypothesised that electromagnetic field exposures shorten this latency. There is no suggestion in the data that exposure to the general chemicals of the rubber, petroleum, plastics and chemical industries produces any increased risks from the neoplasms of interest. This is supported by a recent study of rubber workers for the period 1946–85 (Sorahan et al, 1989). This must diminish, although it does not eliminate, the possibility that any excess risk of these neoplasms for electrical and electronics workers is due to the use of solvents in assembly and repair work.

In the case of malignant melanoma the values for the 20–64 years age group appear to reflect the very strong gradient with occupational class exhibited in Tables A9 and Table A10 of Appendix A both for this and for the 65–74 years age group. However, this pattern does not persist here into the older age group. Plumbing, heating and ventilating fitters, and gas fitters appear to have one of the highest relative proportional mortality ratios as they do for most of the other neoplasms.

3.1.5 Conclusions on the risks to workers from exposure to electromagnetic fields

It is by no means certain that there is a real additional cancer risk to electrical and electronic workers or, if it is real, whether exposures to solvents and metal fumes arising in the manufacture, repair and assembly of equipment are involved. By reference to the current mortality rates among workers in the 20–64 years age group (Tables A5, A6 and A9 of Appendix A) and taking the derived relative risks at their face value, the annual average extra risk of leukaemia (including acute myeloid leukaemia) for those employed in electrical and electronic occupations is about 1×10^{-5} and for neoplasms of the brain about 2×10^{-5}. The total extra annual risk for these cancers is therefore about 3×10^{-5}. This is likely to be a considerable overestimate of the possible risk to the average worker because of the confounding factor of occupational class. If the risk is real and due to electromagnetic field exposure it is impossible to draw any conclusion about the risk to the more highly exposed or those exposed to specific frequencies.

Interestingly, in contrast to much of the expressed concern, it is neoplasm of the brain rather than leukaemia which involves the highest absolute excess risk.

No attempt has been made to estimate any excess absolute risk for malignant melanoma of the skin because in the literature the risk appears to be confined to Swedish workers and elsewhere to workers who are only peripherally concerned with electrical or electronics work; also there is a strong occupational class factor in the risk and it is associated with sun-bathing.

An annual risk of 3×10^{-5} is within the levels regarded as acceptable for occupational risks (Royal Society, 1983), although above a level regarded as acceptable for members of the public. It must be pointed out that the additional risks of leukaemia and neoplasms of the brain appear to be exceeded in a number of other occupations which have no specific association with electromagnetic fields. Other than for exposures to benzene and vinylchloride, the risks for these occupations are probably no better established than they are for electrical and electronics workers.

3.2 Residential population studies

In discussing their finding that childhood cancer seemed to be increased among the population who lived or had lived in homes in close proximity to high current configurations (HCCs) of domestic electricity supply cables, Wertheimer and Leeper (1979) in their seminal paper suggested four possibilities for the association:

(a) both cancer and HCCs may be associated with some third factor giving a spurious correlation, although they could find no indication of such a factor,

(b) the magnetic fields produced by the currents in the cables caused cancer,

(c) the HCCs might have an indirect effect on the distribution of some ambient environmental carcinogen (one suggestion being that return currents in the water supply pipes increased the levels of lead in the drinking water),

(d) alternating magnetic fields might affect the development of cancer indirectly, possibly by some effects on the immune system or on cell proliferation.

It seems clear from their scoring system for the cables or wires and other details in their paper, that the study was conceived around a basic hypothesis that linked magnetic fields to childhood cancer. It is pertinent to enquire to what extent their scoring system and others based on distance from wire and cable configurations reflect the magnetic field levels, particularly since it is the magnetic field aspect that has dominated most subsequent studies.

To a greater extent than in the UK, homes in the USA are supplied with electricity by a system of overhead cables. These consist of primary cables carrying current at a potential of between 4 and 13 kV to pole-mounted transformers. Secondary cables from the transformers with potentials of 240 V between phases and 110 V to a neutral line distribute the current to houses at the domestic potential. Both the primary and secondary cables vary in thickness according to the possible power load and the date of installation. In general, the thicker cables will be carrying higher currents and generating higher magnetic fields. In some places in the USA the cables are buried, as they are more usually in the UK. Primary cables in the UK usually convey the current at a potential of 11 kV and supply transformers at street level which reduces the potential to 415 V between phases or 230 V to neutral. Buried cables are twisted together in a helical structure with supply and return currents flowing in opposite directions. The result is that there is some cancellation of the magnetic fields produced by the different wires in the cables and these fields are rather less than those produced by equivalent separated wires used in overhead cables.

To a greater or lesser extent, depending on the type and age of the domestic wiring in a home, the return currents may flow not through the wires but through the water pipes and ground. Where these unbalanced return currents exist, there may be higher magnetic fields than where the supply and return currents are more equally balanced in the cables.

In addition to the magnetic fields the presence of electric cables results in electric fields within the home. Unlike the magnetic fields these are very little influenced by the electric current and power consumption; also they do not vary between homes or within the home to the same extent as the magnetic fields. Although associations between cancer risk and magnetic fields have been indicated, this has not been the case for electric fields. There will be no further discussion of electric fields here. Further details can be found in the reports and papers of Savitz and his co-workers (Savitz, 1987, 1988; Savitz et al, 1988).

Wertheimer and Leeper devised a scoring system based on the number, thickness and

distance from a home of primary and secondary overhead cables. In their first paper in 1979 they distinguished only two categories: high current configurations, HCCs, and low current configurations, LCCs. In their second paper in 1982 this system was refined to provide four categories:

(a) very high current configurations, VHCCs,
(b) ordinary high current configurations, OHCCs,
(c) ordinary low current configurations, OLCCs,
(d) end pole.

The last consisted of ho' ıes at the end of a secondary distribution system with no nearby primary cable. In the study of ..avitz (1987) this was termed a very low current configuration, VLCC. Savitz and his co-workers added a fifth category, 'buried'. In the Savitz study these wire code categories were supplemented by measurements of magnetic flux densities and electric fields in several rooms of a number of homes. The magnetic flux density measurements were averaged for each home. The medians and ranges of these average magnetic flux densities for each wire code configuration are shown in Figure 5. Although the difference between the two lowest categories is minimal, the median values clearly increase with the wiring category despite the considerable overlap of the ranges. If cancer risks do increase with the level of magnetic field exposure, any risks derived from the system of categorisation are likely to be underestimated due to the overlap.

In a study in Sweden where, as in the UK, the domestic supply potential is 230 V at a frequency of 50 Hz and the supply cables tend to be buried, Tomenius (1986) measured magnetic flux densities outside the front doors of homes. Figure 6 shows a plot of the medians and ranges for these measurements as a function of distance from a 200 kV power transmission line. There is again a considerable overlap of the ranges.

It may be concluded that while distance from electrical distribution and transmission cables and the nature of the cables may be a predominating influence on the magnetic flux density within a home in many cases, details of the supply to the home and the wiring within it produce a degree of variation that will confound any attempt to quantify risks due to magnetic field exposures based solely on distribution line configurations. The effect of the confounding would be an underestimation of any real risks. One advantage of wiring codes is that it is possible to obtain a rough estimate of the magnetic flux density levels in a home without having to gain entry and make direct measurements. This, however, is mainly applicable to situations where the supply is carried in overhead cables and not where it is carried in buried cables. A study by Kaune et al (1987) has shown that for overhead cables it is possible to derive better indices of the magnetic flux density than those used by Wertheimer and Leeper.

The greater use of buried supply cables combined with the higher domestic supply voltage, which requires smaller electric currents for the same power consumption, will, in general, result in lower magnetic flux densities in British homes compared to American ones. There have been very few extensive studies in either country, but the few studies performed have tended to confirm the expectation. Table 23 compares the minimum, median and maximum fields reported by Myers et al (1985) for British homes with those reported by Savitz (1987). The measurements reported by Savitz are for minimum and maximum use of electrical appliances in the home, ie low and high power consumption. Since many electrical appliances are only used intermittently and the magnetic flux density decreases rather rapidly with distance from most of this equipment, the magnetic flux densities under conditions of low power consumption are regarded by some as more representative

of the average exposure. Some of the measurements by Myers relate to the proximity of a high potential power transmission line (in excess of 132 kV).

3.2.1 American studies

In their 1979 paper Wertheimer and Leeper studied 344 persons under the age of 19 years who died of cancer in the greater Denver area of Colorado between 1950 and 1973 and who were also born in that area. These were matched for date of birth by a similar number of controls. For cases and controls an attempt was made to obtain both the birth and the 'death' address, the latter being the address at the time when the case died. Of the cases, 109 died at their birth address; of the controls 128 were still living at their birth address when their matched case died. There were 272 birth address and 328 death addresses for both cases and controls. Table 24 summarises the odds ratios for the HCC addresses of cases and controls with respect to the LCC addresses. The analysis is given for stable addresses, ie those cases and controls with the same addresses at birth and 'death', and for birth and 'death' addresses. For all the combinations the odds ratios are significant at the 5% level. The breakdown by the major types of childhood cancer in Table 25 shows significantly elevated odds ratios for leukaemia and cancers of the nervous system, and not significantly elevated values, possibly because of small numbers, for lymphomas and other cancers. Apart from the other cancers, the choice of birth or 'death' address did not produce much change in the odds ratios. They did not appear to be influenced by age of onset when a comparison was made of onset between 0 and 5 years and between 6 and 18 years, by urban or suburban living, or by the father's occupational class. The odds ratio for living near a traffic route in comparison with elsewhere was 1.36 (95% confidence interval (CI) 0.96–2.03) and is not significant. The odds ratio for males 2.65 (95% CI 1.71–4.11) seemed marginally higher than for females, 2.11 (95% CI 1.33–3.36).

Fulton et al in 1980 published a study of childhood leukaemia cases, not necessarily deaths, whose age at onset was less than 20 years in the period 1964–1978 in Rhode Island. They used a rather different scoring system from that of Wertheimer and Leeper, although also based on distances from wiring configurations. There were 119 cases and 240 controls matched to the cases for date of birth. Unfortunately, they chose to compare birth addresses for controls with the various addresses at which the cases had lived, which could confuse an interpretation of the data. Their results, Table 26, do not indicate any higher risk associated with their 'Very high' category. Although Wertheimer and Leeper had shown little differences between comparisons of birth and 'death' addresses, they suggested that Fulton et al's method of comparison biases the controls towards high current configurations (Wertheimer and Leeper, 1980) thus reducing the derived risk. By selecting from Fulton et al's data only those cases and controls where the case was less than 8 years of age and using addresses occupied by both cases and controls between December 1957 and December 1978, they produced a set of data yielding an odds ratio for HCC cases of 1.67 (95% CI 1.02–2.67). Such selective manipulation of the data is slightly suspect, however.

In 1982 Wertheimer and Leeper reported another study in the Denver area, this time of adult cancers. Cases consisted of persons who had died of cancer in the period 1968–1975 and persons who in 1979 had survived at least 5 years from a diagnosis of cancer. There was some randomised selection of those cases whose age was in excess of 62 years and of those who had lung cancer. The purpose of this selection was to keep the sample to a manageable size – 515 cases and their controls came from two small towns in Colorado, Boulder and Longmont, and 664 from the city of Denver and its suburbs, a total of 1179 cases and matched controls. For Boulder and

Longmont the controls were a random sample of live subjects from the two towns matched with the cases for age, sex and socioeconomic status of the home neighbourhood. In the case of Denver city and its suburbs, the controls were randomly selected addresses within two blocks of the case address. This last method of selecting control addresses may have produced some overmatching, as the authors admit, and could have reduced any differences between case and control addresses. Wertheimer and Leeper derived wiring codes for all the addresses. The resulting data are given in Table 27, together with the mean magnetic flux densities obtained for these wiring codes by Savitz (1987). In the two towns and in the Denver suburbs there are significant trends of increasing relative risk with wiring configuration, but not for the city of Denver. Combining all the data produces a significantly increasing trend and an estimate for the increase in relative risk per nanotesla of 0.36% (95% CI 0.16–0.55).

Wertheimer and Leeper derived another parameter for the examination of the relative risks. This they call the C-ratio, which is defined as

$$\text{C-ratio} = \frac{\text{Number of matched pairs in which the case address has a higher wiring code} \times 100}{\text{Number of matched pairs in which the control address has a higher wiring code}}$$

While this statistic might be an indicator of increased risk, it is difficult to use for the purpose of quantification. In view of the range of magnetic flux densities corresponding to any wiring code (Figure 5), there is no guarantee, for example, that a higher code for a case in respect to its control involves a higher magnetic field exposure.

Using the C-ratio, Wertheimer and Leeper point out that lower values are obtained for those who live in Denver city and higher values for those who live either in the small towns or in the Denver suburbs. However, this is quite evident from the data in Table 27. This tendency is used by Wertheimer and Leeper to argue that the greater possibility for exposure to urban-industrial carcinogens in the central Denver area produces competing and confounding risks, but it may well be that in the highly urbanised area there is less difference in mean magnetic flux densities as estimated by wiring codes than in other areas. Such an effect combined with overmatching of cases and controls could explain the observations.

The data obtained for the use of C-ratios are suggestive that the greatest risk is for those in the age range 19–54 years (Table 28), and that it is greatest for diagnoses occurring 6–9 years after occupying the appropriate address (Figure 7). Wertheimer and Leeper also present data that suggest that no risk is apparent for those who left a high code address 3 years before diagnosis. Obviously, these data can only be derived from the two small towns where the controls were based on persons and not addresses. Wertheimer and Leeper suggest that these observations indicate a short latency for the effect of a high wiring configuration.

In a later paper, Wertheimer and Leeper (1987) use the same case-control population, together with the C-ratio parameter, to examine the risks for different types of cancer. These data are presented in Table 29. In general, the number of address pairs relevant to each type of cancer is too small to generate significant results. It is worth noting, however, that those which attain significance are cancers of the nervous system, which presumably includes brain tumours, and tumours of the female breast and uterus. These two latter categories would not have been examined in the occupational data described on page 18. It is particularly notable that the indicated risk for leukaemia is not elevated, and that for stomach cancer indicates a protective effect. The patterns

they observe for apparent latency, for ages for greatest risk and for urban/suburban differences are held by Wertheimer and Leeper to be consistent with electromagnetic fields acting as cancer promoters. An alternative view is that the relative uniformity of the C-ratios across the cancer types shown in Table 29 is suggestive that a factor unrelated to magnetic field exposure has biased the comparison of cases and controls.

In both their main papers Wertheimer and Leeper admit that the majority of the codings of addresses were not done blind and, in view of their apparent commitment to the hypothesis that wiring codes and possibly magnetic fields are causally related to cancer, this has been the subject for legitimate criticism by reviewers. In all other published studies those doing the codings were not aware of whether the address was that for a case or a control. However, in both papers Wertheimer and Leeper present data to indicate that there has been no bias in their coding and in view of the results of Savitz (1987) it must be concluded that the bias if it existed had very little effect on their results.

As part of the New York State Power Lines Project, which was a comprehensive series of biological and epidemiological studies of the effects of 60 Hz electromagnetic fields, a study was made of adult acute non-lymphocytic leukaemia (ANLL). This was the subject of both a report (Stevens, 1987) and a paper (Severson et al, 1988). Cases were living and dead persons aged 20–79 years diagnosed as suffering from one of the acute non-lymphocytic leukaemias (80% acute myeloid leukaemia, AML) in the state of Washington between January 1981 and December 1984. Living controls matched to the cases for age and sex were obtained by a system of random digit telephone dialling. Totals of 114 cases and 133 controls were involved. Wiring codes according both to the Wertheimer and Leeper prescription and to that of Kaune were obtained for addresses occupied by both cases and controls during a period of 15 years before a reference date (diagnosis for the cases and pseudo-diagnosis for the controls). All cases (or next of kin) and controls were interviewed and magnetic field measurements were made where the person had lived for at least 1 year in the home before the reference date. Unlike the wire coding, the magnetic field measurements could not be made without knowing a case or control was involved. Cases tended to be of a lower socioeconomic status than the controls and more of them were smokers. The study showed a significant association of ANLL with smoking [odds ratio 1.75 (95% CI 1.03–2.99)] and particularly for AML [odds ratio 1.89 (95% CI 1.08–3.33)]. The effect of this confounding influence would be to reduce the risk estimates associated with wiring codes and magnetic field exposures.

The results based on either the Wertheimer–Leeper coding or the scheme of Kaune show no indication of an increased risk with higher exposure (Table 30). Based on the magnetic field measurements, the relative risk estimates per nanotesla can be derived and are shown in Table 31. Although three of these give a positive association, the confidence intervals are such that none is significantly different from zero; that based on the measurements in different rooms weighted by the degree of occupancy under conditions of low power consumption shows a negative association. The most appropriate conclusion is that there is no effect, but with the range of confidence intervals on these estimates, neither real risks nor beneficial effects of exposure can be dismissed. The study showed a slight positive risk associated with electric blanket, electric pad and electrically heated water bed use, which was not statistically significant [odds ratio 1.5 (95% CI 0.9–2.4)].

Wertheimer and Leeper have circulated a manuscript (Wertheimer and Leeper, 1988) in which using the Stevens–Severson data they associate electrically heated bed use and their own early HCC scoring scheme to show a positive risk, although not a statistically significant one. However,

it is noteworthy that their own 1987 study of adult cancers indicated that there was no leukaemia risk. The increased cancer risk in their study is associated with other cancers.

The most comprehensive study of the relation between childhood cancers and residential exposures is that of Savitz and his co-workers. This, like that of Stevens and Severson, was undertaken as part of the New York State Power Lines Project and the two studies are probably the most reliable of all those published so far. The particular strength of the study is that in a series of related reports the confounding factors of traffic density and parental smoking are explored. It has been the subject of one paper (Savitz et al, 1988) and two reports (Savitz, 1987, 1988). It is based on all childhood cancer cases under the age of 15 years diagnosed between January 1976 and December 1985 in the Denver Standard Metropolitan Statistical Area. There is no overlap with Wertheimer and Leeper's childhood cases which were diagnosed in the period 1950–1973.

From an initial database of 356 cases, 252 (71%) could be traced for inclusion in the study. Of those included in the study, 29% were leukaemias (23% ALL), 19% brain tumours, 10% lymphomas, and 10% soft tissue tumours; other cancers made up the remaining 31%. By random digit telephone dialling controls were obtained to match the cases for age, sex and telephone exchange area; although specific individual matching was not attempted. Controls were restricted to those who were occupying the same home as that which they occupied when the equivalent case was diagnosed. One consequence is that in terms of residence the controls were a more stable and affluent population than the cases. A total of 222 controls were obtained who were prepared to be interviewed.

Wiring codes based on the Wertheimer–Leeper scoring system were obtained for addresses of cases and controls. In the analysis the codes appropriate to birth addresses, addresses at diagnosis and 2 years before diagnosis were employed. At the time of interview electric and magnetic field measurements were made for those homes which had also been occupied at the time of diagnosis. Measurements were made in different rooms and at the front door under conditions of maximum and minimum electric power consumption.

Table 32 shows the results in terms of the Wertheimer–Leeper configuration codes and the means of the average magnetic fields obtained under conditions of low power. Significant trends are obtained with the codes appropriate to the time of diagnosis and 2 years before. The trend with wiring code at time of birth is not significant, but this may be because of the smaller numbers available for this analysis. The data for 2 years before diagnosis produce a value for the increase in relative risk of 0.73% per nanotesla (95% CI 0.07–1.40%). A somewhat different picture emerges when the data are analysed in terms of the magnetic field measurements made in homes of cases and controls under conditions of low and high power consumption (Tables 33 and 34). The observed trends in risk with magnetic field exposure are all consistent with chance findings. Under conditions of low power consumption the relative risk at 2 years before diagnosis is estimated to increase at 0.12% for every nanotesla increase in magnetic field. The 95% confidence interval on this estimate (–0.24–0.48%) embraces a decrease in risk with increasing magnetic field strength. The estimates based on the magnetic fields under conditions of high power use are if anything lower and even more uncertain. Tables 35 and 36 present analyses of the data for the specific neoplastic diseases of leukaemia and brain tumours. The estimate of trend associated with the leukaemia data is slightly less likely to be due to chance than the trends for all cancers or for brain tumours, but it would be unwise to read into this observation any suggestion that leukaemia rather than any other childhood cancer is likely to be associated with magnetic fields in the home.

The stronger association with wire codes than with measured magnetic fields raises the

possibility that there is another factor other than the magnetic field which is causally related to cancer and associated with the wiring codes. Table 37 gives the reported traffic densities associated with the wiring codes – there is a clear and significant association. However, the association of cancer cases with this traffic density as reported by Savitz (1988) just fails to achieve significance (Table 37), although not less than with the measured magnetic fields. In a later paper (Savitz and Feingold, 1989), the connection with traffic density is explored in greater detail with increased numbers of cases and controls. The odds ratio for all childhood cancers at traffic densities greater than 500 per day in this later study is reported as 1.7 (95% CI 1.0–3.8) which is significant, and that for all childhood leukaemias as 2.1 (95% CI 1.1–4.0). Moreover, the data show an increasing risk with increasing density, the odds ratio for all cancers at densities in excess of 10,000 vehicles per day being 3.1 (95% CI 1.2–8.0). A significant increased risk of brain tumours is also demonstrated. In commenting on these data Savitz and Feingold caution against making any association with general traffic generated air pollution, but mention that benzene might be involved. They do not comment on the implications for the study of associations with wiring codes and magnetic fields. They do comment that the result is not materially affected by adjusting for wiring codes. A similar comment in relation to the effect of adjusting for traffic density on odds ratios associated with wiring codes is made in the earlier paper.

