1

Summary and Recommendations

Background

1.1 The widespread use of mobile phones is a recent phenomenon. Their use has escalated over the past decade and to many they are now an essential part of business, commerce and society. Over the Christmas 1999 period alone approximately 4 million phones were sold in the UK and at present (April 2000) there are about 25 million mobile phones in circulation. This is equivalent to nearly one phone for every two people (see paragraph 2.16)

1.2 The fact that so many people own mobile phones attests to their perceived importance to the general public. The advent of third generation systems will extend the use of most forms of communications technologies, including fax, e-mail and Internet access. **The use of mobile phones and related technologies will continue to increase for the foreseeable future.**

1.3 The extensive use of mobile phones has been accompanied by public debate about possible adverse effects on human health. The concerns relate to the emissions of radiofrequency (RF) radiation from the phones (the handsets) and from the base stations that receive and transmit the signals (paragraphs 3.3–3.7). For the general population, the levels of exposure arising from phones held near to the head or other parts of the body are substantially greater than whole-body exposures arising from base stations (paragraphs 4.28–4.36).

1.4 There are two direct ways by which health could be affected as a result of exposure to RF radiation. These are by thermal (heating) effects caused mainly by holding mobile phones close to the body, and as a result of possible non-thermal effects from both phones and base stations (paragraphs 5.5–5.26).

1.5 There can also be indirect effects. There is evidence that using a mobile phone whilst driving can increase the risk of accidents. Also some people's well-being may be adversely affected by the environmental impact of mobile phone base stations sited near their homes, schools or other buildings, as well as by their fear of perceived direct effects (paragraphs 5.264, 6.44 and 6.45).

Sources of Exposure

1.6 Mobile phones and base stations emit RF radiation. In both cases levels of exposure generally reduce with increasing distance from the source. For mobile phones, exposures will be principally to the side of the head for hand-held use, or to the parts of the body closest to the phone during hands-free use.

1.7 For base station emissions, exposures of the general population will be to the whole body but normally at levels of intensity many times less than those from handsets (paragraphs 4.28–4.36). Base stations communicate with mobile phones within a defined area or "cell". These can be of

three types: *macrocells, microcells and picocells* depending upon their size and the power output of the antenna (paragraph 4.9).

1.8 *Macrocells* provide the main structure for the base station network. The base stations for macrocells have power outputs of tens of watts and communicate with phones up to about 35 kilometres (22 miles) distant. There are at present about 20,000 macrocells covering the country (paragraph 4.9). We believe that this number will continue to increase. Measurements that have been made (see paragraphs 4.30–4.36) indicate that exposures of the general population from these sites are typically many hundreds, or thousands of times lower than existing exposure guidelines. There are concerns, nevertheless, about whether the emissions from all base stations are uniformly low, about whether the emissions could cause unknown health effects, and whether, with the increased use of mobile telecommunications, their output will have to rise.

1.9 *Microcells* are used to infill and improve the main network, especially where the volume of calls is high. They are sited in places such as airports, railway stations and shopping malls. Their number is rapidly increasing in line with the growth in demand for mobile phones. The microcell base stations emit less power than those for macrocells and their range is a few hundred metres. We understand that exposures above guidelines do not occur, provided the case surrounding the antenna is kept in place. However, as with some other items of electrical equipment – for example, lasers in CD equipment – there is a possibility of overexposure if the case is removed.

1.10 *Picocell* base stations have a lower power output than those of microcells (a few watts) and are generally sited inside buildings. It is likely that the number of picocells within buildings will substantially increase. Although we are satisfied that their emissions should not exceed the guidelines, the system of audits that we propose (paragraph 1.40) will provide an independent check on the output not only from picocell antennas but from all base station types.

1.11 As well as mobile phone base stations, there are a large number of other RF emitting sources in our environment, including antennas for radio, television and paging (paragraphs 4.20–4.22). Exposures of individuals to RF radiation from these sources will depend upon their proximity and may be above those from mobile phone base stations, although still well below guidelines.

Current Guidelines on Acceptable Levels of Exposure to Radiofrequency Radiation

1.12 Government has in place national guidelines (paragraphs 6.19–6.26, 6.32) established by the National Radiological Protection Board (NRPB) on the maximum levels of exposure to RF radiation emitted from mobile phones, base stations and other sources ("the NRPB guidelines"). These guidelines were established in 1993 when mobile phone technology was in its infancy. The guidelines were based on a comprehensive review of the scientific literature carried out by NRPB, a statutory body, which advises Government on radiological issues related to health.