Savitz et al (1988) explore a range of possible confounding factors using a dichotomous analysis of the wiring codes (ie combining addresses with VHCC and OHCC codes for comparison with the combined addresses of OLCC, VLCC and buried codes). They were unable to find any factor that materially affected the odds ratios. Their data for smoking and non-smoking by the mothers in pregnancy yield an odds ratio for smoking mothers for all cancers of 1.43 (95% CI 0.93–2.21) and for X-rays of the fetus 1.44 (95% CI 0.84–2.45). These odds ratios are not very different from those associated with wiring codes and slightly more significant than those associated with the measured magnetic fields. Relations between childhood cancer and smoking by either parent were reported in a further paper (John et al, 1991).

Two other points are worth noting. Firstly, the ages of cases at the time of diagnosis with high coding addresses tends to be greater than those of the cases with low coding addresses and those of the controls (Table 39). This does not support the hypothesis that magnetic fields act as promoters in a way that produces cancers earlier in life, and thus elevates the odds ratios for the highest wiring code. Secondly, there seems to be no association of childhood cancers with the number of electrical appliances in use within the home (Table 40), although in a subsequent paper (Savitz et al, 1990) weak associations are claimed with usage of electric over-blankets by the children and prenatally by their mothers. The most pronounced associations are for leukaemia [odds ratio 1.7 (95% CI 0.8–3.6)] and brain tumours [odds ratio 2.5 (95% CI 1.1–5.5)] with prenatal use by the mothers.

Two studies have failed to find any link between the use of electric over-blankets and adult cancers. The first (Preston-Martin et al, 1988) was a case-control study of 137 patients diagnosed with acute or chronic myeloid leukaemia aged 20–69 years between 1979 and 1985 from the University of Southern California Cancer Surveillance Program. For acute myeloid leukaemia the relative risk was 0.9 (95% CI 0.5–1.6) and for chronic myeloid leukaemia 0.8 (95% CI 0.4–1.6). The second (Verreault et al, 1990) was of 214 men aged 20–69 years diagnosed with germ cell testicular cancer between 1981 and 1984 from a cancer registry serving 13 centres of western Washington. The age adjusted relative risk for those using electric blankets was 1.0 (95% CI 0.7–1.4).

3.2.2 Swedish and British studies

The principal American studies have been concerned with overhead electricity supply cables at 60 Hz. As already remarked, in Sweden and Britain the power frequency is 50 Hz and the supply cables are more usually buried. The reported positive linkage between childhood cancer incidence and magnetic fields found in the Swedish study was therefore of considerable interest.

Tomenius (1986) studied 716 cases of cancer in the 0–18 years age group diagnosed in the County of Stockholm between 1959 and 1973. This study is probably the best of those made in relation to 50 Hz exposures, since unlike the others an attempt was made to obtain a direct measure of the magnetic fields involved and comparatively large numbers were studied. Controls were closely matched to the cases for age, sex and their church district of birth. Birth and diagnosis addresses (or equivalent for controls) were obtained. Of the 2187 homes so identified, measurements of magnetic field strength were made at the front doors of the 2099 that had not been demolished and were occupied. Although in the American studies (Savitz, 1987; Kaune et al, 1987), there was some correlation between magnetic fields in different parts of the home, it is not clear to what extent this applies to Swedish and British homes. The Kaune studies also showed variations with the time of day at which measurements were taken. Tomenius does not indicate whether all the measurements were made at identical times of day. It seems likely that measurements outside the front door of a home are at least as good as, if not better than, wiring codes for indicating the general level of magnetic field exposure of the inhabitants.

Tomenius also recorded the proximity within 150 m to the homes of different types of electrical construction. The odds ratio for the different types of construction are given in Table 41. Taken altogether the odds ratio of 1.36 (95% CI 1.01–1.84) is significant, as is that for the proximity of a 200 kV transmission line. The latter influenced the magnetic field levels at the front doors (Figure 6). The data in Table 42 suggest that it is the associated magnetic fields rather than the proximity of the electrical construction that increases the odds ratios. The data in both these tables are for the combination of birth and 'diagnosis' addresses. Possibly confounding these observations is the fact that the controls were more likely than the cases to have identical addresses for both birth and 'diagnosis' – this is likely to increase the odds ratio. Table 43 gives the odds ratios by magnetic field levels for stable birth and diagnosis addresses, birth addresses and 'diagnosis' addresses. The former yields a rather higher odds ratio; those for the birth and 'diagnosis' addresses are comparable. When broken down by types of cancer (Table 44), the most significant and highest risk is for tumours of the central nervous system. There is no indicated risk for leukaemia. Analysis by age suggested that the risks were significant only for the 0–4 years age group.

A criticism of the Tomenius paper has been the choice of the 300 nanotesla (nT) level for the dichotomous analysis, which was chosen to provide a more direct comparison with the original Wertheimer and Leeper (1979) paper. This type of analysis is always suspect, since a choice of a slightly different level can in some instances produce an entirely different result. Rather interestingly there is virtually no difference between the means of the magnetic fields measured outside the front doors of the cases (69 nT) and those of the controls (68 nT) and for those living in close proximity to the 200 kV lines for whom a high odds ratio is obtained (Table 41). The mean of the fields measured and the case addresses (182 nanotesla) is less than that for the controls (329 nT).

Additional data were given by Tomenius in 1982 (Table 45) in a poster presented at the International Symposium on Occupational Health and Safety in Mining and Tunnelling, Prague, 1982. The statistical analysis demonstrates that there is no real trend for the odds ratios to increase

with increasing magnetic field. For comparison purposes, the data yield an increase in the relative risk of 0.02% for every nanotesla increase in magnetic field. The 95% confidence interval on this estimate (–0.10–0.14%) embraces a beneficial effect as might be expected.

There have been three published British studies (McDowell, 1986; Coleman et al, 1988; Myers et al, 1990) on the possible cancer risk associated with living in proximity to electrical installations or power lines. That by Myers et al is a substantially revised version of an earlier preliminary publication (Myers et al, 1985).

McDowall using maps identified and traced 7631 persons who at the time of the 1971 population census were living within 50 m of an electrical substation or other electrical installation or within 30 m of an overhead power line in East Anglia. Both in terms of age and occupational class distributions these were a reasonable match to the total population of East Anglia. There were slightly fewer persons aged over 65 years in the study population probably due to the absence of any institutions for the elderly in close proximity to the electrical installations. The standardised mortality ratios were calculated by comparison with the East Anglia population for the period April 1971 to December 1983. These are listed for the various diseases in Table 46. The only cancer for which there appears to be an increased risk with proximity to the installations is that for the lung; as McDowell pointed out this was mainly in women. The SMR for suicide calculated for the entire study population was 75 (95% CI 37–154) and for those living within 15 m of an installation 143 (95% CI 33–639). The latter is based on two deaths. The values are not significant. The data for all cancers are presented in a slightly different fashion in Table 47. Presented in this way there is a very slight indication, although statistically quite insignificant, of an increased risk with proximity to the installations.

Coleman et al studied cases of leukaemia occurring in the four South London boroughs of Bromley, Croydon, Merton and Sutton during 1965–1980. They used two types of control: other cancer cases matched individually for sex, age and year of diagnosis, and individuals aged 18 years and over drawn at random without matching from the 1975 electoral roll for Bromley. An obvious weakness of the study is that if cancer generally, rather than leukaemia specifically, is associated with living in proximity to an electrical installation, then using cancer cases as controls is not likely to indicate a leukaemia risk. The electrical installations identified in this study were high tension overhead power transmission lines and electrical substations. The odds ratios for distance from a substation are shown in Table 48 using cancer controls for comparison. There is no indication of an increasing risk with proximity to the substation. The numbers for those living in proximity to the transmission lines are small and little can be deduced from them. The slight indication of an increased risk at less than 24 m from a substation is due to acute and chronic lymphocytic leukaemia cases (53% of all leukaemia cases). The odds ratio for these two types of leukaemia combined at a distance of less than 24 m compared to 100 m is 1.65 (95% CI 0.99–277), which is almost significant. The values for leukaemia cases combined with the cancer controls with respect to the non-cancer individuals is shown in Table 49. There is a slight indication of increasing risk with proximity to the substations, but this is quite insignificant. Moreover, since these controls were not matched to the cases this observation is rather doubtful.

The study by Myers et al was based on childhood cancer cases diagnosed under the age of 15 years and born within the Yorkshire Health Region. A total of 374 case and 588 control addresses at the time of birth were identified and their proximity to overhead power, electricity supply and transmission lines derived from maps. Magnetic fields at the addresses due to these lines were calculated on the basis of records for electric current levels in the lines. Although

dichotomous analyses-both for distances closer than 100 m and for magnetic fields greater than 10 nT gave odds ratios slightly greater than unity that were statistically insignificant, taken as a whole there was no indication from the results of any association between the cancer cases either with proximity at birth to the lines or with the calculated magnetic fields. This was also the case for non-solid and solid tumours separately analysed. Owing to the small numbers, there was a low statistical power for detecting relative risks of less than about 2.0–2.5. It may also be the case, as indicated by the Savitz study (Table 32), that it is the place of residence of the child for some years before diagnosis rather than at birth which determines any possible risk.

3.2.3 Populations exposed to radar emissions

Lester and Moore (1982a) studied cancer incidence and mortality in the city of Wichita in Kansas, USA, for the period 1975–1977 in respect of possible exposure to radar from a civilian airport and an airforce base. They claimed a correlation between possible exposure and incidence but not between possible exposure and mortality. They also claimed in a second paper (Lester and Moore, 1982b) a correlation between cancer incidence in counties throughout the USA and the presence in the county of an airforce base. This finding was later disputed (Polson and Merritt, 1985; Lester, 1985). The merits of these papers, which are concerned with a quite different type of exposure than the others discussed here, are difficult to evaluate due to insufficient detail. The first paper (Lester and Moore, 1982a) indicates that age structure, social class and urbanisation are important confounding factors, but these are not discussed in relation to their second paper.

3.2.4 Summary and discussion

The studies discussed above are summarised in Table 40. The overall impression is that a strong association between childhood leukaemia and proximity to electrical installations has only been found in the Denver area of Colorado. It has not been found elsewhere, although an association has been found with other childhood cancers in Sweden as was also the case in Denver. For adults no association with proximity to electrical installations has been found for the leukaemias, but an association has been found with other cancers, but only strongly in the Denver area. Where it has been possible to investigate associations with different levels of measured or calculated magnetic field exposure, the associations are weaker and of less significance than they are with proximity to electrical installations.

In comparison with the other areas studied, Denver is at quite a high altitude (approximately 1500 m), with a higher background of ionising cosmic ray radiation (460 $\mu Sv\ y^{-1}$) compared with other places (310 $\mu Sv\ y^{-1}$). There is no evidence, however, that cancer rates are higher in Denver than elsewhere in the USA (Savitz and Zuchermann, 1987). The ionising radiation background is unlikely to be an explanation for any of the results, particularly since in the studies both cases and controls come from the same general area.

The study by Savitz (1988) of childhood cancers is the most comprehensive yet published. In terms of accepted standards of epidemiology this study could be criticised because it was only able to obtain data on about 70% of the possible cases and controls that could have been studied, and on an even smaller proportion for actual magnetic field exposures. It is not clear what positive or negative biases may have been introduced by the omission of data from missing cases or by the method of selecting controls. Similar criticisms could be made of some of the studies, including those in the UK that have not shown any significantly excess risks; in particular, many of these have

very small numbers of cases and controls and therefore very low statistical power for the detection of small excess risks. The stronger link with wiring codes than with magnetic field measurements is a puzzling feature of all those studies indicating positive risks. Recently there has been a report of a yet unpublished study of childhood cancer in the Los Angeles County of California which also apparently finds a stronger association with wiring codes than with measured magnetic fields (Pool, 1991). It may be, as has been suggested (Savitz et al, 1989), that wiring codes are a better measure of the long-term magnetic field exposure than measurements made over a comparatively short time. The body of data provided by the Savitz studies includes suggestions of possible confounding factors that may be linked to wiring codes such as traffic density and socioeconomic status, which could include effects of maternal smoking and degree of access to medical care. The relative risks of childhood cancer associated with some of the factors that are apparent in the Savitz data are shown in Table 51. Some of the relative risks are comparable with those associated with wiring codes and all are greater than that associated with measured magnetic fields. Some of them are comparable with regional variations in childhood cancer mortality in the UK (Table 52). It is not clear whether the differences in socioeconomic status and behaviour found in the Savitz studies apply to the cases and controls in the Wertheimer–Leeper studies, since using their C-ratio parameter they indicate higher risks to persons of higher rather than lower status. However, it is clear from the Savitz studies that high wire codes are associated with greater traffic densities (Table 37 and Savitz and Feingold, 1989).

The original Wertheimer–Leeper study of childhood cancer deaths spanned a period of time during which considerable progress was made in the treatment of childhood leukaemia with increased survival rates. It is possible, therefore, that the ability to access medical care and the timing of that access linked to socioeconomic status may have influenced their results. This argument would not apply to the same extent to the Savitz study, since it was concerned with incidence rather than death and covered a later period. Nevertheless, it is interesting that the average age at diagnosis for their cases increases with the wiring code level (Table 39), possibly indicating a delay in seeking medical treatment and linked to socioeconomic status. Tomenius presented no information on the socioeconomic status of his cases and controls.

Even if the results have been confounded by factors of smoking and socioeconomic status, the nature of the links between wiring codes and magnetic fields are such that it is possible to argue that any risks from magnetic fields derived from wiring codes are more likely to be underestimates rather than overestimates. The evidence from risk estimates derived directly from the measured or calculated magnetic field levels tends to refute this argument. These are considerably more uncertain and smaller than those derived from the wiring codes.

The data of Wertheimer and Leeper yield an increase in the risks for adult cancers of 0.36% (95% CI –0.12–0.48) for every nanotesla increase in the magnetic field (Table 27) based on wiring codes. In contrast, the Stevens estimate for acute non-lymphocytic leukaemia based on field measurements is 0.13% per nanotesla (95% CI –0.28–0.54%), Table 31, and his weighted estimate indicates a beneficial effect, ie –0.02% per nanotesla (95% CI –0.47–0.43). Wertheimer and Leeper's own data, however, did not indicate any increased risk for leukaemia. Since the average background magnetic field in British homes is possibly about 20 nT (Table 27), the Wertheimer–Leeper estimate implies that about 7% of adult cancers are due to magnetic fields in the home. Despite the five-fold increase in the consumption of electric power in Britain between 1950 and 1980, the cancer mortality for the 15–64 years age has fallen by about 4% (Table 1 and Table A1 in Appendix A). It may be, of course, that the mortality would have fallen even more

quickly in the absence of magnetic fields from the electricity supply, but the indication is that if there is any real risk it is very probably less than that implied by the Wertheimer–Leeper study.

In respect of childhood cancers the relative risk based on the wiring codes from the data of Savitz is 0.73% per nanotesla (95% CI 0.07–1.40), Table 32. The estimate derived from the magnetic field measurements is 0.12% per nanotesla (95% CI –0.24–0.48%), Table 33. The data of Tomenius yield a value that is a factor of four lower. These risk values include the leukaemia and brain tumour risks. If true, the Savitz value would imply that about 2.5% of childhood cancers are due to domestic magnetic fields in Britain. The current incidence of childhood cancer is about 100 per 1,000,000 per year (ie about double the mortality which has declined over the years, Table A1, Appendix A). If an annual excess risk of 10 per 1,000,000 is acceptable, as suggested by the Royal Society (1983), then the implied acceptable level of magnetic flux density for members of the public is about 80 nT. This level will be reached in close proximity to electrical appliances in most homes. The confidence intervals on the risk estimates are such that a beneficial effect of the exposure cannot be ruled out, or at the other extreme that the implied tolerable level might be as low as 20 nT and that about 10% of childhood cancers are due to domestic fields.

It must be emphasised that the risk estimates are extremely unreliable. They are not supported by studies elsewhere and there is the possibility that the effects of smoking, socioeconomic status and access to medical care have some part in exaggerating any real risks. The span of the estimates includes the possibility of beneficial effects of exposure. The relative risk to adults of leukaemia, if it is real, is if anything likely to be less than that for other cancers. This is not the case for children, but there is no reason to emphasise the leukaemia risk rather than the general cancer risk to children.

Prudence suggests that the indication of cancer risk from magnetic field exposure should be taken seriously and research to determine the reality of the risk and quantify it within closer bounds should be pursued.

3.3 Effects of paternal exposure

Savitz (1986) surveyed the literature on associations between the fathers' occupation and exposure to chemicals and cancer in their children. The survey identified significantly elevated relative risks for all types of cancer to children whose fathers were workers with metal and machinery, with motor vehicles and other aspects of transportation, with farming and farm products and with chemical solvents. The same groups of children had rather higher relative risks in the range 1.5–7 specifically for cancers of the nervous system and rather lower in the range 1.1–2.5 for leukaemias and lymphomas.

One notable report included in the Savitz survey was that of Spitz and Johnson (1986) who found a significantly elevated risk of neuroblastoma (an extracranial nervous system tumour of the adrenal glands) for children of electronics workers. They investigated deaths from this tumour in children under the age of 15 years during the period 1964–1978, who were born in Texas. For 157 identified cases twice as many controls were randomly selected to have the same birth year distribution. Although there were some slight differences in racial composition between the cases and controls (rather more white males in the cases), the two populations were closely matched for all other relevant factors, ie parental age, urban versus rural residence. Because of unknown parental occupation, 7 of the cases and 47 of the controls were excluded from the analysis. The study showed a significant relative risk of 3.17 (95% CI 1.13–8.89) for children whose fathers had

been exposed to aromatic and aliphatic hydrocarbons. Included among these were children of electrical and electronics workers, who are shown as three separate groups in Table 53. The significantly elevated risk of 11.5 (95% CI 1.4–96.9) is confined to the children of electronics workers. Their finding was based on the same data that indicated pre-term birth was a protective factor (Johnson and Spitz, 1985), which led them to suggest that some factor introduced transplacentally at the end of pregnancy was the cause. This produced some comment in relation to the effect of the father's exposure to electromagnetic fields (Sahl, 1986; Spitz and Johnson, 1986). The two findings are not necessarily incompatible, since only 11% of the children had fathers employed in electrical or electronics work, but their latter finding points more to solvent than electromagnetic field exposures as a possible causal factor.

An attempt to replicate the Spitz and Johnson study was made by Wilkins and Hundley (1990) in a case-control study of 101 cases of neuroblastoma in children born during the period 1942–1967 and registered in the Columbus (Ohio) Children's Tumor Registry. The indicated risks worth noting from this study were for parental employment in rubber, plastics and synthetics industries, 1.9 (95% CI 0.4–8.2); service occupations, 2.0 (95% CI 0.99–4.2); packaging and materials handling, 2.0 (95% CI 0.7–6.1). For electrical and electronics occupations equivalent to the Spitz and Johnson 'narrow' definition, which excludes salesmen and repairmen, the risk was 1.9 (95% CI 0.4–9.7) and for the 'broader' definition 0.7 (95% CI 0.3–1.5). The authors suggest that this result is consistent with that of Spitz and Johnson. A similar study was carried out on 104 children diagnosed between 1970 and 1979 from the Greater Delaware Valley Pediatric Tumor Registry and the tumour registry of the Children's Hospital of Philadelphia by Bunin et al (1990). The risk for the 'narrow' definition was 1.3 (95% CI 0.4–4.1) and for the 'broader' definition 1.0 (95% CI 0.4–2.3). While these authors did not consider that these results supported the Spitz and Johnson observations, they did find a small, insignificant risk when either the father or the mother had an electrical or electronics job [for the fathers an odds ratio of 4.0 (95% CI 0.4–195)].

A later study by Spitz and Johnson in conjunction with others (Johnson et al, 1987) was concerned with intracranial (brain) and central nervous system tumours in children. As in their earlier study of extracranial neuroblastoma, the cases were drawn from Texas but for a slightly longer overlapping period, 1964–1980. Significantly elevated risks were found for children whose fathers worked in the printing and graphics arts [4.5 (95% CI 1.1–14.7)], chemical and petroleum refining [3.0 (95% CI 1.1–8.5)] or who were chemical or drug salesmen [10.0 (95% CI 1.2–85.5)]. In a further paper (Johnson and Spitz, 1989) using the same database, they explored associations with parental involvement in the use, repair and manufacture of electronic equipment. For employment in all industries involving potential exposure to low frequency electromagnetic fields the risk was of borderline significance at 1.64 (95% CI 0.96–2.82). For the electronics manufacturing industries the indicated risk was higher and marginally more significant at 3.56 (95% CI 1.04–12.24). Owing to the small numbers, further breakdown by type of employment does not yield significant risks, but gives the impression that the odds ratios were higher in those industries particularly concerned with manufacture and assembly in which there was no elevated exposure to electromagnetic fields. As the authors point out, these industries involve potential exposures to chlorinated diphenyls and naphthalenes, epoxy and phenolic resins, solder and soldering flux fumes, synthetic waxes and vanishes, machine oils, platinum and tellurium.

Wilkins and Koutras (1988) studied brain cancer in Ohio-born children during the period 1959–1978. Their 491 cases were matched with a similar number of controls for sex, race and date of birth. They found significantly elevated odds ratios for the children whose

fathers' occupation at the time of birth was in structural work [2.1 (95% CI 1.4–3.1)], in construction [2.0 (95% CI 1.0–3.8)] and in agriculture and farming [2.0 (95% CI 1.0–4.1)]. They also found an elevated risk for children whose fathers' occupation was electrical assembly, installation or repair in the machinery industry: odds ratio 2.7 (95% CI 1.2–6.1). These odds ratios were adjusted for parental age, birth order, birth weight, and the proportion of farmland in the mother's country of residence.

A similar study of children in New York State diagnosed as suffering from central nervous system tumours between 1968 and 1977 was conducted by Nasca et al (1988). They identified 338 children who were eligible for inclusion in the study and whose mothers they were able to interview. For each case two controls were identified matched for birth, of whom 676 mothers were interviewed. Significantly elevated risks were found for the children whose fathers at the time of birth had been employed in an industry where there was a possibility of exposure to ionising radiation [1.83 (95% CI 1.32–2.53)], but not for occupations involved in such exposure [1.07 (95% CI 0.74–1.56)]. A marginally significant odds ratio was obtained for children whose fathers were employed in the petroleum industry [3.14 (95% CI 0.98–10.80)]. The odds ratio for children whose fathers were employed in occupations with possible electromagnetic field exposures, although elevated, were not significant [for a narrow definition of occupation 1.70 (95% CI 0.80–3.59) and for a broad definition 1.61 (95% CI 0.83–3.11). All the odds ratios were reduced for father's occupation at the time of diagnosis.

Two studies of leukaemias in children have failed to report any association between these and the father's involvement with electromagnetic fields, although in both cases questions about radar and microwave exposure were asked. Lowengart et al (1987) studied cases of forms of acute leukaemia in children under the age of 10 years diagnosed between 1980 and 1984 in Los Angeles County. They interviewed the mothers of 159 such cases and 136 mothers of children who were close friends of the cases. They found significant associations between the cases and the father's involvement in manufacturing and machinery industries, and their exposure to chlorinated solvents, spray paints and dyers during and after the mothers' pregnancies. They also found significant associations with the exposure of either parent to household and garden pesticides and incense burning in the home. Buckley et al (1989) studied the less common form of acute childhood leukaemia, acute non-lymphocytic leukaemia, and also found a significant association with exposure of parents and children to pesticides.

3.4 Summary and conclusions

The only significant association between employment of a father in an electrical or electronics occupation and cancer or other adverse effect in his children has been between neuroblastoma and an electronics occupation. The nature of this association is suggestive of a link to solvent rather than to electromagnetic field exposure. Data from an associated study failed to show any association between brain tumours in the children of workers with electromagnetic fields. Of two other studies, one did find a significant association and in the other it was not significant. It seems likely that if there is a real association it is rather weak. All studies found significant associations with occupations that do not have electromagnetic field connotations.

4 Summary, conclusions and recommendations

From the evidence presented in the companion reports (Kowalczuk et al, 1991; Sienkiewicz et al, 1991; Saunders et al, 1991) it seems clear that the biological effects of electromagnetic fields have only been unequivocally demonstrated *in vivo* at field intensities such that either

(a) the induced currents in the bodies of humans and animals produce direct stimulation of nervous tissue, or

(b) the energy absorption results in heating to the extent that the temperature regulatory responses are unable to maintain the overall body temperature within about 1 degree celsius of that which would be normal for the environmental conditions.

While there is some evidence from experiments with intact animals that biological effects may occur at lower field intensities, these effects tend to be within the normal range of biological variation. So far there is no evidence that low levels of electromagnetic fields can induce changes in the genetic material of cells, ie in the DNA, and lead directly to cancer or birth defects. There is some evidence that they might alter the expression of DNA in the synthesis of proteins and promote the more rapid development of malignant cells *in vitro* and of cancers induced in animals by other agents. There is a growing body of evidence that effects, whose health implications are unclear, can be produced *in vitro* at field intensities that are very much lower than those at which direct stimulation or overt heating of tissues in culture is likely. Some of this evidence is indicative of effects which do not demonstrate a linear dependency on either frequency or intensity – not merely in that effects are not proportional to intensity or frequency, but also in that 'windows' of intensity and frequency have been reported. This presents a considerable challenge to scientific understanding and explanation. Although the evidence for this is not well established, it is not possible to assert *a priori* that there will be no short-term or long-term effects on human health from exposures at levels of electromagnetic fields below those at which acute effects are likely to be manifest or that lower levels of exposure would necessarily pose a smaller risk than higher levels.

An obvious problem in the studies of human populations so far published is that the measures of exposure have almost always been indirect, relying either upon job descriptions or upon distance from electrical installations. In general, such imprecision in the estimation of exposure in epidemiological studies would be to produce an underestimate of any real risks. However, there may be other indirect risk factors that either result in an impression of risk from the exposure when none exists, or result in the actual risk from the exposure being overestimated. Contributing to an impression of a risk when none exists or the exaggeration of an actual risk is the fact that there is usually a bias against publication of null results, ie studies which show no risk may not be published because such a result is not normally interesting.

4.1 Effects on general health

As regards effects on general health and on physical and mental performance, the bulk of the evidence points to there being no effects at levels to which people are normally exposed from power supplies and from radiofrequency and microwave transmissions. Nevertheless, some studies have indicated minor changes in levels of a protein in the cerebrospinal fluid and in blood cell concentrations and functions in those persons occupationally exposed to rather high levels close to, or exceeding, the restrictions advised by NRPB (1989). Such changes do not appear to have had any effect on the health of those studied. While there does not appear to be any increased risk of cataract formation for exposures to microwave radiation below the reference level of 50 W m^{-2}

advised by NRPB, there have been no reported studies of retired workers who may have had high exposures in the past.

4.2 Effects on birth outcomes

The studies that have been made in connection with the use of visual display units (VDUs) suggest that for the average female user there is no influence on birth outcome. Although there is some evidence that very intensive use may increase the risk of spontaneous abortion, it is possible that this is a result of stressful working conditions. The increase in risk seems to be comparable to that associated with physical exertion or stress in pregnancy. For example, the studies by McDonald et al (1988a,b), while finding a significant relative risk of spontaneous abortion among women using VDUs more than 15 hours a week of 1.23, also found relative risks from lifting of 1.32, from physical effort of 1.26, from working long hours of 1.13 and from shift work of 1.17. Later studies provided evidence that women who suffer a spontaneous abortion are more likely to recall their use of VDUs than those who do not, which would give a bias towards finding a false indication of risk.

There is limited evidence from the USA that the use of electric over-blankets, which are energised while the user is in bed, may increase the spontaneous abortion risk. (In the UK electric under-blankets are more common and are not meant to be energised when the user is in bed.) The over-blankets are reported to expose the users to 60 Hz magnetic fields of about 2000–3000 nT in comparison with the normal domestic background of 50–60 nT. Such blankets probably represent the source of the highest domestic exposures in the USA. As a result of this evidence and that from the intensive use of VDUs, it may be worth carrying out further studies of highly exposed groups of women such as those who use radiofrequency heaters, despite the fact that one study of such a population has reported no effect. By the fragmented nature of the industries in which they work, an epidemiological study of the longer term effects of exposure on the women themselves or on men working with such machines would face formidable problems.

4.3 Cancer risks

The cancers most often identified in studies indicating increased risks to workers with electrical or electronic occupations have been leukaemia (usually acute myeloid leukaemia) and brain tumours, although one or two studies have indicated increased risks of other cancers. The absolute values of the indicated increased risks for these two identified types of cancer are comparable at about 1 to 2 per 100,000 per year, and total between 2 and 4 per 100,000 per year. These are likely to be considerable overestimates owing to the confounding factor of occupational class. Nonetheless, if the risk is real, there may be some more highly exposed workers or those exposed to particular frequencies who will be at greater risk. The greatest increase in risk appears to be to those who are engaged in the repair and assembly of equipment; this may not involve any excess exposure to electromagnetic fields, but may involve exposure to a variety of chemical and metal fumes. It has to be noted that there does not appear to be any excessive risks to welders who, as an occupational group, will be exposed to some of the highest magnetic fields, and also that one study has not shown any increased leukaemia risk to adult users of electric over-blanket. Moreover, none of the studies of the populations thought to have high exposures from overhead domestic power lines has demonstrated an increased leukaemia risk to adults. There must be a suspicion that if the indicated increases in risks are real, then they arise from the chemical and metal fume

exposures and, because of a confounding factor of occupational class, may have been exaggerated. However, some of the *in-vitro* biological studies have indicated that electromagnetic fields could exert a promotional role in the development of cancer. It may be, therefore, that it is the combination of both electromagnetic field exposures and chemical and metal fume exposures that is responsible for the increased risks, if these are real. It must also be noted that the average increase in risk is about the same as, and often less than, those associated with other environmental and workplace agents and, as an average, the absolute value is not excessive in terms of contemporary views on acceptable risk levels.

Published studies that have identified the most significant excess cancer risks associated with living in proximity to electrical installations have been for one area of Sweden and for the Denver area of the USA. In the Denver studies the strongest associations were with the local densities of overhead domestic electrical supply wires and cables when expressed as wiring codes. The Swedish study examined proximity to a wider range of electrical installations including high voltage transmission lines and, as the Denver studies, showed only an insignificant association with measured magnetic fields compared to the association with living in proximity to the installations. In the most comprehensive of the Denver studies there was a strong correlation between the wiring codes and traffic density, and the relative risks of childhood cancer associated with high traffic densities were equal to, or greater than, those associated with wiring codes and of greater significance than those associated with magnetic fields.

There must be considerable doubts as to whether there are real increases in cancer risks to children, and even greater doubts as regards risks to adults, from domestic magnetic fields. Within the UK the risks are unlikely to exceed those associated with the variations in cancer risks between different areas of the country. So far no studies in the UK have suggested any significant excess cancer risk from living in proximity to an electrical installation or from domestic magnetic field levels, but most have had only a limited ability to detect a small excess risk. The available evidence is not sufficient to justify excessive concern about magnetic field levels in the UK from domestic wiring, electrical appliances, power lines, etc, but neither is there any justification for complacency. As further evidence accumulates it may be necessary to advise that exposures should be restricted to levels below those which appear at present to be acceptable.

The nature of the evidence linking parental occupations in the electrical and electronic industries with cancer in their children is somewhat similar to that indicating increased risks of cancer to the workers themselves in that it appears to be associated with chemical and metal fume exposures. To some extent, therefore, the two strands of evidence are mutually supportive. The indicated risk to the children seems to be confined to neuroblastoma; an increased risk of either leukaemia or brain cancer does not seem to be suggested. Associations of these latter cancers with parental exposures to solvents, paints and pesticides have been reported in studies that found no associations with parental exposures to electromagnetic fields. There is no intention here to imply that these other associations are necessarily well founded.

4.4 Recommendations for research priorities

The following recommendations might be made in regard to the main priorities for further research and study.

(a) Efforts should be made to confirm the low level effects reported for *in-vitro* studies for a wide range of frequencies, intensities and modulation patterns. Biophysical explanations

for confirmed positive results should be sought and tested. For those exposure protocols that can be demonstrated to cause effects *in vitro*, it may be necessary to carry out *in-vivo* experiments with animals to demonstrate consequential effects on health.

(b) The nature and levels of exposure of those electrical and electronic occupational groups for whom an excess cancer risk has been indicated should be investigated in detail in comparison with appropriate groups for whom there is no suggestion of an excess risk. Such investigations should include details of exposures to chemical and metal fumes.

(c) Where highly exposed groups can be identified, studies should be made of cancer incidence and, for any groups of highly exposed women, of birth outcomes in relation to comparable control groups.

(d) If a group of elderly, retired persons who have worked with microwave radiation can be identified, incidence of cataract formation should be examined.

(e) Studies should be made of the levels of exposure to electromagnetic fields from external power cables and installations, from house wiring patterns and from electrical appliances. These studies should investigate not only the levels of exposure but also their diurnal and seasonal variations.

Some of these recommendations are already being implemented in the UK.

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Table 1

Electromagnetic energy usage and life expectancy in England and Wales

Year	Electric power[a] generated (GW h)	Radiofrequency[b] transmitted power (MW)	Life expectancy[c]	
			Male	Female
1930	8,165	0.5	59	63
1950	38,265	1.9	66	72
1960	85,719	9.1	68	74
1970	168,230	48.7	69	75
1980	204,752	70.2	71	77

Sources

(a) Handbook of Electricity Supply Statistics, 1987. London, Electricity Council.
(b) BBC - private communication.
(c) Expectation of life at birth for 3 year periods beginning at year listed, ie for 1950 corresponds to years 1950-1952. Office of Population Censuses and Surveys Reports DH1 No.6 (1978), No. 13 (1982). London, HMSO.

Table 2

Childhood mortality in England and Wales

Period	Rates per 1000 total births		
	Still births	Deths in first week of life	Deaths after first week and under 1 year
1931-35	41.0	22.4	39.5
1940	37.2	21.3	35.5
1945	27.6	18.0	28.0
1950	22.6	15.2	14.4
1955	23.2	14.6	10.3
1960	19.8	13.3	8.5
1965	15.8	11.3	7.7
1970	13.0	10.6	7.6
1975	10.3	9.1	6.6
1980	7.2	6.1	5.8
1985	5.5	4.4	5.0

Sources

Mortality Statistics: Childhood and Maternity. Office of Population Censuses and Surveys. Report Series DH3 No. 3 (1978), No. 8 (1980), No. 19 (1985). London, HMSO.

Table 3

Some typical sources of exposure and exposure levels to electromagnetic fields*

Exposure condition	Electric field strength	Magnetic field strength
Natural environment		
Fair weather - static fields	120–150 V/m	50 μT
Stormy weather - static fields	10 kV/m	50 μT
50 Hz field	10^{-4} V/m	0.001 nT
Man-made fields		
50 Hz, 400 kV power line midspan	10 kV/m	40 μT
50 Hz, 400 kV power line 25 m from midspan	1 kV/m	8 μT
500–1600 kHz, 100 m from AM broadcast antenna	20 V/m	
27 MHz, 4W CB radio 12 cm from antenna	100–600 V/m	0.2–0.8 A/m
470–854 MHz, TV broadcast max within 1 km of TV mast	3 V/m	$8\ 10^{-2}$ A/m
Domestic		
30 cm from TV/VDU - static field	0.5–10 kV/m	
50 Hz, ambient, distant appliances	1–10 V/m	10–1000 nT
50 Hz, 30 cm from appliances	10–250 V/m	10 μT–1 mT
15 kHz, 30 cm from TV/VDU	1–10 V/m	1000 nT
Occupational		
50 Hz, 0.5–1 m from induction furnaces		100 μT–10 mT
50 Hz, substations, etc.	10–20 kV/m	up to 50 μT
3 kHz, 50 cm from billet heater coil	70 V/m	380 μT
0.15–10 kHz, 0.1–1 m from induction heaters		15 μT–1.25 mT
250–675 kHz, at operator positions from induction heaters	2–100 V/m	0.2–22 μT
10–80 MHz, 15 cm from dielectric heaters	20–800 V/m	0.1–0.9 A/m
27–450 MHz, 2 cm from low power mobile antennas	200–1350 V/m	
470–854 MHz, TV aerial riggers	30–300 V/m	0.1–1 A/m

* The field strength values in the table are indicative of those to which people may be or have been regularly exposed. The information is collated from a variety of sources and is intended to provide a feeling for the magnitude and range of field strengths encountered. There are many other sources of potential exposure not referred to, and there is no significance to be implied from their omission.

Table 4

Tissue conductivity, ratios of internal to external electric field and fields required to induce a 5 μV signal (equated to noise level)[a] in the membrane of a 100 μm cell at different frequencies

Frequency	Approximate tissue conductivity (Siemens m^{-1})	Ratio of internal to external electric field	Internal current density (mA m^{-2})	External electric field (V m^{-1})	External magnetic field[b] (A m^{-1})
10 Hz	0.2	8×10^{-9}	20	12,500,000	8500 (11 mT)
50 Hz	0.2	4×10^{-8}	20	2,500,000	1700 (2.1 mT)
100 Hz	0.2	8×10^{-7}	20	1,250,000	850 (1.1 mT)
1 kHz	0.3	6×10^{-7}	30	167,000	85 (110 μT)
10 kHz	0.4	4×10^{-6}	40	25,000	8.5 (11 μT)
100 kHz	0.5	3×10^{-5}	50	3,300	0.85 (1.1 μT)
1 MHz	0.6	3×10^{-4}	60	330	0.085 (110 nT)
10 MHz	0.8	2×10^{-3}	70	50	0.0085 (11 nT)

Notes

(a) 5 μV is the approximate value of the natural electrical noise in the membrane. An 0.1 V m^{-1} electric field in the surrounding tissue will induce a 5 μV signal across the cell membrane.

(b) For a body radius of 15 cm.

Table 5

Mortality ratios* by exposure class for ex-American-Navy personnel (1950-1974)

Cause of death	Exposure class			Chi-squared test for trend	
	Low	Moderate	High		
All diseases	1.04	0.83	1.19	$\chi^2 = 0.27$	$p = 0.60$
All malignant neoplasms	0.96	0.95	1.18	$\chi^2 = 0.73$	$p = 0.39$
of digestive organs	0.85	1.10	1.19	$\chi^2 = 0.62$	$p = 0.43$
of respiratory tract	0.85	1.13	1.15	$\chi^2 = 0.72$	$p = 0.40$
of lymphatic and haematopoietic system	0.83	1.06	1.40	$\chi^2 = 1.90$	$p = 0.17$
other malignant neoplasms	1.19	0.68	1.06	$\chi^2 = 0.76$	$p = 0.38$
Disease of circulatory system	1.07	0.85	1.08	$\chi^2 = 0.17$	$p = 0.68$
Other diseases	1.08	0.61	1.46	$\chi^2 = 0.30$	$p = 0.58$

* Mortality ratios are standardised for year of birth and the combined experience of the whole population is taken as standard.

Table 6

Admission rates per 1000 per year for selected diagnoses to Naval Hospitals 1952-1954 and 1956-1959 for ex-American-Navy personnel

Diagnosis	Exposure class		
	Low	Moderate	High
Diseases of:			
blood and blood forming organs	0.2	0.2	0.00
nervous system	1.4	1.4	1.6
eye and adnexa	1.7	1.9	1.5
ear, nose and throat	2.0	1.9	3.1
circulatory system	10.9	11.2	11.8
urinary system and male genital system	8.9	7.5	9.3
skin and cellular tissue	8.3	7.1	8.6
bones and organs of movement	7.0	6.4	7.0
Acute respiratory infection	17.5	14.9	18.4
Other diseases of respiratory system	1.3	1.1	1.9
Allergic endocrine system, metabolic and nutritional diseases	2.2	1.7	2.0
Neoplasms	7.1	6.6	5.9
Mental, psychoneurotic and personality disorders	6.4	4.9	5.6
Congenital malformations	1.0	0.8	1.2
Accidents, poisonings and violence	27.5	21.6	29.8
Totals for all causes	127.3	114.0	130.3

Table 7

Disability compensation rates per 1000 for ex-American-Navy personnel in 1976

Diagnosis group	Exposure class		
	Low	Moderate	High
Musculoskeletal	13.9	8.8	16.9
Organs of special sense	5.0	3.7	6.0
Systematic conditions	0.2	0.2	0.7
Respiratory	5.6	4.2	7.3
Cardiovascular	4.8	3.3	6.7
Digestive	7.5	5.7	7.8
Genitourinary	3.3	2.4	2.7
Hemic, lymphatic	0.5	0.0	0.4
Skin	6.9	6.3	8.2
Endocrine	1.5	1.1	1.6
Neurological	1.6	1.6	2.3
Nerves	1.3	1.1	0.4
Epilepsies	0.6	0.3	0.4
Mental conditions	7.1	3.9	6.5
Other	0.6	0.5	0.3

Table 8

Estimated odds of committing suicide according to measured magnetic field strength, based on data given by Perry et al (1981)

Measured field strength (nT)	Odds of suicide	95% confidence interval
0–20	0.59	0.40–0.87
20–40	1.27	1.04–1.55
40–100	0.74	0.61–0.89
≥100	1.38	1.08–1.77
All	1.00	

Table 9

Summarised relative risks for VDU usage found by McDonald et al, 1988a

Effect	Current pregnancy		Previous pregnancy	
	Relative risk	90% CI	Relative risk	90% CI
Spontaneous abortion:				
by individual analysis	1.19*	1.09–1.30	0.97*	0.89–1.05
by occupational groups according to level of usage	1.06+	0.80–1.40	1.01+	0.70–1.30
Still-birth	0.82*	0.47–1.33	0.71*	0.42–1.13
Pre-term	1.08*	0.98–1.18	-	-
Low birth weight	1.03*	0.92–1.15	-	-
Congenital defect	0.94+	0.78–1.13	1.12	0.89–1.43

* Relative to all working women.
\+ Relative to working women in the study not using VDUs.

Table 10

Spontaneous abortions by time spent per week on VDU usage (McDonald et al, 1988a)

Time spent per week	Relative risk	90% CI
No use	0.96	0.92–1.00
Up to 15 hours	1.14	1.00–1.29
More than 15 hours	1.23	1.09–1.38

Note Analysed relative to all working women.

Table 11

Adjusted odds ratios for miscarriage and birth defects for first trimester VDU usage (Goldhaber et al, 1988)

Time spent per week	Miscarriage		Birth defects	
	Odds ratio	95% CI	Odds ratio	95% CI
No use	1.0		1.0	
< 5 hours	0.9	0.6–1.4	0.9	0.4–1.9
5–20 hours	1.0	0.6–1.6	1.4	0.7–2.7
> 20 hours	1.8	1.2–2.8	1.4	0.7–2.9

Note Analysis, as indicated, relative to those in study not using VDUs.