1.13 In 1998 the International Commission on Non-Ionizing Radiation Protection (ICNIRP) published its own guidelines (paragraphs 6.27–6.31) covering exposure to RF radiation. These were based on essentially the same evidence as that used by NRPB, and for workers the limits on exposure are similar. However, under the ICNIRP guidelines, the maximum levels of exposure of the public are about five times less than those recommended for workers. The reason for this approach was the possibility that some members of the general public might be particularly sensitive to RF radiation. However, no detailed scientific evidence to justify this additional safety factor was provided.

1.14 The ICNIRP guidelines for the public have been incorporated in a European Council Recommendation (1999), which has been agreed in principle by all countries in the European Union (EU), including the UK. In Germany the ICNIRP guidelines have been incorporated into statute (paragraph 6.33).

1.15 Both the NRPB and ICNIRP guidelines are based on the need to avoid known adverse health effects. At the time these guidelines were drawn up, the only established adverse effects were those caused by the heating of tissues.

Main Conclusions on the Possible Effects of Mobile Phone Technology on Human Health

1.16 Despite public concern about the safety of mobile phones and base stations, rather little research specifically relevant to these emissions has been published in the peer-reviewed scientific literature. This presumably reflects the fact that it is only recently that mobile phones have been widely used by the public (paragraphs 2.1–2.12) and as yet there has been little opportunity for any health effects to become manifest. There is, however, some peer-reviewed literature from human and animal studies, and an extensive non-peer-reviewed information base, relating to potential health effects caused by exposure to RF radiation from mobile phone technology.

1.17 **The balance of evidence to date suggests that exposures to RF radiation below NRPB and ICNIRP guidelines do not cause adverse health effects to the general population (Chapter 5, paragraphs 6.33–6.42).**

1.18 **There is now scientific evidence, however, which suggests that there may be biological effects occurring at exposures below these guidelines** (paragraphs 5.176–5.194, 6.38). This does not necessarily mean that these effects lead to disease or injury, but it is potentially important information and we consider the implications below.

1.19 There are additional factors that need to be taken into account in assessing any possible health effects. Populations as a whole are not genetically homogeneous and people can vary in their susceptibility to environmental hazards. There are well-established examples in the literature of the genetic predisposition of some groups, which could influence sensitivity to disease. There could also be a dependence on age. **We conclude therefore that it is not possible at present to say that exposure to RF radiation, even at levels below national guidelines, is totally without potential adverse health effects, and that the gaps in knowledge are sufficient to justify a precautionary approach** (Chapter 5, paragraphs 6.35–6.42).

1.20 In the light of the above considerations **we recommend that a precautionary approach to the use of mobile phone technologies be adopted until much more detailed and scientifically robust information on any health effects becomes available** (Chapter 5, paragraphs 6.35–6.42).

1.21 We note that a precautionary approach, in itself, is not without cost (paragraph 6.16) but we consider it to be an essential approach at this early stage in our understanding of mobile phone technology and its potential to impact on biological systems and on human health.

1.22 In addition to these general considerations, there are concerns about the use of mobile phones in vehicles. Their use may offer significant advantages – for example, following accidents when they allow emergency assistance to be rapidly summoned. Nevertheless, the use of mobile phones whilst driving is a major issue of concern and experimental evidence demonstrates that it has a detrimental effect on drivers' responsiveness. Epidemiological evidence indicates that this

effect translates into a substantially increased risk of an accident. Perhaps surprisingly, current evidence suggests that the negative effects of phone use while driving are similar whether the phone is hand-held or hands-free (paragraph 5.213). **Overall we conclude that the detrimental effects of hands-free operation are sufficiently large that drivers should be dissuaded from using either hand-held or hands-free phones whilst on the move** (paragraphs 5.201–5.214, 5.262–5.263 and 6.93–6.95).

1.23 We consider below ways in which a precautionary approach to the use of mobile phone technology might be adopted.

A Precautionary Approach and Related Issues

1.24 **We recommend that national and local government, industry and the consumer should all become actively involved in addressing concerns about possible health effects of mobile phones (paragraph 6.40).**

1.25 Our recommendations focus on five areas:

- advice to Government,
- advice to industry,
- research requirements,
- the need for better public information and consumer choice,
- the role of NRPB.