Table 12

All leukaemias risk in occupational groups with possible electromagnetic field exposures

Study	Time, place, occupation	Age range (years)	Risk measure	Risk estimate (95% confidence interval)	Comment
1. Milham, 1982	1950-79, Washington State electrical and electronic workers	≥20	PMR	1.37 (1.15-1.62)	Acute leukaemia risk: 1.63 (1.24-2.15)
2. Wright et al, 1982	1972-79, Los Angeles County electrical and electronic workers	Not stated	PIR	1.29 (0.92-1.80)	AML risk: 2.07 (1.31-3.28)
3. McDowall, 1983	1970-72, England and Wales electrical and electronic workers	15-74	PMR	0.98 (0.78-1.21)	AML risk: 1.04 (0.73-1.48)
4. McDowall, 1983	1973, England and Wales electrical and electronic workers	≥15	RR (death) case control	Not given	AML risk: 2.3 (1.4 - 3.7)
5. Coleman et al, 1983	1961-79, SE England electrical and electronic workers	15-74	PIR	1.17 (0.96-1.41)	AML risk: 1.23 (0.87-1.74)
6. Vågerö and Olin, 1983	1961-73, Sweden electrical and electronics workers	15-74	RR (incidence)	No identified leukaemia risks.	
7. Pearce et al, 1985	1973-83, New Zealand electrical and electronics workers	≥20	RR (incidence) case control	1.70 (0.97-2.97)	-
8. Milham, 1985a	1971-83, California and Washington States amateur radio operators	Not stated	PMR	1.90 (1.23-2.95)	AML risk: 2.89 (1.48-5.66)
9. Milham, 1985b	1950-82, Washington State electrical and electronics workers	≥20	PMR	1.36 (1.14-1.62)	Acute leukaemia risk: 1.62 (1.25-2.10)
10. Vågerö et al, 1985	1958-79, Sweden telecommunications manufacture	Not stated	RR (incidence)	No identified leukaemia risks.	
11. Olin et al, 1985	1930-79, Sweden electrical engineers	Not stated	SMR	0.90 (0.10-3.2)	
12. Calle, Savitz, 1985	1963-75, Wisconsin State electrical and electronics workers	≥20	PMR	1.03 (0.82-1.29)	Acute leukaemia risk: 1.13 (0.82-1.56)
13. Stern et al, 1986	1952-80, New Hampshire State shipyard workers	Not stated	RR (death) case control	3.00 (1.29-6.98)	Myeloid leukaemia risk: 2.33 (0.77-7.06) Similar risks for carpenters Ionising radiations involved

Table 12 Continued

Study	Time, place, occupation	Age range (years)	Risk measure	Risk estimate (95% confidence interval)	Comment
14. OPCS, 1986	1979-80, 1982-83, Great Britain electrical and electronics engineers	20-64	SMR	2.02 (1.36-2.98)	AML risk: 1.49 (0.74-3.01)
15. OPCS, 1986	1979-80, 1982-83, Great Britain electricians, fitters, plant operators, etc.	20-64	SMR	1.20 (0.91-1.60)	AML risk: 1.55 (1.05-2.29)
16. Flodin et al, 1986	1977-82, Swedish districts electrical workers	20-70	RR (incidence) case control	Not given	AML risk: 3.5 (1.5 - 9.5)
17. Törnqvist, 1986	1961-79, Sweden power linesmen	20-64	SMR	1.25 (0.60-2.30)	-
18. Törnqvist, 1986	1961-79, Sweden power station operators	20-64	SMR	0.96 (0.55-1.57)	-
19. Howe (Coleman, Beral, 1988)	1965-73, Canada power, telephone linesmen	Not stated	SMR	2.41 (0.97-4.97)	AML risk: 2.33 (0.26-8.40)
20. Milham, 1988	1979-84, Washington and California States amateur radio operators	Not stated	SMR	1.24 (0.87-1.72)	AML risk: 1.76 (1.03-2.85)
21. Juutilainen et al, 1988	Not stated, Finland electrical and electronics workers	Not stated	RR (incidence)	1.23 (0.74-2.04)	
22. Cartwright et al, 1988	1979-86, Yorkshire electrical workers	≥15	RR (incidence) case control	-	AML risk: 2.40 (1.04-5.46)
23. Pearce et al, 1988	1980-84, New Zealand electrical and electronics workers	≥20	RR (incidence)	1.62 (1.04-2.52	AML risk: 1.21 (0.38-3.85) CLL risk: 3.36 (1.27-8.89)
24. Gallagher et al, 1990	1950-84, British Columbia electrical and electronics workers	20-65	PMR	1.04 (0.71-1.52)	-

Notes: The following groups of studies probably contain overlapping data and may not be independent: 1,8,9 and 20; 6,10,11,16,17 and 18; 3 and 5: 4 and 5: 7 and 23.
SMR, standardised mortality ratio; PMR, proportional mortality ratio; PIR, proportional incidence ratio; RR, relative risk.

Table 13

Relative risks to polish military personnel by age and length of exposure (Szmigielski et al, 1988)

Age group (years)	Period of exposure (years)			
	> 2	2 - 5	5 - 15	> 15
20-29	4.0	7.4	-	-
30-39	-	3.8	5.6	9.7
40-49	-	0.9	4.7	5.5
50-59	-	-	1.4	1.7

Table 14

Brain tumour risks by attributed exposure to electromagnetic fields (Lin et al, 1985)

	Primary brain tumours			Unspecified brain tumours		
Attributed exposure	Cases	Controls	Odds ratio (95% confidence interval)	Cases	Controls	Odds ratio (95% confidence interval)
No exposure	323	360	1.00	286	294	1.00
Possible exposure	128	99	1.44 (1.06-1.95)	87	95	0.94 (0.68-1.31)
Probable exposure	21	12	1.95 (0.94-3.91)	19	15	1.30 (0.60-2.78)
Definite exposure	27	14	2.15 (1.10-4.06)	15	10	1.54 (0.68-3.38)
	Chi-squared for trend 11.32 p = 0.08%			Chi-squared for trend 0.68 p = 41%		

Note Among primary brain tumours there were 20 cases and 34 controls with unknown occupations, and among unspecified brain tumours 25 cases and 18 controls with unknown occupations.

Table 15

Brain tumour and other cancer risks to workers exposed to electromagnetic fields and other agents (Milham, 1985b)

Neoplasm and exposure	Observed cases	Expected cases	PMR (95% confidence interval)
Brain (7th ICD 193.0)			
EM only	19	21.3	89 (56-143)
EM + other	82	60.5	136 (108-171)*
All	101	81.8	123 (100-151)*
Pancreas (7th ICD 157)			
EM only	42	37.4	112 (82-154)
EM + other	132	111.3	119 (99-142)
All	174	148.7	117 (100-137)*
Lung (7th ICD 162)			
EM only	170	170.9	99 (85-116)
EM + other	619	523.3	118 (109-129)*
All	789	694.2	114 (106-122)*
Lymphosarcoma (7th ICD 200.1)			
EM only	12	8.2	146 (80-268)
EM + other	29	23.3	124 (85-183)
All	41	31.5	130 (94-180)
Other lymphosarcomas* (7th ICD 200.2, 202)			
EM only	16	8.1	198 (116-338)*
EM + other	35	23.0	152 (107-217)*
All	51	31.1	164 (122-221)*

* Non-Hodgkins lymphomas
* Significant at 5% level.

Table 16

Brain tumour risks by attributable exposure to electromagnetic fields (Speers et al, 1988)

Attributable exposure	Cases	Controls	Odds ratio (95% confidence interval)
No exposure	92	132	1.00
Possible exposure	68	83	1.18 (0.77-1.78)
Probable exposure	11	5	3.16 (1.06-9.39)
Definite exposure	6	0	∞ (1.64-∞)
	Chi-squared for trend 7.72 p = 0.55%		

Table 17

Brain tumour risks from exposure to radiofrequencies and microwaves according to occupation (Thomas et al, 1987)

Occupation	Relative risks* (95% confidence interval)
Electrical or electronic job	2.3 (1.30-4.2)
No eiectrical or electronics job	1.0 (0.5-1.9)

* As given by the authors relative to cases and controls and adjusted for educational class.

Table 18

Brain tumour risk in occupational groups with possible electromagnetic field exposures

Study	Time, place, occupation	Age range (years)	Risk measure	Risk estimate (95% confidence interval)
1. Milham, 1979	1950-71, Washington State aluminium workers	Not stated	SMR	0.99 (0.30-3.34)
2. Robinette et al, 1980	1950-74, USA naval radar and radio technicians	Not stated	PMR	2.08 (1.26-3.44)
3. Lin et al, 1985	1969-82, Maryland State electrical and electronic workers	≥20	SMR	2.78 (2.03-3.80)
4. Milham, 1985b	1950-82, Washington State electrical and electronic workers	≥20	PMR	1.23 (1.00-1.51)
5. McLaughlin et al, 1987	1961-79, Sweden electrical and electronic workers	Not stated	SIR	0.90 (0.71-1.14)
6. Thomas et al, 1987	1979-81, Eastern USA electrical and electronic workers	Not stated	RR (death) case control	1.60 (1.00-2.40)
7. Speers et al, 1988	1969-78, East Texas electrical and electronic workers	35-79	RR (death) case control	2.11 (0.77-5.81)
8. Milham, 1988	1979-84, Washington and California States amateur radio operators	Not stated	SMR	1.39 (0.93-2.00)

Notes: The following groups of studies may contain overlapping data and may not be independent: 1 and 2; 2 and 3; 2 and 4; 2 and 7.
SMR, standardised mortality ratio; PMR, proportional mortality radio; SIR, standardised incidence ratio; RR, relative risk.

Table 19

Standardised mortality ratios and relative proportional mortality ratios (see text) for occupational groups and specific diseases for British workers 1979-1980, 1982-1983, ages 20-64 years

9th ICD code / Disease / Group	140-239 All neoplasms SMR	390-459 Diseases of circulatory system SMR	204-208 All leukaemias SMR RSMR	205.0 Acute myeloid leukaemia SMR RSMR	204.1 Chronic lymphatic leukaemia SMR RSMR	191 Neoplasms of brain SMR RSMR	172 Malignant melanoma of skin SMR RSMR
Group I Electrical and electronics engineers and technicians	116*	112*	134* 118 (98-243)	147* 130 (99-173)	76 72 (37-138)	123* 109 (92-129)	141* 125 (94-165)
Group II Electrical equipment operators, electroplaters, welders	125*	119*	95 78 (53-115)	71 62 (32-120)	26 32 (8-130)	107 88 (64-121)	113 96 (57-164)
Group III Engineers and technicians not electrical or electronic	67*	68*	101 151 (118-193)*	109 162 (112-235)*	141 219 (129-373)*	94 141 (113-175)*	104 156 (108-226)*

Note Bracketed values - 95% confidence intervals.

*Significantly different from 100 at the 5% level or less.

Table 20

Proportional mortality ratios and relative proportional mortality ratios (see text) for occupational groups and specific diseases for British workers (males) 1979-1980, 1982-1983, ages 65-74 years

9th ICD code / Disease / Group	140-239 All neoplasms PMR	390-459 Diseases of circulatory system PMR	204-208 All leukaemias PMR RSMR	205.0 Acute myeloid leukaemia PMR RSMR	204.1 Chronic lymphatic leukaemia PMR RSMR	191 Neoplasms of brain PMR RSMR	172 Malignant melanoma of skin PMR RSMR
Group I Electrical and electronics engineers and technicians	102	103	109 106 (73-153)	130 127 (70-230)	97 95 (48-189)	131 128 (83-198)	99 96 (38-242)
Group II Electrical equipment operators, electroplaters, welders	103	99	80 80 (41-159)	17 23 (5-110)	82 83 (25-272)	88 88 (39-199)	0 17 (2-153)
Group III Engineers and technicians not electrical or electronic	113*	104	123 115 (65-205)	110 102 (40-262)	86 82 (27-245)	124 115 (58-230)	97 91 (23-361)

Note Bracketed values - 95% confidence intervals.

*Significantly different from 100 at the 5% level or less.

Table 21

Standardised, proportional and relative proportional ratios for plumbers, heating and ventilating fitters, and gas fitters (males) 1970-1980, 1982-1983

Age group (years) / 9th ICD code / Disease		140-239 All neoplasms	390-459 Diseases of circulatory system	204-208 All leukaemias	205.0 Acute myeloid leukaemia	204.1 Chronic lymphatic leukaemia	191 Neoplasms of brain	172 Malignant melanoma of skin
15-64	SMR	132*	117*	83	111	67	123	67
	RPMR			68 (43-105)	90 (50-211)	67 (21-211)	100 (73-178)	54 (26-114)
65-74	PMR	111*	96	115	90	148	153	107
	RPMR			114 (62-210)	90 (32-394)	143 (52-394)	152 (74-312)	105 (26-429)

Note: Bracketed values - 95% confidence intervals.
*Significantly different from 100 at 5% level or less.

Table 22

Standardised, proportional and relative proportional mortality ratios for chemical, petroleum, rubber and plastics workers (males) 1970-1980, 1982-1983

Age group (years) / 9th ICD code / Disease		140-239 All neoplasms	390-459 Diseases of circulatory system	204-208 All leukaemias	205.0 Acute myeloid leukaemia	204.1 Chronic lymphatic leukaemia	191 Neoplasms of brain	172 Malignant melanoma of skin
15-64	SMR	155*	136*	122	162	167	133	127
	RPMR			85 (55-133)	85 (62-206)	130 (53-319)	93 (73-178)	89 (46-172)
65-74	PMR	105*	99	82	109	59	42	74
	RPMR			81 (41-160)	107 (39-292)	58 (14-242)	44 (17-115)	77 (17-354)

Note: Bracketed values - 95% confidence intervals.
*Significantly different from 100 at 5% level or less.

Table 23

Magnetic flux densities in British and American homes
(Myers et al, 1985; Savitz, 1987)

	Magnetic flux densities, nanotesla					
	British homes				American homes*	
	Lounge	Kitchen	Bedroom	Bathroom	Low power	High power
Minima	2	2	1	1	2	4
Median	17	22	13	15	47	66
Maxima	220	230	130+	130+	461	1208

*Average over several rooms.

+Near to overhead power transmission line.

Table 24

Odds ratios for HCC configurations and all childhood cancers
in persons under 19 years (Wertheimer and Leeper, 1979)

Configuration and type of address	Case addresses	Control addresses	Odds ratio (95% confidence interval)
Stable residence			
LCC	61	102	
HCC	48	26	3.09 (1.74-5.48)
Birth addresses			
LCC	110	115	
HCC	53	29	1.91 (1.13-3.22)
'Death' addresses			
LCC	138	152	
HCC	81	48	1.86 (1.22-2.84)

Table 25

Odds ratios by type of childhood cancer for HCC configurations (Wertheimer and Leeper, 1979)

Cancer type configuration and type of address	Case addresses	Control addresses	Odds ratio (95% confidence interval)
Birth address			
Leukaemia			
LCC	84	107	
HCC	52	29	2.28 (1.34-3.91)
Lymphoma			
LCC	21	26	
HCC	10	5	2.48 (0.73-8.37)
Nervous system			
LCC	35	45	
HCC	22	12	2.36 (1.03-5.41)
Other			
LCC	31	39	
HCC	17	9	2.38 (0.93-6.06)
'Death' address			
Leukaemia			
LCC	92	126	
HCC	63	29	2.98 (1.78-4.98)
Lymphoma			
LCC	26	33	
HCC	18	11	2.08 (0.84-5.16)
Nervous system			
LCC	36	49	
HCC	30	17	2.40 (1.15-5.01)
Other			
LCC	45	46	
HCC	18	17	1.08 (0.50-2.36)

Table 26

Odds ratio for leukaemia cases (age <20 years)in Rhode Island (Fulton et al, 1989)

Exposure and address categories	Case addresses	Control addresses	Odds ratio (95% confidence interval)
All leukaemia			
Any case address			
Very low	46	56	-
Low	50	56	1.09 (0.63-1.88)
High	55	56	1.20 (0.70-2.05)
Very high	48	56	1.04 (0.60-1.81)
Acute leukaemia			
Stable case address*			
Very low	16	56	-
Low	19	56	1.19 (0.55-2.54)
High	15	56	0.94 (0.42-2.08)
Very high	7	56	0.44 (0.17-1.15)

*Same birth and diagnosis address.

Table 27

Wiring configurations in Denver, Colorado area, and adult cancer cases (Wertheimer and Leeper, 1982)

Location	Code	Mean magnetic field (nanotesla)	Cases addresses	Controls addresses	Odds ratio (95% confidence interval)	χ^2 for trend	% relative risk+ per nanotesla (95% confidence interval)
Longmont and Boulder	End pole	53	53	68	1.0		
	OLCC	71	262	287	1.17 (0.79-1.74)	10.1; p=0.15%	0.47 (0.18-0.76)
	OHCC	122	146	129	1.45 (0.94-2.23)		
	VHCC	212*	54	31	2.23 (1.26-3.95)		
Denver Suburbs	End pole	53	17	39	1.0		
	OLCC	71	150	147	2.34 (1.27-4.32)	5.13; p=2.4%	0.53 (0.07-0.99)
	OHCC	122	69	57	2.78 (1.42-5.42)		
	VHCC	212*	19	12	3.63 (1.45-9.12)		
Central Denver	End pole	53	29	41	1.0		
	OLCC	71	230	225	1.45 (0.87-2.41)	0.60; p=44%	0.13 (-0.20-0.45)
	OHCC	122	115	112	1.45 (0.84-2.50)		
	VHCC	212*	35	31	1.60 (0.81-3.14)		
All combined	End pole	53	99	148	1.0		
	OLCC	71	642	659	1.46 (1.10-1.92)	12.9; p0.03%	0.36 (0.16-0.55)
	OHCC	122	330	298	1.66 (1.23-2.23)		
	VHCC	212*	108	74	2.18 (1.48-3.22)		

* Estimate.

+ Logistic model (Breslaw and Day, 1980).

Table 28

Wertheimer-Leeper C-ratios by age of case

Age range (years)	Number of address pairs in which		C-ratio (95% confidence interval)
	Case high	Control high	
19–54	139	69	201 (151–269)
55–69	188	165	114 (92–140)
70+	182	133	137 (109–171)

Table 29

Wertheimer-Leeper C-ratios by type of cancer

Type	Number of address pairs for which		C-ratio (95% confidence interval)
	Case high	Control high	
Nervous system	25	11	227 (112-462)*
Leukaemia	23	23	100 (56-178)
Hodgkin's lymphoma	12	8	150 (61-367)
Non-Hodgkin's lymphoma	26	18	144 (79-263)
Multiple myeloma	8	3	267 (71-1005)
Ovary	18	14	129 (64-259)
Uterus	18	6	300 (119-756)*
Breast	87	53	164 (117-231)*
Prostate	30	19	158 (89-281)
Kidney	10	6	167 (61-459)
Bladder	24	14	171 (89-331)
Mouth	13	8	163 (67-392)
Pancreas	30	18	167 (93-299)
Stomach	14	16	88 (43-179)
Colon/rectum	61	55	111 (77-160)
Bronchus/lung	52	44	118 (79-177)
Other/unknown	58	51	114 (78-166)
Total	509	367	139 (121-159)*

*Significant at 5% level.

Table 30

Cases of adult ANLL and controls by wiring configuration (Sevenson, 1988) 3-10 years before reference data

Wire code or magnetic field	Cases addresses	Controls addresses	Odds ratio* (95% confidence interval)
Wertheimer-Leeper coding			
VLCC	42	44	
OLCC	21	37	0.60 (0.29-1.22)
OHCC	21	23	0.77 (0.35-1.68)
VHCC	5	6	0.79 (0.22-2.89)
Kaune estimate (nt)			
0-50	29	28	
51-199	46	64	0.69 (0.37-1.32)
200+	14	18	0.75 (0.31-1.80)

*As given by authors and controlled for age, sex, smoking, family income and race.

Table 31

Relative risk estimates per nanotesla for adult ANLL as given by Stevens, 1987, based on magnetic field measurements at time of interview

Measurement conditions	% relative risk per nanotesla (95% confidence interval)
Low power use (unweighted)	0.13 (-0.28-0.54)
Low power use (weighted)	-0.02 (-0.47-0.43)
High power use (unweighted)	0.25 (-0.14-0.64)
High power use (weighted)	0.15 (-0.28-0.58)

Table 32

Analysis of all childhood cancers in terms of wire codes (and their mean magnetic fields at low power consumption) based on data from Savitz, 1988

Wire code	Mean magnetic field (nanotesla)	Cases addresses	Controls addresses	Odds ratio (95% confidence intervals)	χ^2 for trend	% relative risk per nanotesla* (95% confidence interval)
At birth						
Buried	49	29	26			
VLCC	53	10	6	1.49 (0.48-4.68)	0.55	0.29 (-0.47-1.04)
OLCC	71	24	38	0.57 (0.27-1.18)	p=46%	
OHCC	122	28	20	1.26 (0.57-2.74)		
VHCC	212	4	3	1.20 (0.28-4.75)		
2 years before diagnosis						
Buried	49	36	47			
VLCC	53	11	15	0.96 (0.39-2.33)	4.80	0.73 (0.07-1.40)
OLCC	71	50	56	1.17 (0.65-2.08)	p=2.9%	
OHCC	122	30	28	1.40 (0.71-2.74)		
VHCC	212	8	2	5.22 (1.11-17.30)		
At diagnosis						
Buried	49	95	88			
VLCC	53	29	17	1.58 (0.81-3.07)	4.21	0.47 (0.03-0.91)
OLCC	76	107	102	0.97 (0.65-1.45)	p=4.1%	
OHCC	122	70	44	1.47 (0.92-2.37)		
VHCC	212	19	8	2.20 (0.92-5.28)		

* Logistic model (Breslaw and Day, 1980).

Table 33

Analysis of all childhood cancers in terms of magnetic fields under conditions of low power use based on data from Savitz, 1988

Magnetic field range (nanotesla)	Mean magnetic field (nanotesla)	Cases addresses	Controls addresses	Odds ratio (95% confidence intervals)	χ^2 for trend	% relative risk per nanotesla+ (95% confidence intervals)
At birth						
0-<65	38	24	45			
65-<100	83	11	10	2.06 (0.77-5.55)	0.72	-0.20 (-0.67-0.27)
100-<250	175	9	16	1.05 (0.41-2.74)	p=40%	
250+	350*	1	6	0.42 (0.08-2.18)		
2 years before diagnosis						
0-<65	38	49	80			
65-<100	83	12	19	1.03 (0.46-2.31)	0.42	0.12 (-0.24-0.48)
100-<250	175	17	22	1.26 (0.61-2.61)	p=52%	
250+	350*	4	5	1.32 (0.38-4.55)		
At diagnosis						
0-<65	38	75	134		1.08	
65-<100	83	20	28	1.28 (0.67-2.42)	p=30%	0.13 (-0.12-0.39)
100-<250	175	23	33	1.25 (0.68-2.28)		
250+	350*	10	12	1.49 (0.61-3.61)		

* Estimate.

+ Logistic model (Breslaw and Day, 1980).

Table 34

Analysis of all childhood cancers in terms of magnetic fields under conditions of high power use based on data from Savitz, 1988

Magnetic field range (nanotesla)	Mean magnetic field (nanotesla)	Cases addresses	Controls addresses	Odds ratio (95% confidence interval)	χ^2 for trend	% relative risk per nanotesla+ (95% confidence interval)
At birth						
0- <65	38	23	35			
65-<100	83	5	12	0.63 (0.20-2.04)	0.50	
100-<250	175	15	20	1.14 (0.49-2.67)	p=48%	-0.15 (-0.56-0.26)
250+	350*	2	8	0.44 (0.11-1.78)		
2 years before diagnosis						
0- <65	38	37	61			
65-<100	83	15	19	1.30 (0.59-2.87)	0.15	0.06 (-0.23-0.34)
100-<250	175	22	33	1.10 (0.56-2.16)	p=70%	
250+	350*	9	12	1.24 (0.48-3.22)		
At diagnosis						
0- <65	38	61	99			
65-<100	83	23	33	1.13 (0.61-2.10)	0.05	0.03 (-0.21-0.26)
100-<250	175	32	54	0.96 (0.56-1.65)	p=82%	
250+	350*	13	18	1.17 (0.54-2.56)		

* Estimate.

+ Logistic model (Breslaw and Day, 1980).

Table 35

Analysis of childhood leukaemia in terms of magnetic fields under conditions of low power use at diagnosis based on data from Savitz, 1988

Magnetic feld range (nanotesla)	Mean magnetic field (nanotesla)	Cases addresses	Controls addresses	Odds ratio (95% confidence interval)	χ^2 for trend	% relative risk per nanotesla+ (95% confidence intervals)
0- <65	38	21	134			
65-<100	83	4	28	0.99 (0.35-2.82)	1.71	0.24 (-0.12-0.61)
100-<250	175	7	33	1.35 (0.53-3.45)	p=19%	
250+	350*	4	12	2.17 (0.71-6.64)		

Table 36

Analysis of childhood brain tumours in terms of magnetic fields under conditions of low power use at diagnosis based on data from Savitz, 1988

Magnetic field range (nanotesla)	Mean magnetic field (nanotesla)	Cases addresses	Controls addresses	Odds ratio (95% confidence intervals)	χ^2 for trend	% relative risk per nanotesla+ (95% confidence intervals)
0- <65	38	15	134			
65-<100	83	4	28	1.37 (0.46-4.04)	0.21	
100-<250	175	4	33	1.13 (0.39-3.30)	p=65%	0.11 (-0.36-0.57)
250+	350*	2	12	1.64 (0.42-6.35)		

* Estimate.

\+ Logistic model (Breslaw and Day, 1980).

Table 37

Association of traffic density with wiring code (Savitz, 1988)

Wiring code	500+ vehicles per day	<500 vehicles per day	Odds ratio (95% confidence interval)
Buried	10	43	
VLCC	9	12	3.23 (1.07-9.73)
OLCC	25	63	1.71 (0.74-3.91)
OHCC	21	27	3.34 (1.37-8.17)
VHCC	6	2	12.20 (2.20-43.28)

χ^2 for trend = 8.87; p = 0.3%.

Table 38

Childhood cancer risk by traffic density (Savitz, 1988)

Traffic density	Cases addresses	Controls addresses	Odds ratio (95% confidence interval)
<500 vehicles per day	213	194	
500+ vehicles per day	28	14	1.82 (0.93-3.56)

Table 39

Average ages at diagnosis according to wiring configurations (Savitz, 1987)

Wire code	Average age at diagnosis	
	Cases	Controls
Buried	5.5	6.5
VLCC	5.4	8.5
OLCC	7.8	7.4
OHCC	6.8	6.8
VHCC	7.1	5.4

Table 40

Cases and controls by number of electrical appliances in use within the home (Savitz, 1987)

Number of appliances	Cases	Controls	Odds ratio (95% confidence interval)
0–2	30	17	
3–4	80	69	0.66 (0.33–1.29)
5–6	81	74	0.62 (0.32–1.22)
7+	47	44	0.61 (0.29–1.25)

Table 41

Odds ratios for childhood cancer addresses by proximity to an electrical construction (Tomenius, 1986)

Type of construction	Cases addresses	Controls addresses	Odds ratio (95% confidence interval)
None within 150 m	1010	892	
Electric subway	20	17	1.04 (0.54-2.00)
Electric railway	36	22	1.45 (0.84-2.47)
Transformer	12	14	0.76 (0.35-1.65)
Substation	7	5	1.24 (0.39-3.91)
6-200 kV transmission lines	12	6	1.77 (0.66-4.73)
200 kV transmission lines	32	12	2.17 (1.13-4.17)
All constructions within 150 m	119	77	1.36 (1.01-1.84)

Table 42

Odds ratios for childhood cancer addresses by magnetic field levels and proximity to an electrical construction (Tomenius, 1986)

Field and construction	Case addresses	Control addresses	Odds ratio (95% confidence interval)
<300 nT, no construction	983	882	
≥300 nT, no construction	27	10	2.42 (1.17-5.03)
<300 nT, plus construction	112	73	1.38 (1.01-1.87)
≥300 nT, plus construction	7	4	1.57 (0.46-5.38)

Table 43

Odds ratios for childhood cancer addresses by type of address (Tomenius, 1986)

Type of address and magnetic field	Case addresses	Control addresses	Odds ratio (95% confidence interval)
Stable birth and diagnosis			
<300 nT	242	407	
≥300 nT	10	3	5.61 (1.53-20.57)
Birth address			
<300 nT	661	673	
≥300 nT	21	8	2.67 (1.18-6.08)
Diagnosis address			
<300 nT	676	689	
≥300 nT	23	9	2.60 (1.20-5.67)

Table 44

Odds ratio for childhood cancer addresses by type of cancer (Tomenius, 1986)

Type of cancer and magnetic field	Case addresses	Control addresses	Odds ratio (95% confidence interval)
Leukaemia			
<300 nT	239	202	
≥300 nT	4	10	0.34 (0.10–1.09)
Lymphoma			
<300 nT	130	115	
≥300 nT	2	1	1.77 (0.16–19.77)
Nervous system			
<300 nT	281	250	
≥300 nT	13	3	3.86 (1.09–13.69)
Other malignant			
<300 nT	352	309	
≥300 nT	11	0	∞ (2.18–∞)
Benign			
<300 nT	93	79	
≥300 nT	4	0	∞ (0.54–∞)
All			
<300 nT	1095	955	
≥300 nT	34	14	2.12 (1.13–3.97)

Table 45

Odds ratios for tumour cases (0-18 years) in the County of Stockholm by magnetic field strength (Tomenius,1982)

Magnetic field range (nanotesla)	Approximate mean field (nanotesla)	Cases	Controls	Odds ratios (95% confidence interval)
0-99	50	902	751	1.00
100-199	150	159	167	0.79 (0.63-1.01)
200-299	250	34	37	0.77 (0.48-1.23)
300-399	350	12	5	2.00 (0.70-5.70)
>400	450*	22	9	2.04 (0.93-4.45)

* The median value for this dose group is 450 nanotesla.
χ^2 for trend = 0.09; p = 76%.

Table 46

Standardised mortality ratios by distance from electrical installations in East Anglia (McDowall, 1986)

Cause of death	9th ICD code	Distance (m) 0-14 SMR (95% CI)	15-34 SMR (95% CI)	0-34 SMR (95% CI)	35-50 SMR (95% CI)
All neoplasms	140-208	103 (68-50)	105 (85-128)	105 (87-126)	95 (76-117)
Cancer of stomach	151	50 (1-279)	107 (46-210)	95 (48-188)	122 (56-231)
Lung cancer	162	215 (118-361)	119 (79-171)	140 (101-193)	103 (67-152)
Breast cancer	174	37 (1-206)	122 (61-219)	102 (57-185)	110 (53-202)
Leukaemia	204-208	143 (4-796)	77 (9-278)	91 (28-297)	120 (25-351)
Other lymphatic	200-203	333 (69-974)	59 (7-212)	117 (46-294)	147 (48-343)
Diseases of the circulatory system	390-459	94 (71-122)	84 (72- 98)	86 (75- 99)	80 (68- 93)
Respiratory diseases	460-519	127 (77-195)	103 (79-134)	108 (86-137)	83 (61-111)

Table 47

Odds ratio for all neoplasms with distance from electrical installation in East Anglia based on McDowall, 1986

Distance (m)	Cases	Expected	Odds ratio (95% confidence interval)
35–50	89	93.7	
15–34	97	92.4	1.11 (0.74–1.66)
0–14	27	26.2	1.08 (0.59–2.00)

Table 48

Relative risks of leukaemia in comparison with cancer controls with distance from electrical substations (Coleman et al, 1988)

Distance from substation (m)	Leukaemia addresses	Cancer addresses	Odds ratio (95% confidence interval)
≥100	430	796	
50–99	244	456	0.99 (0.82–1.20)
25–49	62	129	0.89 (0.64–1.23)
0–24	35	51	1.27 (0.81–1.98)

χ^2 for trend 0.04; p = 84%.

Table 49

Relative risks of leukaemia and other cancers with distance from electrical substations based on Coleman et al, 1988

Distance from substation (m)	Leukaemia and cancer control addresses	Population control addresses	Odds ratio (95% confidence interval)
≥100	329	145	
50–99	154	69	0.98 (0.70–1.39)
25–49	32	13	1.08 (0.55–2.13)
0–24	14	4	1.43 (0.51–4.00)

χ^2 for trend 0.20; p = 65%.

Table 50

Summary of residential population studies of associations between cancer and electrical installations

Study	Area	Time	Age range (years)	Association with proximity to electrical installations		Association with magnetic field exposure	
				Leukaemias	All cancers	Leukaemias	All cancers
Childhood studies							
Wertheimer and Leeper, 1979	Denver	1950-1973	<19	Significant	Signficant	NA	NA
Fulton et al, 1980	Rhode Island	1967-1975	<20	Not significant	NA	NA	NA
Myers et al, 1990	Yorkshire	1970-1979	<15	NA	Very slight, insignificant	NA	Very slight, insignificant
Tomenius, 1982, 1986	Stockholm County	1959-1973	<18	NA	Significant	Not significant	Not significant
Savitz, 1987	Denver	1970-1979	<15	Significant	Significant	Not significant	Not significant
All ages							
McDowall, 1986	East Anglia	1971-1983	All	None	Very slight, insignificant	NA	NA
Coleman et al, 1988	South London	1965-1980	All	Very slight, insignificant	Very slight, insignificant	NA	NA
Adults							
Wertheimer and Leeper, 1982 1987	Denver	1967-1975	>19	Not significant	Significant	NA	Significant
Stevens, 1987	Washington State	1981-1984	20-79	None	NA	None	NA
Sevenson et al, 1988							

NA = Not Available

Table 51

Factors associated with increased risk of childhood cancer in the Denver Studies of Savitz et al

Factor	Relative risk (95% confidence interval)	Reference
Wiring codes 2 years before diagnosis: OHCC and VHCC versus buried and VLCC	1.67 (0.91-3.08)	Savitz, 1988
Magnetic fields 2 years before diagnosis: >100 nanotesla versus <65 nanotesla	1.14 (0.62-2.10)	Savitz, 1988
Traffic density		
>500 vehicles per day versus <500	1.70 (1.0-2.86)	Savitz, and Feingold, 1989
>10,000 vehicles per day versus <500	3.06 (1.12-8.37)	
Smoking by both parents	1.50 (0.9-2.6)	John et al, 1991
X-ray of fetus	1.44 (0.84-2.45)	Savitz, 1988

Table 52

Childhood cancer mortality rates in United Kingdom and Northern Ireland, 1988 (OPCS, 1990)

Age group and region	Mortality rates per 1,000,000 all cancers (relative to England)	
	Male	Female
Age 1-4 years		
England	43.7 (1.00)	55.1 (1.00)
Wales	52.8 (1.19)	27.7 (0.50)
Scotland	45.7 (1.05)	31.9 (0.58)
Northern Ireland	53.3 (1.22)	18.9 (0.34)
Age 5-14 years		
England	40.9 (1.00)	33.9 (1.00)
Wales	55.0 (1.34)	52.4 (1.55)
Scotland	55.4 (1.35)	32.4 (0.96)
Northern Ireland	60.5 (1.48)	39.5 (1.17

Table 53

Odds ratios for neuroblastoma in children whose fathers were employed in electrical or electronics occupations (Spitz and Johnson, 1985)

Father's occupation	Cases	Controls	Odds ratio (95% confidence interval)
Not electrical or electronics	133	256	
Electronics	6	1	11.5 (1.4–96.9)
Electricians, linesmen, welders, utility workers	7	11	1.2 (0.5–3.2)
Electrical equipment salesmen, repairmen	4	4	1.9 (0.5–7.8)
All electrical and electronics occupations	17	16	2.0 (1.0–4.2)

Sources	Region		Wavelength	Frequency
Magnets, earth's field; friction; batteries	Static			Zero
Electric power lines and cables Domestic and industrial appliances Induction heaters	EXTREMELY LOW FREQUENCIES	ELF	(6000 km at 50 Hz) More than 100 km	Less than 3 kHz
Television sets Visual display units (VDUs) AM radio RF heat sealers FM radio Television Cellular telephones	RADIO FREQUENCIES (RF)	Very Low Frequency VLF	100 km to 10 km	3 kHz to 30 kHz
		Low Frequency LF	10 km to 1 km	30 kHz to 300 kHz
		Medium Frequency MF	1 km to 100 m	300 kHz to 3 MHz
		High Frequency HF	100 m to 10 m	3 MHz to 30 MHz
		Very High Frequency VHF	10 m to 1 m	30 MHz to 300 MHz
		Ultra High Frequency UHF	1 m to 10 cm	300 MHz to 3 GHz
Microwave ovens Communications Radar	MICROWAVES	Super High Frequency SHF	10 cm to 1 cm	3 GHz to 30 GHz
		Extra High Frequency EHF	1 cm to 1 mm	30 GHz to 300 GHz
		Infrared		

FIGURE 1 Electromagnetic fields and their sources

(1000 Hz = 1 kHz; 1000 kHz = 1 MHz; 1000 MHz = 1 GHz;1,000,000,000 Hz = 1 GHz)

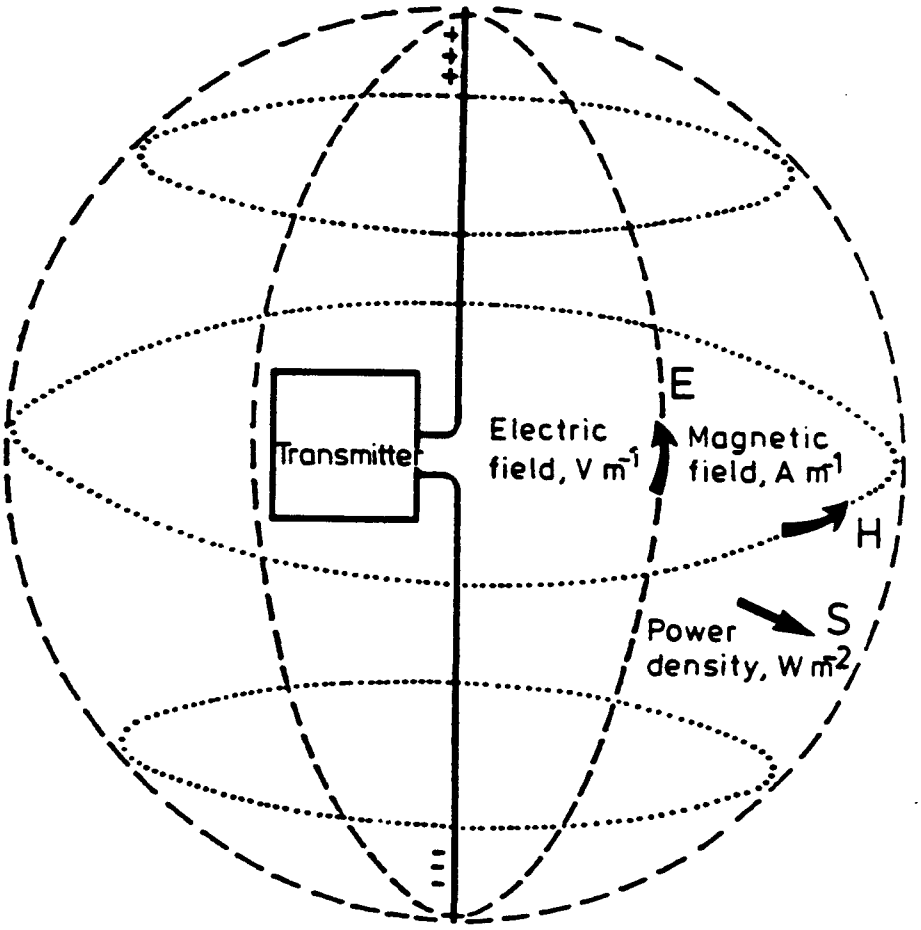

FIGURE 2 Electric and magnetic field emissions from a dipole antenna

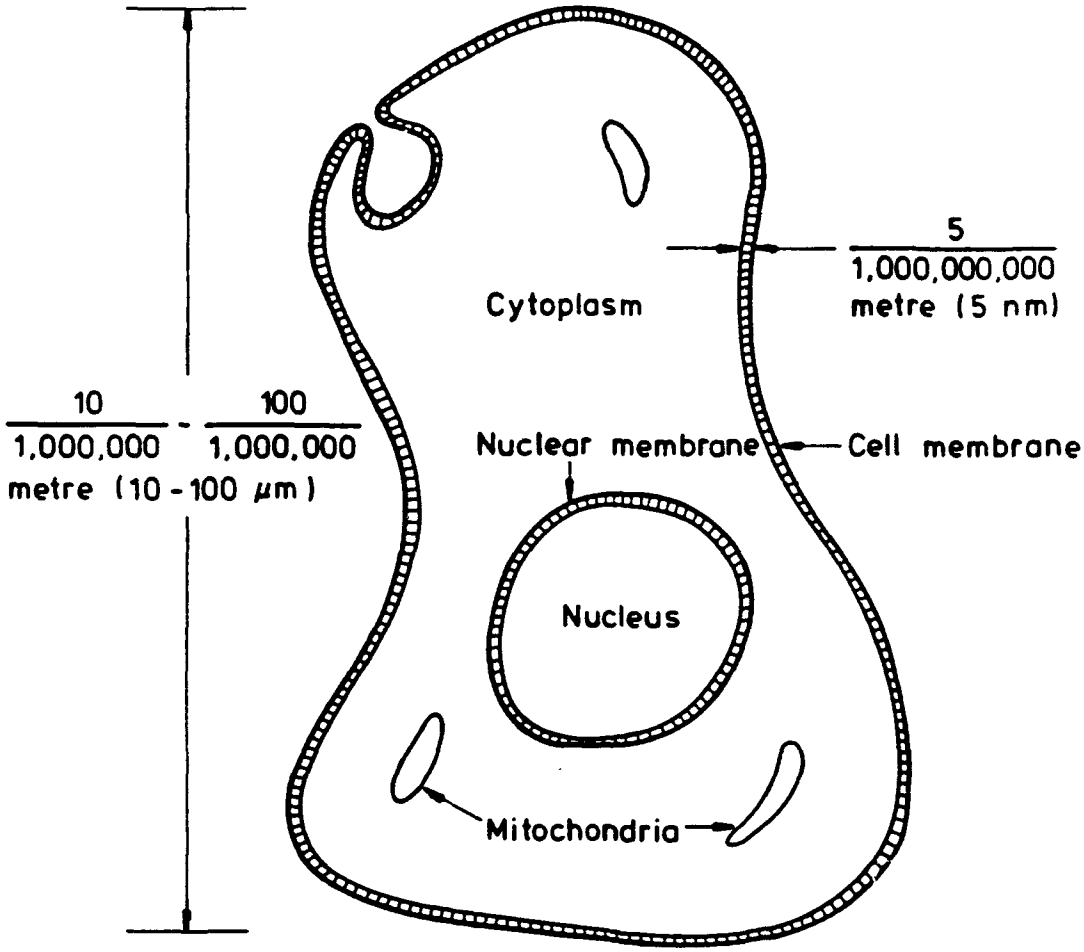

FIGURE 3 Schematic diagram of a cell

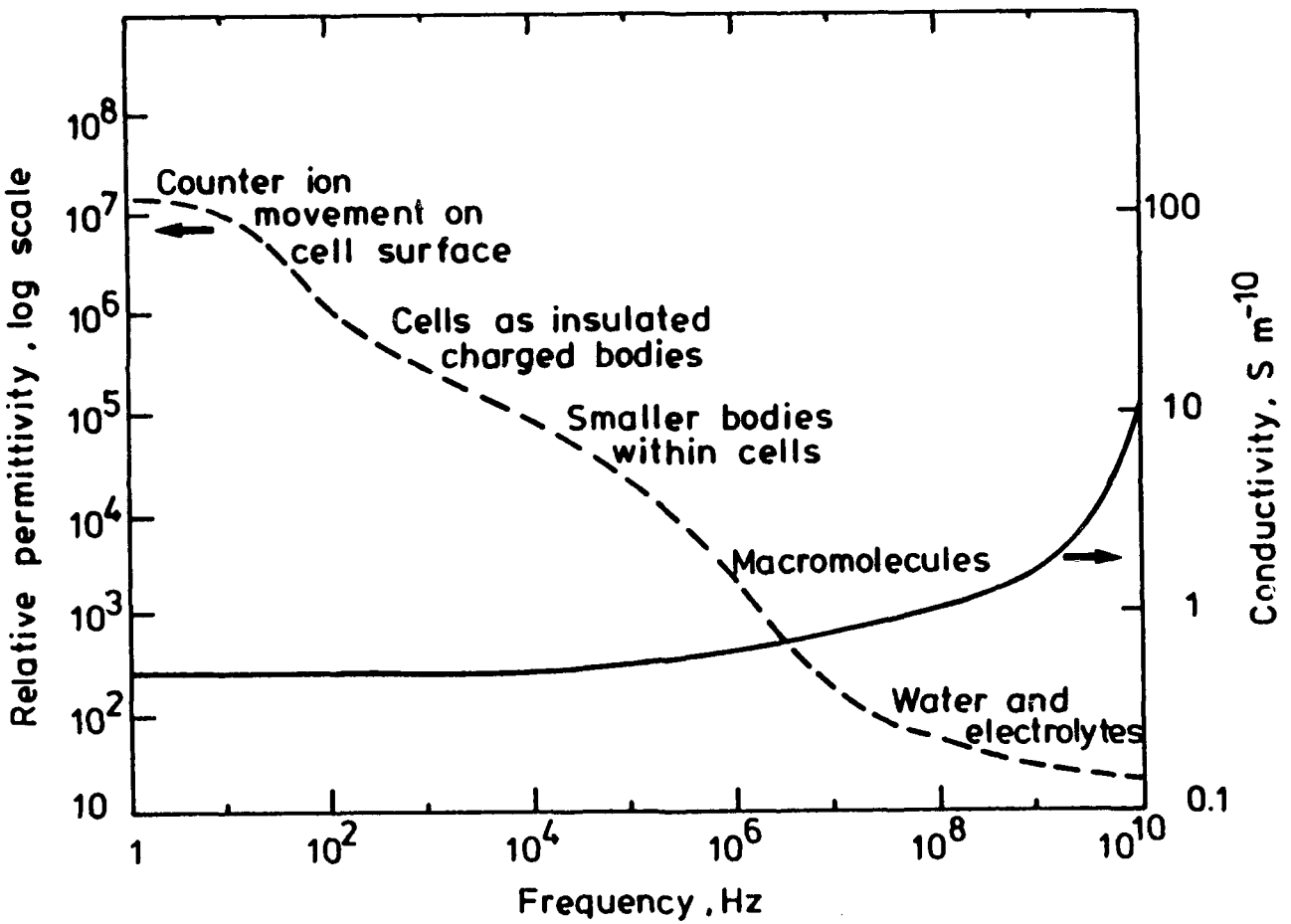

FIGURE 4 **Relative permittivity and electrical conductivity of muscle tissue indicating the possible cellular components that influence the relative permittivity at different frequencies**