Advice to Government

1.26 We recognise that the mobile phone industry impacts on people and business around the world and that the UK is a global leader in telecommunications technology. There are benefits that the development of mobile telecommunications can bring, provided there is no adverse impact on health. It is against this general backcloth that we make our recommendations.

Standards

1.27 **We recommend that, as a precautionary approach, the ICNIRP guidelines for public exposure be adopted for use in the UK rather than the NRPB guidelines.** This would bring the UK into line with other countries in the European Union and accord with the Recommendations of the House of Commons Select Committee on Science and Technology Report on Mobile Phones and Health (1999) (paragraphs 6.19–6.42).

1.28 **We are not convinced of the need to incorporate the ICNIRP guidelines in statutes.** We believe that they are liable to change as more scientific information on possible health effects becomes available (paragraph 6.36).

1.29 It would be sensible, in line with the precautionary approach, to set in place a long-term follow-up of workers who are occupationally exposed to RF radiation at relatively high levels. **We recommend that a register of occupationally exposed workers be established and that cancer risks and mortality be examined to determine whether there are any harmful effects. If any**

adverse effects of exposure to RF radiation are identified then the Health and Safety Executive should establish a system of health surveillance (paragraph 5.240).

Planning issues

1.30 The siting of base stations in residential areas can cause considerable concern and distress. At all our open meetings and in written evidence we heard concerns about the location of base stations in sensitive sites. These include schools, residential areas and hospitals. This concern relates, in part, to the fact that base stations up to 15 m (48 ft) in height can be installed in residential areas without the need for a full planning application. We consider this to be unacceptable.

1.31 We are concerned at the indirect adverse impact which current planning procedures are having on those who have been, or are, subjected to the often insensitive siting of base stations. Adverse impacts on the local environment may adversely impact on the public's well-being as much as any direct health effects.

1.32 We recognise that exposures of people in the vicinity of base stations are expected to be well within guidelines yet there is no independent audit to ensure that this is the case (paragraphs 4.30–4.35).

1.33 **We conclude that the balance of evidence indicates that there is no general risk to the health of people living near to base stations on the basis that exposures are expected to be small fractions of guidelines. However, there can be indirect adverse effects on their well-being in some cases** (paragraphs 5.264, 6.44 and 6.45).

1.34 We perceive a lack of clear protocols to be followed in the public interest prior to base stations being built and operated and note that there is significant variability in the extent to which mobile phone operators consult the public on the siting of base stations. We have heard little specific criticism of most of the network operators, apart from Orange. The Department of the Environment, Transport and the Regions and the National Assembly for Wales (DETR, 1998) produced a *Code of Best Practice: Telecommunications prior approval procedures* as applied to mast/tower development. We understand that consideration is being given to extending this to include health concerns (paragraphs 6.104–6.109). We support this development.

1.35 Overall we consider that public concerns about the siting of base stations demand changes in the planning process. Thus:

1.36 **We recommend that for all base stations, including those with masts under 15 m, permitted development rights for their erection be revoked and that the siting of all new base stations should be subject to the normal planning process** (paragraphs 6.43–6.46 and 6.55–6.62).

1.37 **We recommend that, at national Government level, a template of protocols be developed, in concert with industry and consumers, which can be used to inform the planning process and which must be assiduously and openly followed before permission is given for the siting of a new base station** (paragraphs 6.58–6.62). We consider the protocol should cover the following issues.

- All telecommunications network operators must notify the local authority of the proposed installation of base stations. This should cover installations for macrocells, microcells and picocells.
- The local authority should maintain an up-to-date list of all such notifications, which should be readily available for public consultation.

- The operator should provide to the local authority a statement for each site indicating its location, the height of the antenna, the frequency and modulation characteristics, and details of power output.
- Any change to an existing base station which increases its size, or the overall power radiated, should be subject to the normal planning process as if it were a new development.

1.38 **We recommend that a robust planning template be set in place within 12 months of the publication of this report. It should incorporate a requirement for public involvement, an input by health authorities/health boards and a clear and open system of documentation which can be readily inspected by the general public** (paragraphs 6.55–6.62).

1.39 **We recommend that a national database be set up by Government giving details of all base stations and their emissions. This should include the characteristics of the base stations as described in paragraphs 6.47 and 6.48 and should be an essential part of the licence application for the site.**

1.40 **We recommend that an independent random, ongoing, audit of all base stations be carried out to ensure that exposure guidelines are not exceeded outside the marked exclusion zone and that the base stations comply with their agreed specifications. If base station emissions are found to exceed guideline levels, or if there is significant departure from the stated characteristics, then the base station should be decommissioned until compliance is demonstrated** (paragraphs 6.53 and 6.54).