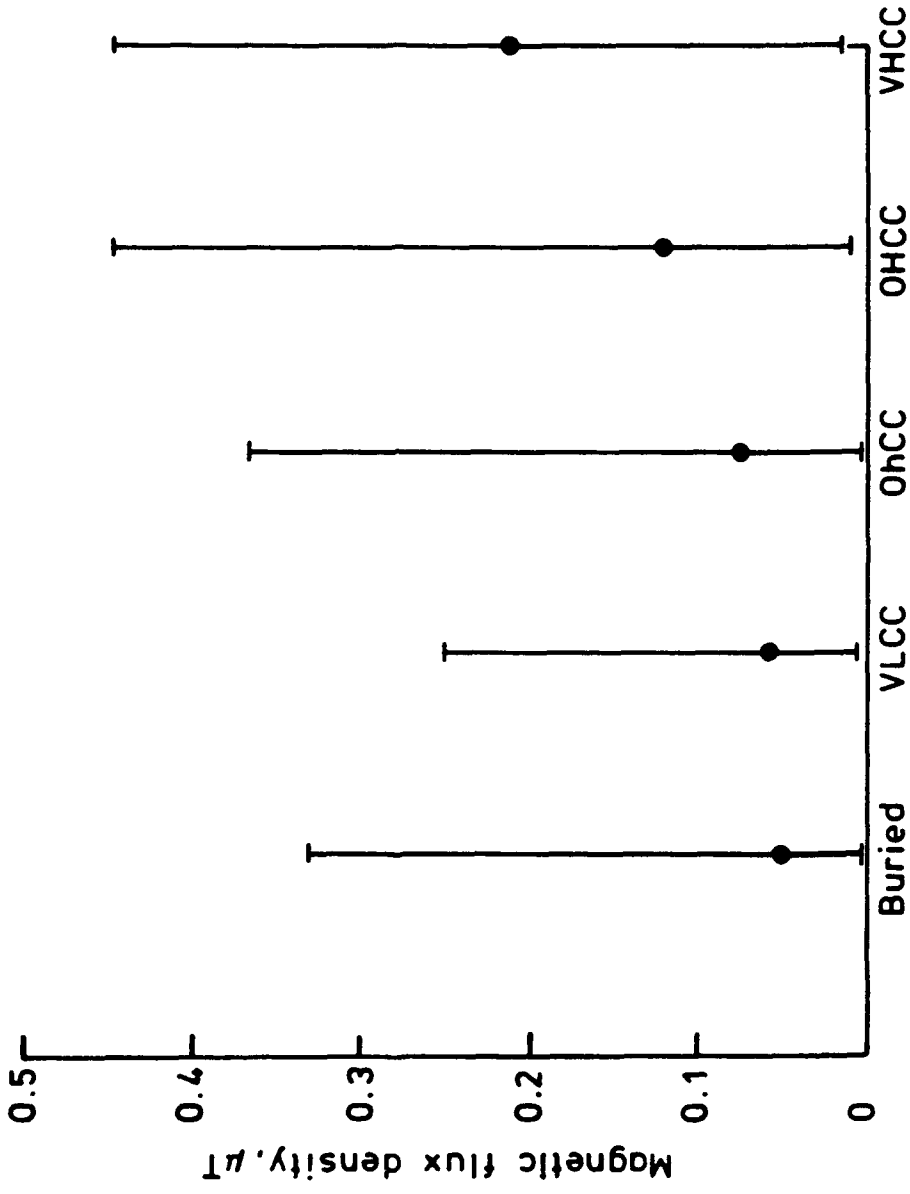

FIGURE 5 Medians and 10–90% ranges of magnetic flux density by wiring code (Savitz, 1987)

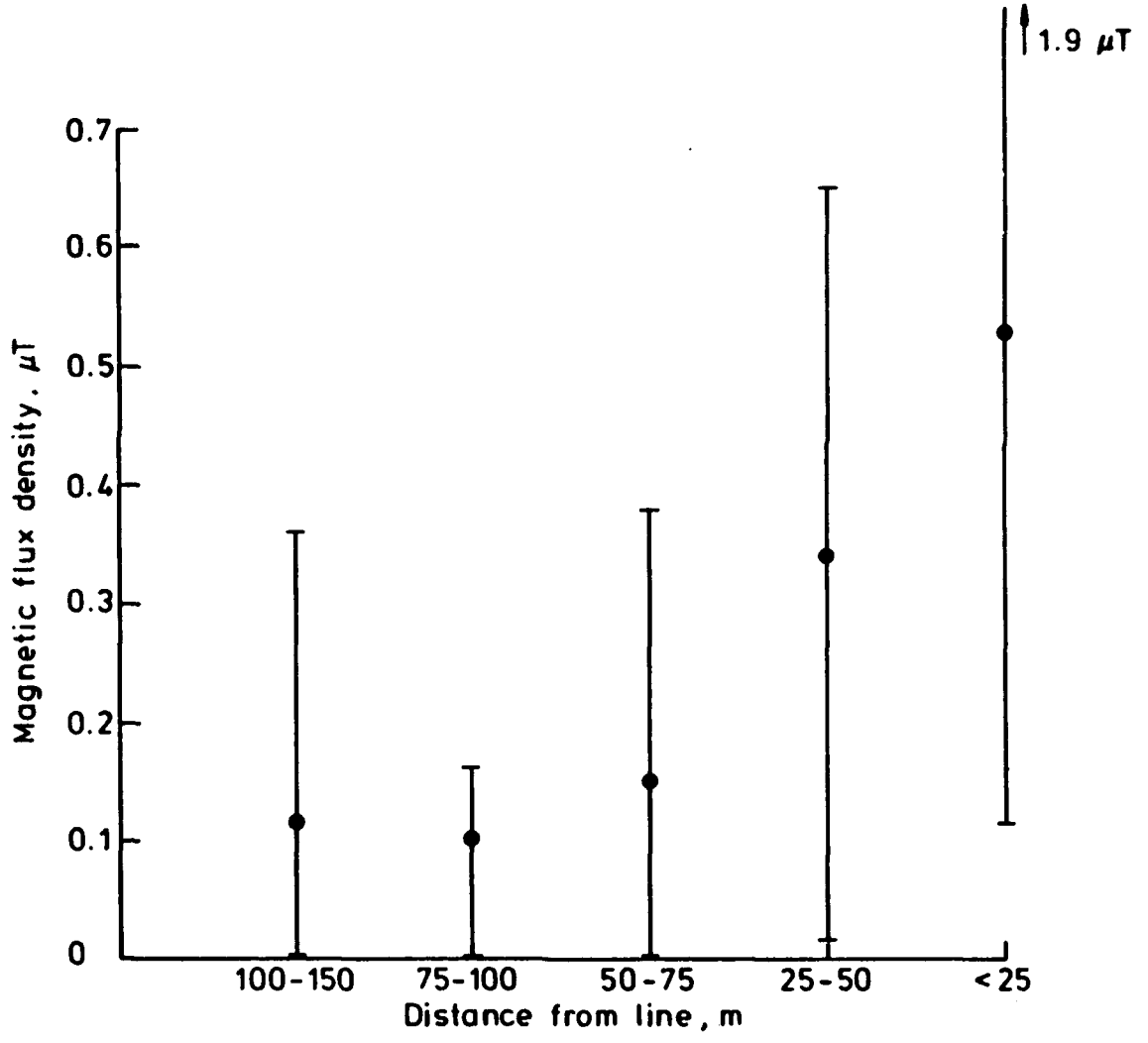

FIGURE 6 Medians and ranges of magnetic flux density with distance from 200 kV lines (Tomenius, 1986)

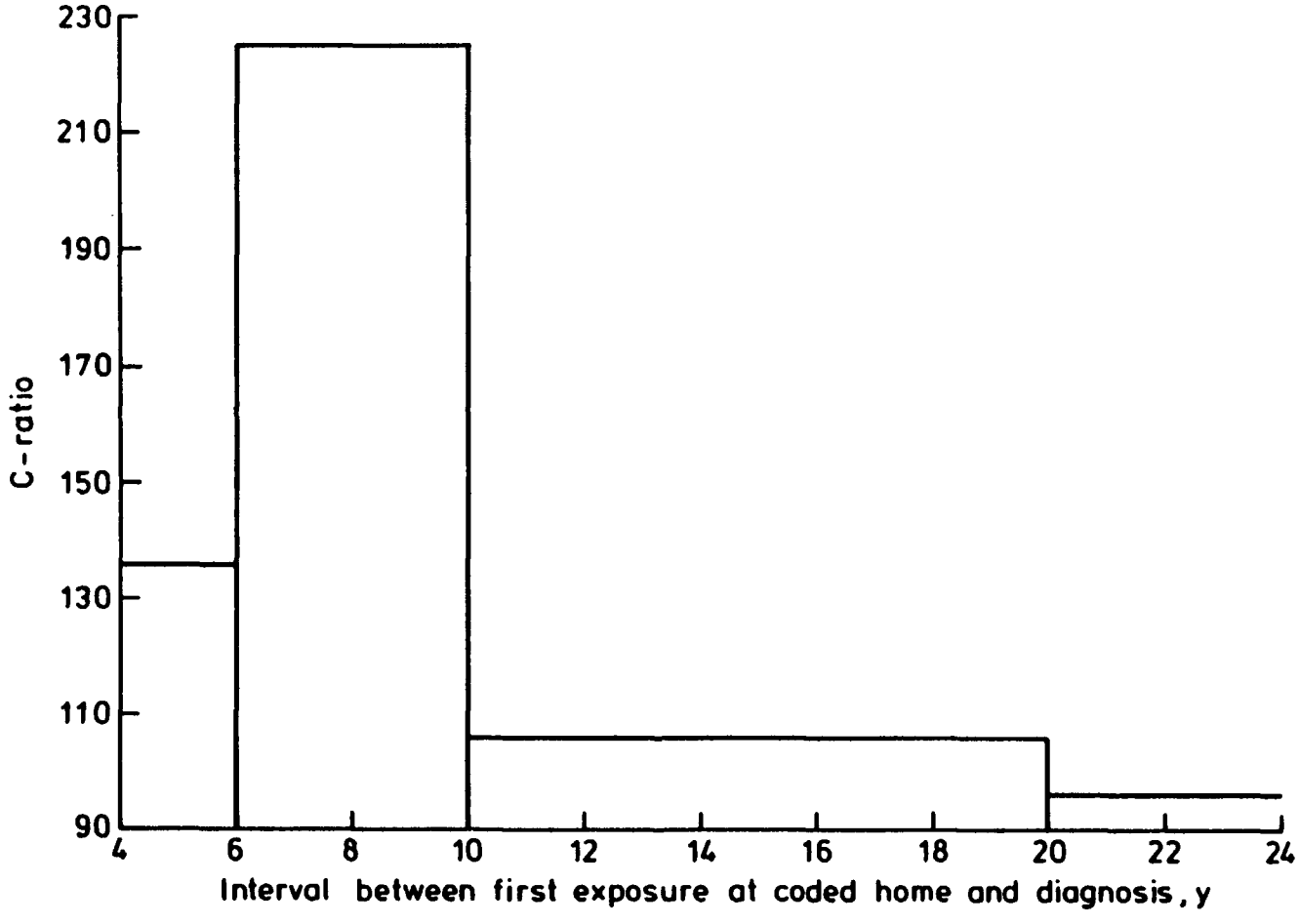

FIGURE 7 Relationship between Wertheimer–Leeper C-ratios and interval between first exposure at a coded home and cancer diagnosis

APPENDIX A

Aspects of the epidemiology of the leukaemias, neoplasms of the brain and malignant melanoma

The objective of this appendix is to describe some general aspects of the epidemiology of those neoplasms (cancers) that have been particularly associated with exposures to electromagnetic fields in order to provide a perspective to the possibility that they may be either initiated or promoted by the exposures. The diseases in question are childhood cancers in general, the leukaemias, neoplasms of the brain and malignant melanoma. The latter neoplasm of the skin is, however, more strongly associated with exposure to ultraviolet radiation – a non-ionising radiation of higher frequency than those radiations considered in this and its companion reports.

1 Mortality trends

It was noted in Section 1 that over the period in which the use of electromagnetic energy in the UK has markedly increased there has also been an increase in life expectancy. Some underlying factors in this increase are revealed by the data in Table A1. This gives the mortality rates in different age groups for all causes of death and for all neoplastic diseases for 1955, 1975 and 1985 for both men and women.

Over the past 30–40 years the mortality rates for children (age groups <1, 1–4 and 5–14 years) have fallen continuously, mainly due to the elimination of the mortality from infectious diseases. There has also been a decrease in the mortality from neoplastic diseases. The two main neoplastic diseases of childhood are leukaemias and malignant neoplasms of the brain and central nervous system (Greenbaum and Shuster, 1985), which account, respectively, for about 30% and 17% of all childhood cancers. The decrease in childhood cancer mortality between 1955 and 1985 is mainly due to increasingly successful treatments that ensure survival (Birch et al, 1988), rather than a decrease in incidence. Unfortunately, cancer registration, although better than it has been in the comparatively recent past, is still considerably less complete than cause of death registration. It is not entirely clear, therefore, whether the incidence of childhood cancers, as distinct from mortality, has changed during the past 30 years in the UK.

For the age groups between 15 and 64 years the mortality rate for all causes has also decreased, although not so dramatically as for children. Apart from the oldest working age group of 55–64 years the mortality rates from neoplastic diseases have also fallen. Within the overall rates there have been changes due to social factors, in particular in relation to lung cancer (Table A2). The incidence of this cancer is increased by a factor of ten or more among those who smoke. As may be seen, over the period 1975–1985 a relative decrease in the smoking habits of men and a relative increase in the smoking habits of women have decreased the mortality from lung cancer in the 35–74 years age group of the former and increased it in the latter. Another important neoplastic disease in men, prostate cancer, does not appear to have changed in the working age groups, but as for the mortality from most other neoplastic diseases, has increased in the elderly (Table A3). Breast cancer mortality, a major neoplastic disease of women, has decreased in the working age groups (Table A4), but also increased in the elderly.

In the elderly of 65 years and over, although the overall mortality rates have also decreased, those for the neoplastic diseases have increased. It must be remarked, however, that it is difficult to interpret changes in the mortality rates for older age groups. Possible reasons for apparent increases are improvements in the medical care and diagnosis of the elderly so that neoplastic diseases at these ages are more often recognised, medical treatments that postpone death from neoplastic diseases to later ages, and possibly changes in the environment. The last could include increased exposure to ionising radiation from medical procedures (UNSCEAR, 1977, Kendall et al, 1980), to chemical pollutants and to non-ionising electromagnetic fields. However, any hypothesis linking environmental factors to an increase in cancer rates must take into account current hypotheses of carcinogenesis as well as the general facts of the age dependence of some cancers.

2 Mechanisms for the induction of neoplastic diseases

The fact that the incidence of fatal neoplastic diseases increases more or less exponentially with age, together with the idea that cancers arise from a change to a single cell in the affected tissue of the body, led to the hypothesis that more than one and possibly three or four steps were required to transform a normal cell into a malignant cancer (Armitage and Doll, 1954). The incorporation into this hypothesis of the concept that some of these steps might confer a growth advantage to the partially transformed cells, which caused them to increase in numbers relative to untransformed cells, enabled the exponential increase in cancer incidence with age to be explained by a two-step hypothesis (Armitage and Doll, 1957). However, some cancers show peaks of incidence or occur only or predominantly in childhood. One of the forms of leukaemia, acute lymphoblastic leukaemia, is an example of the former and a cancer of the eye, retinoblastoma, an example of the second. These can be accommodated within the two-stage hypothesis, which is illustrated in Figure A1 (Moolgavkar and Knudsen, 1981). Techniques of genetic recognition have verified that the majority of malignant neoplasms do in fact originate from the transformation of a single cell (Fialkow, 1976).

The general hypothesis illustrated in Figure A1 is that an initial transforming event occurs in a normal cell (NC) either spontaneously or as the result of the action of a chemical or physical agent. This results in an intermediate cell (IC), which may die out or be removed by the immunological defences of the body or may propagate itself by division to form a clone or colony of partially transformed cells. A further transforming event changes the intermediate cell or one of a clone of such cells into a malignant cancer cell (MC). As part of this hypothesis it is suggested that benign non-metastasising neoplasms might consist of colonies of intermediate cells. Any factor which increases the population of IC cells will very greatly increase the possibility of a final transformation producing a malignant cell. It is suggested that 'promoting agents', which do not themselves appear to cause cancer unless applied subsequently to a carcinogenic initiating agent, may act by increasing the proliferation of IC cells. It is also suggested that the first step of spontaneous transformation may be more likely in tissues in which there is a rapid turnover or proliferation of normal cells; such as the proliferation that occurs in almost all tissues of children as they grow, particularly in the early stages of their development, and in certain other tissues throughout life. Once the first step had occurred natural proliferation would increase the number of cells at risk from a second transformation. In addition, the first step might be genetically inherited or occur *in utero*. Children born with this inheritance would be more at risk and this risk

would be enhanced by the normal proliferation of their cells. This certainly appears to be the case for some children who develop retinoblastoma, although in other children both steps of transformation occur after birth. The transformation steps for this cancer consist of deletions from the homologous arms of a certain chromosome.

Whether or not the two-stage hypothesis of the development of neoplastic diseases is correct, either for all or for particular cancers, it seems undoubtedly the case that the transformation of normal cells into those of malignant neoplasms involves alterations to the nuclear DNA. These alterations can be produced by ionising radiations, by viral infections and by exogenous chemical agents (Knudsen, 1985; Hathaway, 1986). One of the best-known promoting agents, the phorbolester tetradecanol-phobol-acetate (TPA), appears to act on receptor molecules in the cell membrane involving a specific calcium-dependent and lipid-dependent protein kinase enzyme system, protein kinase C. TPA also involves the synthesis in cells of ornithine decarboxylase (Hathaway, 1986). While there is no convincing evidence as yet that electromagnetic fields at frequencies of up to 300 GHz are capable of producing direct modifications of cellular DNA, there is some evidence that such fields could influence the transport of calcium through cell membranes, the synthesis of ornithine decarboxylase within the cells, and aspects of the expression of DNA synthesis by cells (see Sienkiewicz et al, 1991, and Saunders et al, 1991). Consequently, although it is not thought that these fields are capable of initiating neoplastic diseases, there is some evidence that they may act as promoters in cases where there has been spontaneous initiation or initiation by some environmental agent (Easterly, 1981).

3 Leukaemias

The leukaemias are a group of neoplastic diseases of the white cells found in the blood and lymphatic system of the body. The form of leukaemia is recognised by the morphology of white cells involved, the clinical manifestations which involve the bone marrow, thymus, spleen and lymphatic system and the time course of the disease. The white cells and the organs mentioned are responsible for the immunological responses of the body to infections. As the details of these responses have become better understood, finer immunological distinctions have been made between the different types of white cell and hence of certain different types of leukaemia. Closely related neoplastic diseases are lymphomas and multiple myeloma.

All the white cells originate from the bone marrow and some of them are processed by the thymus or by the bursal equivalent tissue, whose exact site in the body is unknown, to become T or B type lymphocytes. These are cells of the lymphatic system. Other white cells from the bone marrow become part of the myeloid system, they are known as monocytes or granulocytes, which are further distinguished as neutrophils, eosinophils and basophils. These myeloid cells originate from the same stem cell system as the red cells or erythrocytes.

The forms of leukaemia which are distinguished by the different types of white cell involved and the clinical time course of the disease are illustrated in Figure A2 (Linet, 1985; Greaves, 1984). Unfortunately, the different types and subtypes of leukaemia are not always recognised in epidemiological studies. It is only since the 1960s that death certificates in the UK have provided reliable information about different types of leukaemia. Even so, death certificates do not distinguish many of the subtypes of leukaemia. Most of the discussion which follows will be in terms of the subdivisions recognised by the 8th and 9th International Classifications of Diseases, Injuries and Causes of Death (ICD). These are:

Type of Leukaemia	8th ICD	9th ICD
All leukaemia	204–207	204–208
Lymphoid leukaemia	204	204
Acute lymphoid leukaemia	204.0	204.0
Chronic lymphoid leukaemia	204.1	204.1
Unspecified	204.9	204.9
Myeloid leukaemia	205	205
Acute myeloid leukaemia	205.0	205.0
Chronic myeloid leukaemia	205.1	205.1
Unspecified	205.9	205.9
Monocytic leukaemia	206	206
Other specified leukaemia	207	207
Leukaemia of unspecified Cell type	207	208

The majority of deaths from leukaemia fall under the classifications of acute lymphoid leukaemia, ALL, chronic lymphoid leukaemia, CLL, acute myeloid leukaemia, AML, or chronic myeloid leukaemia, CML. The discussion of the epidemiological studies will be confined to these main forms and to the total of all leukaemias.

Changes in the mortality rates in the UK from leukaemia over the period 1955–1985 and separately for the four main types of leukaemia at different ages are shown in Tables A5–A7. In general, the incidence of leukaemia is greater in males than in females by a factor of about 1.5, except in childhood. The general features of the distribution in incidence between the different ages and the four main types are shown in Figure A3. Acute lymphoid leukaemia shows a peak of incidence in the 1–5 years age group and there is a slight indication of a further subpeak of incidence in the 15-20 years age group. The former is due to the subtype common-ALL and the second to the thymic subtype T-ALL (Greaves, 1984), Figure A4. Chronic lymphoid leukaemia is almost entirely a disease of adults and particularly of the elderly and unlike the other forms of leukaemia does not appear to be increased by exposure to ionising radiation. It is considered more likely than the other forms to have a genetic component (Linet, 1985; Linet and Cartwright, 1988).

The marked decrease in the mortality rate due to childhood leukaemia between 1950 and 1985 is almost certainly due to improved treatment for this disease. It is probable that the incidence of the disease has not decreased and may even have increased. In the age groups 15–64 years leukaemia mortality has also decreased slightly, possibly also due to improved treatment. In the elderly, over the age of 65 years, chronic lymphoid leukaemia and acute myeloid leukaemia have increased, but acute lymphoid leukaemia and chronic myeloid leukaemia have remained steady or decreased slightly. As remarked earlier these changes in the elderly are not easy to interpret.

3.1 Leukaemias in childhood

Leukaemia mortality in childhood does not appear to depend on the occupational class of the father, which is in marked contrast to the overall mortality rate in children (Table A8). However, districts with increased proportions of the higher occupational classes appear to have higher rates of childhood mortality from leukaemia (Cook-Mozaffari et al, 1989), and there are variations in the reported incidence of the disease by 20% or more between the different regions of England and Wales (Cartwright, 1989). Apparent clustering of childhood leukaemia cases has been noted for some time. The undoubted increase brought about by irradiation *in utero* for medical diagnosis has led to a suspicion that clusters found in the neighbourhoods of nuclear installations must be due to releases of radioactivity. However, it seems unlikely that the known releases can be entirely responsible for these clusters (Black, 1984; COMARE, 1986, 1988, 1989). It has been suggested that a causal factor is the response to an unidentified common infection encouraged by the mixing of populations (Kinlen, 1988, 1989). There is some evidence in the UK for this suggestion and it is supported by the marked increase of childhood leukaemia in Israel during the period 1950–1958 when the population almost doubled due to immigration (Davies et al, 1961). However, it was thought that this may have been due to the increased use of X-rays for diagnosis and treatment in the same period. Greaves, 1989, has pointed out that the suggestion by Kinlen of an infective link supports a more specific biological explanation of childhood leukaemia (Greaves and Chan, 1986). More recently, these childhood leukaemias have been linked to the father's exposure to ionising radiation (Gardner et al, 1990a, b).

The childhood peak in the 1–5 years age group which became apparent in the 1930s in the UK and somewhat later in the USA followed a period from the early part of the century during which childhood leukaemia increased at a rate of about 5% per year (Court-Brown and Doll, 1961). These authors suggested that the increase and the emerging peak were due to:

(a) improvements in diagnosis and accuracy of death certification,

(b) the introduction of sulphonomides and antibiotics which reduced the mortality from pneumonia and other infectious diseases;

(c) improvements in therapy that displaced the age specific mortality rates to later age groups;

(d) a true increase associated with an increase in affluence.

Greaves and Chan (1986), believe that the most important factor, as suggested by Stewart and Kneale (1969), was the reduction in pre-emptive death from infectious diseases that obscured the true incidence of leukaemia. This would explain why the childhood peak is not observed in most developing countries, where infections are still a major cause of child mortality. They suggest that the main reason for the childhood peak in common-ALL is due to the high proliferation rate in precursors of B-lymphocytes in early development. This proliferation rate can be further increased by the immunological response to infections. These cells, because of their function and active enzyme systems, are likely to be at high risk of spontaneous mutation. That is, the immunological response to common infections induces a proliferative stress and rearrangements of DNA in lymphocyte precursors and increases the risk of spontaneous mutation.

The Greaves–Chan hypothesis obviously has elements in common with the generalised hypothesis of cancer development described earlier. The correspondence was made even more explicit in a later paper (Greaves, 1988). In this paper Greaves suggests that the comparative lack of dependence on occupational class or genetic background of the incidence of childhood leukaemia implicates two spontaneous mutations or transformations as causal factors. The first occurring

in utero and followed by developmentally driven cell proliferation of the transformed cell line and the second arising in the course of further cell proliferation and modification in response to an immunological challenge arising after birth. This explanation would not exclude the fact that irradiation *in utero* also appears to be a causal factor (Stewart and Kneale, 1969) or the recent implication that irradiation of the father is another possible factor (Gardner et al, 1990a, b), since these could induce the first transformation step that normally occurs spontaneously. The Greaves explanation encompasses the different age dependencies of common-ALL and T-ALL (Figure A4): the first arising from B-lymphocyte precursors, which proliferate in response to immunological stress, and the second from T-lymphocyte precursors which do not respond to the same extent. Sex differences in the incidence of T-ALL are explained as resulting from the thymic response to levels of sex hormones. Greaves suggests that when immunological challenges occur later in life due to social factors or in the absence of the protecting effects of breast feeding, the proliferating stress in B-lymphocyte precursors may be more pronounced or less regulated. In affluent societies the infectious challenges to infants are reduced or occur at different periods of development than in the less affluent; these may be the factors that govern the peak in childhood incidence and increase the level when populations with different endemic infections and immunological competencies are mixed. The authors are unaware of any evidence that electromagnetic fields at the levels encountered in domestic situations increases the proliferation of any elements of the immunological system, although there are some indications that they might be affected by microwave exposure at high levels (see Kowalczuk et al, 1991; Sienkiewicz et al, 1991; Saunders et al, 1991). If the Greaves–Chan hypothesis is correct then any influence of electromagnetic fields on childhood leukaemia is likely to be insignificant compared to that of infections.

3.2 Leukaemias in adults

The two most common forms of leukaemia in adults are acute myeloid leukaemia and chronic lymphoid leukaemia Chronic myeloid leukaemia and acute lymphoid leukaemia also occur, but to a much lesser extent, (Figure A3).

In the 20–64 years age group for men (Table A9), and the 20–59 years age group for women (Table A10), the absolute mortality from all leukaemias as measured by SMR shows little dependence on occupational class. However, the proportional mortality ratios (PMR1 and PMR2) show a steep, decreasing gradient from the higher to the lower occupational classes that is more marked in men. This is explained by the equally sharp increase with diminishing occupational class of deaths from all causes, of which the major categories are all neoplastic diseases and those of the circulatory system.

Acute myeloid leukaemia, which is the most common form of leukaemia in the working age groups, shows the same pattern of SMR and PMR values as that for all the leukaemias. If anything, SMRs show even less variation with occupational class. The retirement age groups exhibit a similar occupational class dependence of PMRs to the working age groups, but with a less clear distinction between occupational classes I, II and III. (OPCS does not provide SMR values for the retirement age groups.) Chronic lymphoid leukaemia, which after acute myeloid leukaemia is the most common fatal leukaemia below the age of 50 years, shows the same occupational class gradient of PMR values for men. However, there appears to be an increase in the absolute rate as measured by SMR for the lowest occupational group V. A possible reason for this increase may be the greater difficulties in this occupational group of coping with medical regimes that enhance

survival from chronic diseases. However, this pattern does not appear in women. These patterns seem to be repeated in the case of chronic myeloid leukaemia.

For acute lymphoid leukaemia, which contributes less than 10% to the overall mortality rate from the leukaemias, both the absolute and relative rates in men appear to decrease with occupational class. The lower mortality rates make for poorer statistics which may confuse or even conceal any trends. The trends, if real, do not appear to occur among women or in the childhood forms of the disease.

The occupational class features of SMRs and PMRs for the leukaemias must be kept in mind when enquiring into their associations with exogenous agents. Unfortunately, very few epidemiological studies involving possible exposure to electromagnetic fields have made any distinction between the different forms of leukaemia or have addressed occupational class as a confounding factor, particularly when studying relative death rates. Overall, in comparison with the major causes of mortality, the absolute mortality rates from leukaemias have not changed over the past few decades and do not show much association with occupational class for the working age groups.

4 Malignant neoplasms of the brain

Under the 6th ICD malignant neoplasms of the brain were coded together with those of other parts of the central nervous system, for this reason the comparison of the changes in mortality rates is confined to 1975 and 1985 in Table A11. However, some of these neoplasms were classified under neoplasms of the central nervous system also in the 8th ICD. It is not possible to say what effect this may have had on the detailed comparison between the mortality rates in 1975 and 1985, although it may account for the small increases in the working age groups and some of the increase in the retirement age groups. Overall the change in coding appears to account only for an increase of between 3% and 4% (OPCS, 1983), thus implying that there has been a real increase at all ages, although not so marked below the age of about 35 years.

The occupational class dependence shown in Tables A9 and A10 is very similar to that exhibited by the leukaemias, ie a relatively constant absolute mortality rate as indicated by SMRs and a sharp decrease in relative rates measured by PMRs with decreasing occupational class. As for the leukaemias, neoplasms of the brain and central nervous system taken together show very little dependence in children on the father's occupational class, (Table A8).

Distinctions which depend on the position in the brain where they occur and the type of tissue involved are made for brain tumours. However, OPCS reports do not provide sufficient information to allow the time and occupational class dependencies to be derived in the detail given for the leukaemias.

5 Malignant melanoma

Malignant melanoma was classified under number 190 in the 6th ICD and under number 172 in both the 8th and 9th ICDs. There appears to have been a marked increase in the mortality rates from this disease from 1955 to 1985 (Table A12), although the small number of deaths makes this less clear at ages less than 35 years. In the retirement age groups the increase during the 10 year period 1975–1985, as for that for malignant neoplasms of the brain, is rather greater than that for the leukaemias. The difference is possibly not so marked over the 30 year period 1955–1985.

However, unlike either the leukaemias or malignant neoplasms of the brain, for malignant

melanoma the absolute mortality rate, particularly for men, decreases with occupational class, (Tables A9 and A10). Not surprisingly, the gradient in the PMRs is even more marked than for the other diseases. The principal causal factor identified for malignant melanoma is intermittent exposure to ultraviolet radiation, with a strong association with sunburn in early childhood (Longstreth, 1988). The increase in holidays abroad involving exposure to strong sunlight is conjectured to be part of the explanation for both the increasing mortality rate and the occupational class dependence of this disease.

6 Summary and conclusions

In striking contrast to mortality rates from all causes, and the major contributions to mortality of all neoplasms and diseases of the circulatory system, the mortality rates for the leukaemias, neoplasms of the brain and malignant melanoma do not increase in the lower occupational classes. This observation appears to hold for all age groups. Absolute mortality rates for the leukaemias and neoplasms of the brain appear to be independent of occupational class, while the absolute rate from malignant melanoma is highest in the upper occupational classes. This has been linked to lifestyle through exposure to ultraviolet radiation and intermittent sun bathing.

The childhood mortality rates from all causes and all neoplasms have fallen continuously and are now about 40% less than those experienced 30 or 40 years ago. Mortality rates from the leukaemias, mainly acute lymphatic leukaemia has fallen to an even greater extent, but this is probably due to improved treatment. It is not clear whether the incidence of this disease has remained constant or has increased. Mortality rates from malignant neoplasms of the brain appear to have remained constant.

In the working age groups, mortality from all causes and from all neoplasms has also fallen in the past 30 years. However, mortality rates from the leukaemias have remained constant and those from malignant neoplasms of the brain and malignant melanoma have increased – very markedly in the case of malignant melanoma. In these age groups two major neoplastic diseases, prostate cancer in men and breast cancer in women, have remained constant, while lung cancer has decreased in men and increased in women.

In the retired age groups, in which mortality rates from all causes have decreased by some 30% since 1955, those from all neoplasms appear to have increased by about the same percentage, although there are differences between men and women. In the same period mortality rates from the leukaemias and from malignant melanoma have more than doubled and those from malignant melanoma have increased by an even greater factor. However, rather similar increases have also occurred in the major neoplastic diseases of prostate, breast and lung cancer. Some of these apparent increases may be the result of better medical care and improved diagnosis for these age groups.

The absence of any marked dependency on occupational class for the mortality rates from the leukaemias and malignant neoplasms of the brain suggests that if any environmental factor is involved it is of a ubiquitous nature that does not depend on employment. In the case of malignant melanoma a plausible link to lifestyle has been made to explain the occupational class dependency. It is notable that mortality from those malignancies which have been particularly associated with possible exposure to electromagnetic fields, as well as from some of the major neoplastic diseases, have not decreased to the same extent in the working age groups as some other forms of neoplastic diseases and have increased more rapidly in the retirement age groups. This suggests that these

other neoplastic diseases have decreased due to improvements in lifestyles and, as suggested by Greaves (1988) in relation to childhood leukaemia, that those that have not decreased are almost entirely due to spontaneous transformations that are not influenced by the environment. Alternatively, they have been maintained or increased by some environmental factor or factors whose levels have been increasing and which do not affect the other neoplastic diseases. Two environmental factors which are more or less ubiquitous and which have increased since the 1950s are the use of medical X-rays for diagnosis, which has increased by about 2% per cent per year (Kendall et al, 1980), and the generation of electric power at about 7% per year (see Table 1). Radiofrequency transmission power has increased to an even greater extent. There have also, of course, been comparable increases in the use of insecticides, herbicides, the combustion products of fossil fuels and the use of plastics and petroleum products.

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Table A1

Mortality rates in England and Wales from all causes and all neoplasms, 1955, 1975 and 1985

Rates per million in different age groups

Age group (years)	Male: all causes			Female: all causes			Male: all neoplasms			Female: all neoplasms		
	1955	1975	1985	1955	1975	1985	1955	1975	1985	1955	1975	1985
<1	28,815	17,092	10,556	21,887	13,622	8,313	128	79	54	101	81	60
1-4	1,036	677	498	969	522	408	122	79	43	122	57	41
5-14	471	336	255	342	212	184	76	66	51	61	50	36
15-24	1,029	918	751	475	395	295	108	91	73	72	69	50
25-34	1,254	937	844	942	574	461	203	176	132	218	161	163
35-44	2,535	2,067	1,710	1,927	1,502	1,136	571	432	404	711	679	599
45-54	7,657	7,131	5,397	4,699	4,279	3,331	2,108	1,888	1,552	1,920	2,075	1,789
55-64	22,020	19,538	17,105	11,306	10,117	9,703	5,885	5,809	5,629	3,613	4,005	4,411
65-74	54,217	50,347	44,298	32,343	25,780	24,086	11,106	13,279	13,213	6,393	6,618	7,573
75-84	128,521	115,782	104,053	90,908	73,513	64,070	17,166	21,864	23,949	10,396	10,581	11,705
>85	256,243	241,368	223,133	222,862	193,098	178,289	17,487	24,904	32,741	13,743	14,901	18,024

Sources: Registrar General (1956); OPCS (1977, 1987).

Table A2

Mortality rates from lung cancer in England and Wales, 1975 and 1985 (8th and 9th ICD code 162): Rates per million in different age groups

Age group (years)	Male		Female	
	1975	1985	1975	1985
25–34	-	-	-	-
35–44	109	71	49	42
45–54	833	482	287	199
55–64	2,743	2,262	662	818
65–74	5,742	5,100	951	1,465
75–84	7,712	7,905	1,026	1,528
>85	5,096	7,643	929	1,240

Sources: OPCS (1977, 1987).

Table A3

Mortality rates from male prostate cancer for England and Wales, 1975 and 1985 (8th and 9th ICD code 185): Rates per million in different age group

Age group (yrs)	1975	1985
35–44	0	1
45–54	19	19
55–64	168	201
65–74	831	1,110
75–84	2,720	3,149
>85	4,504	6,566

Sources: OPCS (1977, 1987).

Table A4

Mortality rates from female breast cancer for England and Wales, 1975 and 1985 (8th and 9th ICD code 174): Rates per million in different age groups

Age group (years)	1975	1985
25–34	37	37
35–44	255	216
45–54	668	610
55–64	945	1,078
65–74	1,119	1,337
75–84	1,582	1,876
≥85	2,343	3,106

Sources: OPCS (1977, 1987)

Table A5

Mortality rates for all leukaemias for England and Wales (6th ICD code 204; 8th ICD codes 204-207, 9th ICD codes 204-208): Rates per million in different age groups

Age group (years)	Males			Females		
<1	15	19	6	21	23	9
1-4	44	23	9	60	23	10
5-14	26	31	18	23	21	12
15-24	25	23	21	15	15	12
25-34	22	21	25	18	16	13
35-44	34	22	30	26	26	21
45-54	56	55	43	43	35	40
55-64	105	117	109	61	73	75
65-74	205	258	277	110	130	156
75-84	244	480	569	131	267	325
≥85	90	749	916	120	383	556

Sources: Registrar General, (1956); OPCS (1977, 1987).

Table A6

Mortality rates by leukaemia type for England and Wales in males 1975 and 1985: Rates per million in different age groups

Age group (years)	ICD code							
	204.0		204.1		205.0		205.1	
	ALL		CLL		AML		CML	
	1975	1985	1975	1985	1975	1985	1975	1985
<1	13	0	0	0	6	0	0	0
1-4	17	5	0	1	4	1	2	0
5-14	6	13	0	0	6	4	0	0
15-24	10	11	0	0	10	7	1	2
25-34	3	3	0	0	10	12	5	6
35-44	2	5	0	1	13	15	4	8
45-54	2	3	7	5	26	22	14	11
55-64	7	6	35	31	44	48	19	16
65-74	10	9	78	80	86	105	46	48
75-84	20	21	176	208	143	149	87	78
≥85	17	14	366	427	139	210	122	105

Sources: OPCS (1977, 1987).

Table A7

Mortality rates by leukaemia type for England and Wales in females, 1975 and 1985: Rates per million in different age groups

Age group (years)	ICD code							
	204.0		204.1		205.0		205.1	
	ALL		CLL		AML		CML	
	1975	1985	1975	1985	1975	1985	1975	1985
<1	3	6	0	0	13	0	0	0
1–4	15	7	0	0	6	1	0	2
5–14	12	8	1	0	6	3	1	0
15–24	6	3	0	0	7	8	2	1
25–34	2	3	0	0	10	8	3	2
35–44	2	2	1	0	15	13	4	4
45–54	4	4	2	2	19	23	7	7
55–64	5	5	12	10	29	38	19	16
65–74	7	8	27	40	48	60	26	28
75–84	6	12	83	97	99	107	43	48
≥85	14	2	189	227	71	98	51	72

Sources: OPCS (1977, 1987)

Table A8

Standardised mortality ratios for children (1-14 years) in England and Wales by social class of father, 1970-1972

Social class	SMRs						Chi-squared test for trend
	I	II	IIIN	IIIM	IV	V	
Males							
All leukaemias 8th ICD: 204-207	77	95	140	95	87	140	$\chi^2 = 0.77$, $p = 0.38$
Neoplasms of brain and central nervous system 8th ICD: 191-192	93	95	105	95	99	145	$\chi^2 = 1.40$, $p = 0.24$
All causes	75	78	96	99	112	163	$\chi^2 = 247.1$, $p << 0.001$
Females							
All leukaemias 8th ICD: 204-207	117	96	122	94	97	105	$\chi^2 = 0.28$, $p = 0.60$
Neoplasms of brain and central nervous system 8th ICD: 191-192	95	123	126	86	94	103	$\chi^2 = 1.50$, $p = 0.22$
All causes	90	82	94	94	119	156	$\chi^2 = 128.6$, $p << 0.001$

Source: OPCS (1978).

Table A9

Standardised and proportional mortality ratios in Great Britain 1979-1980, 1982-1983 for men in age groups 20-64 years, 65-74 years by occupational class

Cause of death (9th ICD code)	Occupational class											
	I		II		IIIN		IIIM		IV		V	
	SMR	PMR1 PMR2	SMR	PMR1 PMR2	SMR	PMR1 PMR2	SMR	PMR1 PMR2	SMR	PMR1 PMR2	SMR	PMR1 PMR2
All causes	66	100 100	76	100 100	94	100 100	106	100 100	116	100 100	165	100 100
All neoplasms (140-209)	69	103 100	77	102 100	89	94 96	113	106 103	117	101 100	154	94 98
Diseases of circulatory system (390-458)	69	103 108	80	105 106	102	107 106	108	101 99	113	98 97	151	93 94
All leukaemias (204-208)	110	172 115	90	122 131	90	107 108	107	103 95	106	91 93	122	70 75
Acute lymphoid leukaemia (204.0)	137	224 66	97	135 140	116	134 106	100	100 107	98	84 84	91	49 67
Chronic lymphoid leukaemia (204.1)	98	144 126	82	107 135	69	73 106	112	104 93	111	96 83	162	102 87
Acute meloid leukaemia (205.0)	118	184 105	90	121 136	102	110 107	108	104 94	108	92 97	108	61 68
Chronic myeloid leukaemia (205.1)	106	168 144	93	127 116	110	119 118	93	91 97	108	92 98	155	85 78
Malignant neoplasms of the brain (191)	119	184 175	98	132 138	109	116 123	103	98 97	96	82 81	119	69 55
Malignant melanoma of the skin (172)	133	210 152	126	172 174	134	143 116	85	82 87	89	76 61	82	46 82

Source: OPCS (1986).

SMR and PMR1, 20-64; PMR2, 65-74

Table A10

Standardised and proportional mortality ratios in Great Britain 1979-1980, 1982-1983, for women in age groups 20-59 years, 60-74 years by own or husband's occupational class

Cause of death (9th ICD code)	Occupational class											
	I		II		IIIN		IIIM		IV		V	
	SMR	PMR1 PMR2	SMR	PMR1 PMR2	SMR	PMR1 PMR2	SMR	PMR1 PMR2	SMR	PMR1 PMR2	SMR	PMR1 PMR2
All causes	69	100 100	78	100 100	86	100 100	97	100 100	108	100 100	130	100 100
All neoplasms (140-209)	87	125 130	92	119 119	95	111 116	104	106 107	107	99 102	116	90 93
Diseases of circulatory system (390-458)	50	72 87	63	80 94	75	89 94	105	107 99	117	109 100	151	118 103
All leukaeukaemias (204-208)	91	133 150	106	138 121	97	109 114	115	123 114	100	93 110	114	86 91
Acute lymphoid leukaemia (204.0	51	76 172	99	127 94	122	133 103	128	143 176	87	80 154	147	108 37
Chronic lymphoid leukaemia (204.1)	68	98 122	130	167 129	70	84 95	139	141 102	66	61 132	62	48 83
Acute myeloid leukaemia (205.0)	106	156 120	101	130 119	100	113 120	118	126 116	100	93 104	112	84 95
Chronic myeloid leukaemia (205.1)	72	105 227	118	152 119	92	104 116	104	110 110	98	91 95	106	80 125
Malignant neoplasms of the brain (191)	123	179 234	104	135 160	91	104 129	109	114 108	82	76 96	96	73 72
Malignant melanoma of the skin (172)	118	172 208	107	138 143	111	125 125	106	112 102	83	77 107	102	76 85

Source: OPCS (1986).

SMR and PMR1, 20-59; PMR2, 60-74

Table A11

Mortality rates for malignant neoplasms of the brain, 1975 and 1985 (8th and 9th ICD code 191), for England and Wales: Rates per million in different age groups

Age group (years)	Male		Female	
	1975	1985	1975	1985
<1	6	15	17	22
1–4	13	9	9	8
5–14	10	15	8	8
15–24	6	9	9	5
25–34	17	21	13	12
35–44	34	37	24	28
45–54	77	85	53	62
55–64	136	171	81	102
65–74	115	186	61	126
75–84	44	121	22	70
≥85	26	77	6	28

Sources: OPCS (1977, 1987)

Table A12

Mortality rates for malignant melanoma of the skin, 1955, 1975 and 1985 (6th ICD code 190, 8th and 9th ICD codes 172), for England and Wales

Rates per million in different age group

Age group (years)	Male			Female		
	1955	1975	1985	1955	1975	1985
<1	0	0	0	0	0	0
1–4	0	0	0	0	0	0
5–14	1	0	0	0	0	0
15–24	3	1	2	2	2	1
25–34	6	7	5	7	7	9
35–44	9	14	20	8	16	13
45–54	7	21	32	11	26	29
55–64	17	23	37	16	23	31
65–74	21	31	50	24	22	51
75–84	31	38	68	30	50	68
≥85	38	52	105	30	69	100

Sources: Registrar General (1956); OPCS (1977, 1987).

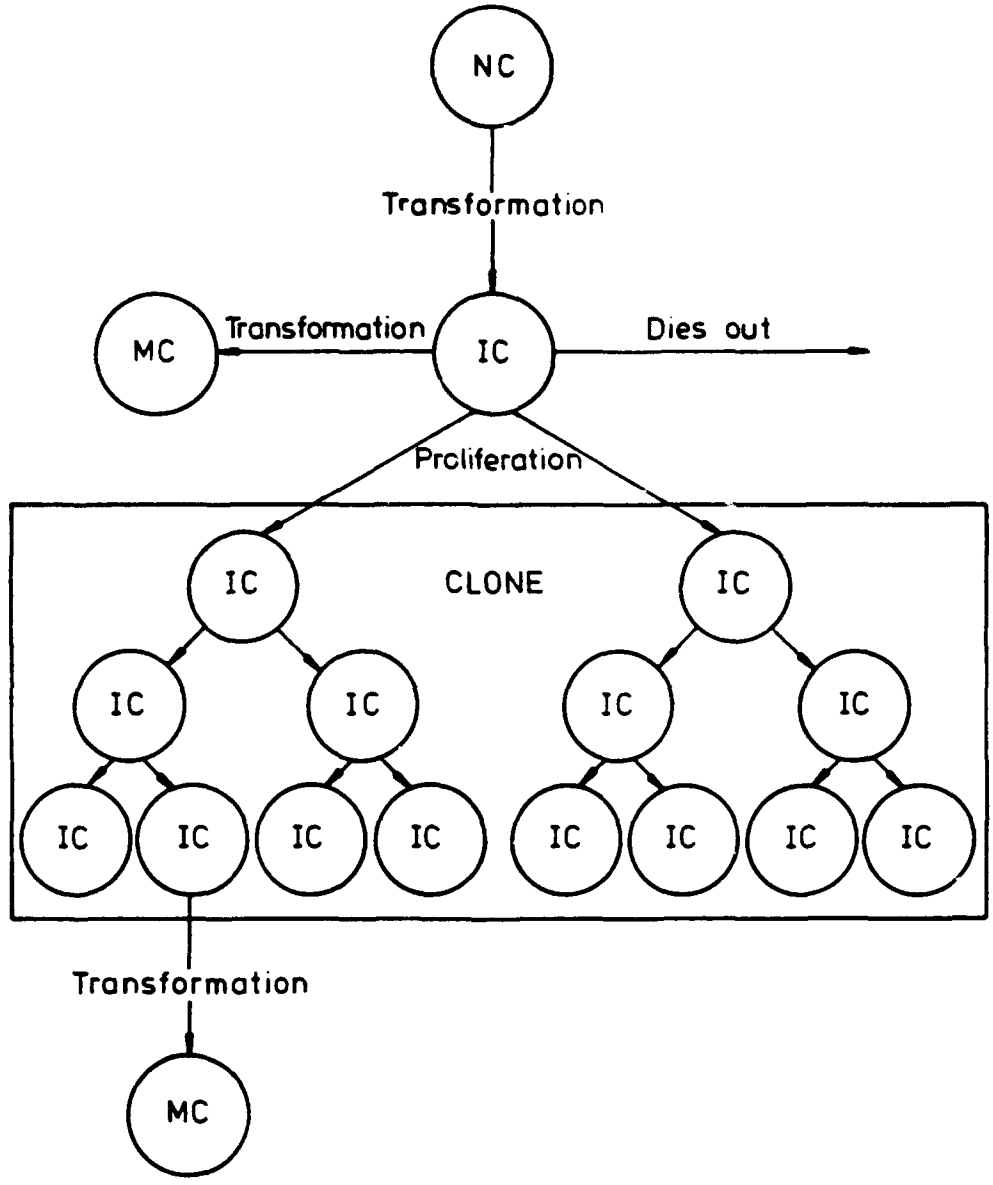

FIGURE A1 **Representation of the development of a malignant cell (MC) from a normal cell (NC) involving two stages of transformation and the possible proliferation of intermediate transformed cells (IC). These intermediate cells may also transform directly into malignant cells or may die out or be removed by the defence mechanisms of the body**

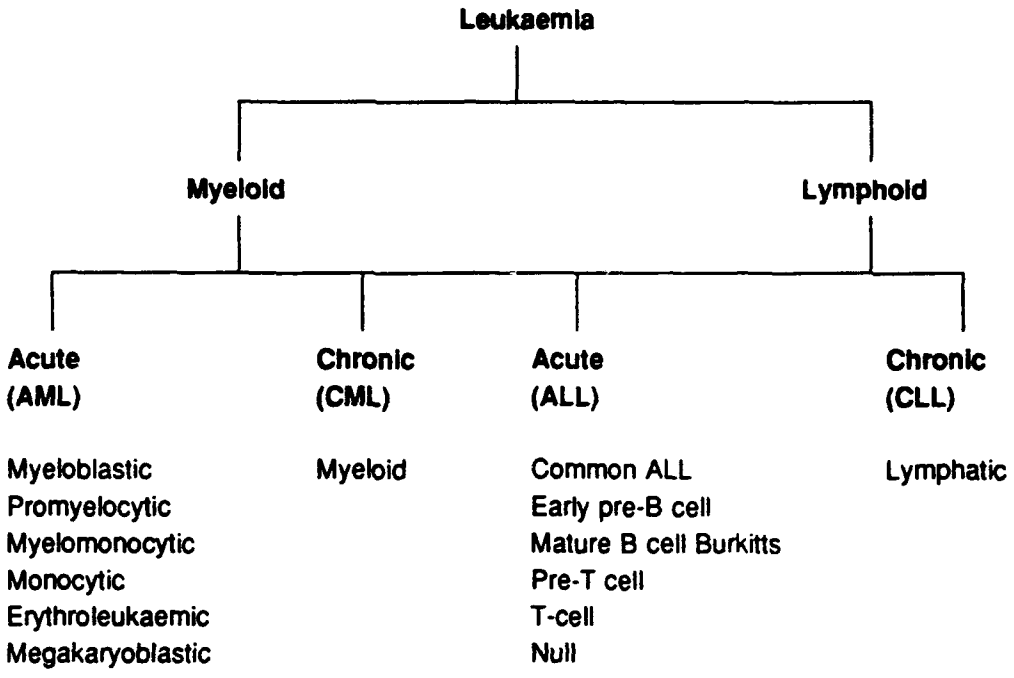

FIGURE A2 **Different types and subtypes of leukaemia**
(Although distinctions were originally made based on the morphology of the blood cells involved, more recently distinctions are based on the immunological characteristics of the cells. Terms such as lymphoid, lymphocytic and lymphatic tend to be used interchangeably. AML, CML, and ALL probably originate from a common stem cell.)

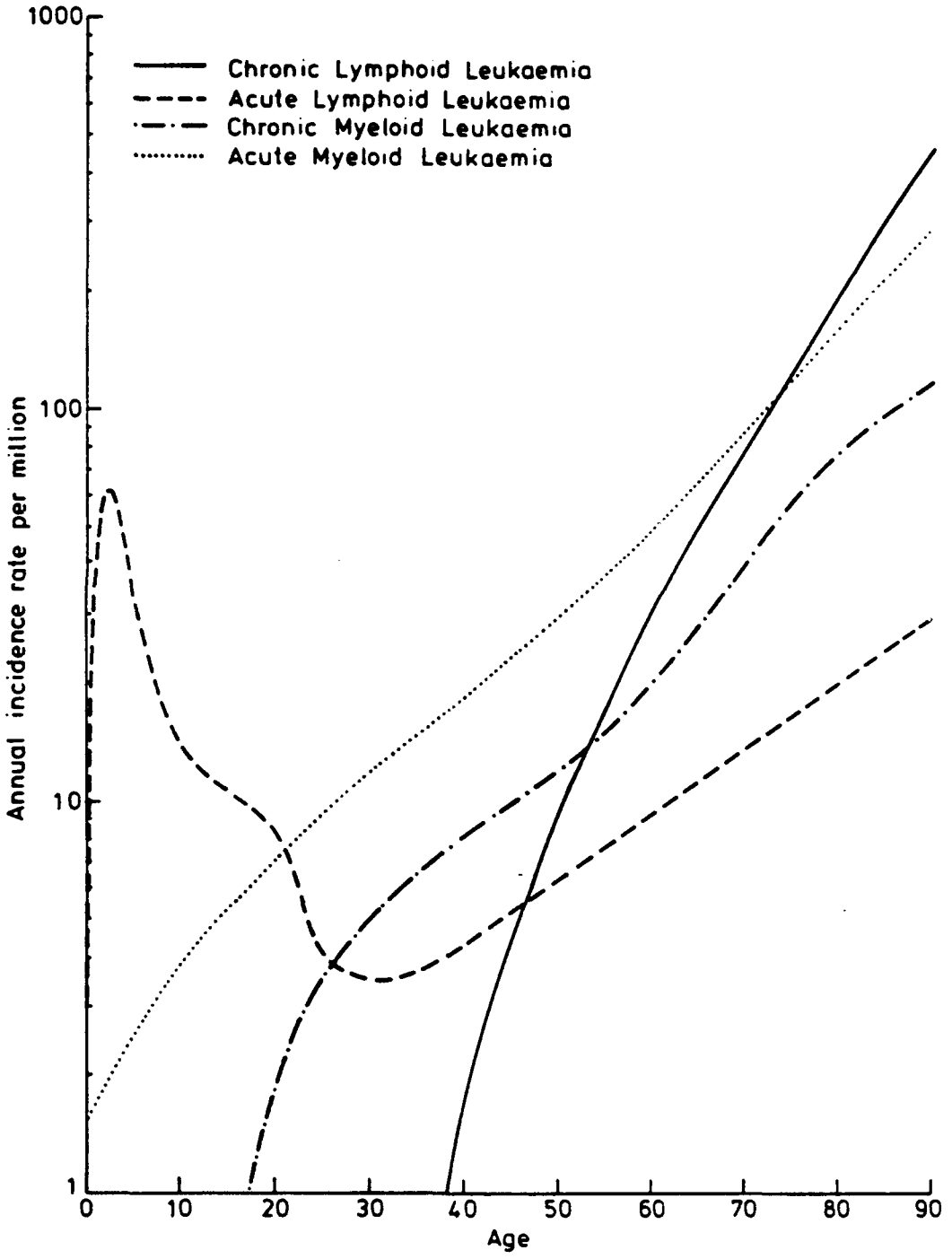

FIGURE A3 **Approximate relative and absolute incidence rates for the main forms of leukaemia in the UK based on data from Greaves, 1988, and OPCS, 1987**

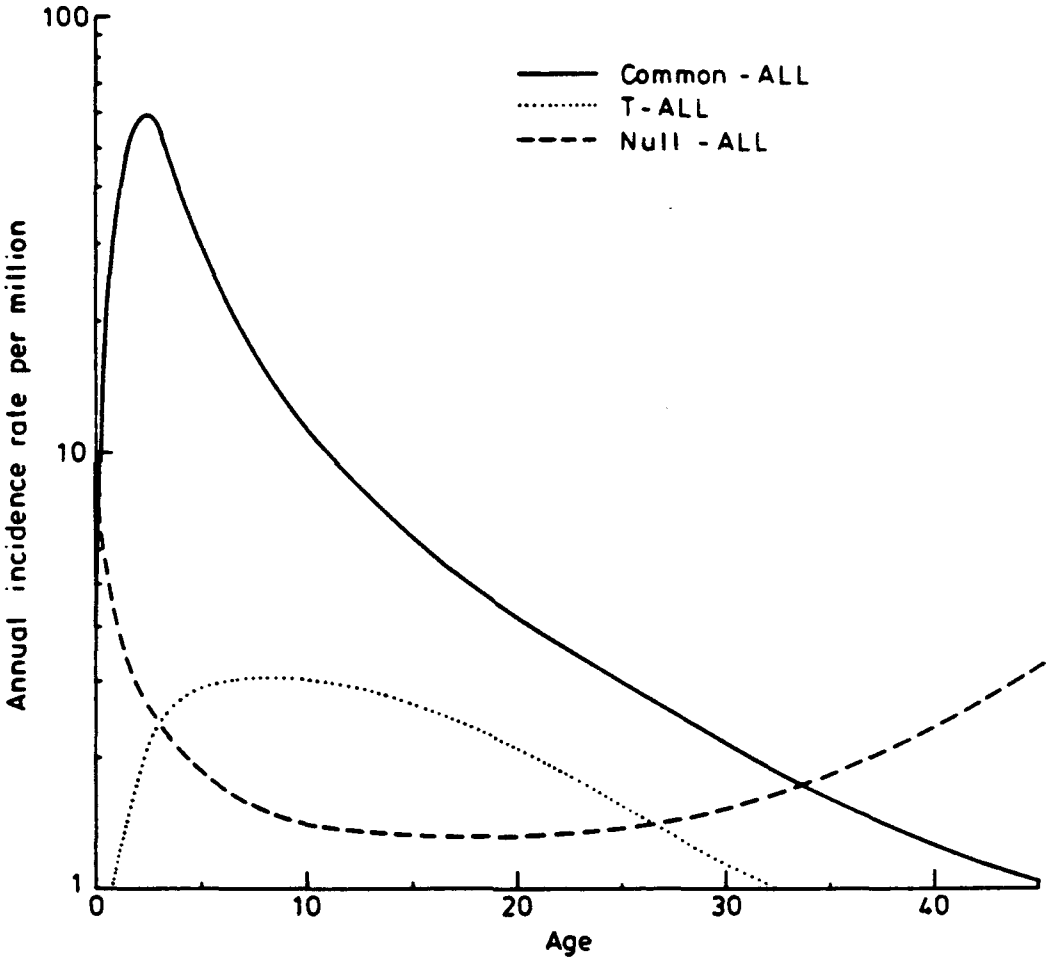

FIGURE A4 **Approximate relative and absolute rates of the subtypes of acute lymphoid leukaemia based on data from Greaves, 1988. Common-ALL originates from a B-lymphocyte precursor and T-ALL from a T-lymphocyte precursor**

APPENDIX B

Some statistical and epidemiological terms

1 Statistical Terms

At various places in this report mention is made of confidence intervals (CI) on numbers (usually ratios) and on the statistical significance of differences between numbers. Sometimes the latter is on the implied difference of a number from an expected value, usually 1.0. The statement that a number such as 2.1 has a 95% confidence interval of, for example 1.5–2.7 [ie 2.1 (95% CI 1.5-2.7)] means that there is a 95% chance (19 in 20 chance) that the range of 1.5 to 2.7 includes the true value of the estimated number and a 5% chance that it does not. The value of 2.1 is the best estimate. The statement that two numbers, such as 2.9 and 3.6, differ significantly at the 5% level implies that obtaining a difference as large as 3.6 - 2.9 = 0.7 by chance alone is 1 in 20. However, this is usually called a 'two-tailed' test. It does not refer to the fact that one specific number is larger than the other, only to the difference between them. The reader will usually be interested that one of the numbers is actually larger (or smaller) than the other; for example, in comparing a study group with a control group. The probability that the number of interest is actually larger (or smaller) than the other by the given amount is given by a 'one-tailed' test in which the probability will usually be about half of that given by the 'two-tailed' test. The possibility that a number for the study group of, says 3.6 is larger than that for the control group of, say, 2.9 by as much as 0.7 by chance alone is generally significant at the 2.5% level, ie 1 in 40, when the difference is significant at the 5% level.

In other places a mention is made of a test for trend. Usually this refers to any trend in the risk of a health effect to change with an increasing level of exposure. The test is based on a statistic known as chi-squared (χ^2) which yields a probability that the apparent trend is due solely to chance. A statement such as the chi-squared for trend is 1.32, p = 0.25, implies that there is a 25% chance that the apparent trend is due to chance alone. Estimates of trend are based on a standard technique known as logistic regression (Breslow and Day, 1980). This yields an estimate of the change in the ratio of the odds of a disease (which, for a rare disease, approximates to the relative risk) associated with a unit of exposure.

2 Epidemiological terms

In epidemiological studies two measures are commonly used in the analysis of mortality. These are the standardised mortality ratio, SMR, and the proportional mortality ratio, PMR.

SMR is the ratio multiplied by 100 of the number of persons in a certain group or occupation dying from a particular cause to the number that would be expected on the basis of age and sex specific mortality rates for the general population as a whole. It is a standardised measure of the absolute death rate in the group compared with the absolute death rate in the general population from the same cause.

PMR is a rather less satisfactory measure, and is the ratio multiplied by 100 of the fraction of all deaths in a certain group or occupation that are due to a particular cause to the corresponding fraction in the general population (with age and sex adjustment). It is a measure of the relative

death rate from a particular cause in the group compared to the relative death rate from the same cause in the general population.

Owing to the difficulties in obtaining data on the total number of people employed in any occupation, as distinct from those who die having been employed in that occupation, PMRs are usually more easily obtained than SMRs.

3 Reference

Breslow, N E, and Day, N E, 1980. Statistical methods in cancer research. I. The analysis of case-control studies. Lyon, IARC Scientific Publications No. 32.