1.41 **We recommend that particular attention should be paid initially to the auditing of base stations near to schools and other sensitive sites** (paragraphs 6.54 and 6.63–6.68).

1.42 **We recommend, in relation to macrocell base stations sited within school grounds, that the beam of greatest intensity** (paragraphs 4.32–4.35 and 6.63–6.68) **should not fall on any part of the school grounds or buildings without agreement from the school and parents. Similar considerations should apply to macrocell base stations sited near to school grounds.**

1.43 **We recommend that in making decisions about the siting of base stations, planning authorities should have the power to ensure that the RF fields to which the public will be exposed will be kept to the lowest practical levels that will be commensurate with the telecommunications system operating effectively** (paragraphs 6.55–6.62).

Exclusion zones

1.44 **We recommend the establishment of clearly defined physical exclusion zones around base station antennas, which delineate areas within which exposure guidelines may be exceeded** (paragraphs 6.49–6.52). **The incorporation of exclusion zones should be part of the template of planning protocols that we advocate**.

1.45 Each exclusion zone should be defined by a physical barrier and a readily identifiable nationally agreed sign with a logo. This should inform the public and workers that inside the exclusion zone there might be RF emissions which exceed national guidelines. **We recommend that the design of the logo should be taken forward by the British Standards Institute and implemented within 12 months** (paragraphs 6.49–6.52).

1.46 **We recommend that warning signs should be incorporated into microcell and picocell transmitters to indicate they should not be opened when in use** (paragraph 6.52)**.**

Use of mobile phones near hospitals

1.47 We are concerned about the indiscriminate use of mobile phones in hospitals and other sites where the RF radiation could possibly interfere with sensitive equipment. **We understand that health authorities/health boards issue guidance on the use of mobile phones. They should ensure that all hospitals comply. This guidance should include the placing of visible warning signs at entrances to buildings to indicate that mobile phones should be switched off** (paragraphs 4.6, 6.91 and 6.92).

Devolution in Scotland, Wales and Northern Ireland

1.48 Where recommendations (paragraphs 1.30–1.46) impact on the devolved responsibilities of the Scottish Parliament, the Welsh National Assembly and the Northern Ireland Assembly then they should be considered by their appropriate authorities or bodies. We have noted with interest the recent report on planning procedures for telecommunications developments produced by the Transport and the Environment Committee of the Scottish Parliament (2000) (paragraphs 6.112–6.117).

Advice to Industry

1.49 We believe that in the global economy of the 21st Century a competitive edge will be generated by developing innovative, technologically advanced and safe products, which can lead the field and win competitive advantage.

1.50 We understand from the Mobile Manufacturers Forum that all mobile phones presently marketed in the UK comply with both NRPB and ICNIRP guidelines. A crucial issue in relation to the exposure of people using mobile phones is the specific energy absorption rate (SAR). This determines the amount of energy absorbed in the body of the user. In most circumstances of use this will be the head. The SAR depends upon the power output of the phone and its design (paragraph 4.37). We understand that an internationally agreed standard testing procedure that will allow the SAR from mobile phones to be compared is being developed and will be finalised this year (2000). Such a procedure should benefit consumers and should also be welcomed by industry. We note that in the case of cars, standard testing procedures for fuel consumption have been developed to inform consumer choice, and have resulted in the development of more efficient engines. We see no reason why, in the case of mobile phones, standard testing procedures should not lead to a progressive reduction in exposures from the equipment.

1.51 **We recommend that an international standard for the assessment of SAR values from mobile phones should be adopted for use in the UK once it has been demonstrated to be scientifically sound** (paragraphs 6.74–6.79).

1.52 **We recommend that information on the SAR values for mobile phones must be readily accessible to consumers** (paragraph 6.77)**:**

- **at the point of sale with information on the box**,
- **on leaflets available in stores giving comparative information on different phones and with explanatory information**,
- **as a menu option on the screen of the phone and as a label on the phone,**
- **on a national web site, which lists the SAR values of different phone types**.

1.53 **If there are currently unrecognised adverse health effects from the use of mobile phones, children may be more vulnerable because of their developing nervous system, the greater absorption of energy in the tissues of the head** (paragraph 4.37)**, and a longer lifetime of exposure. In line with our precautionary approach, at this time, we believe that the widespread use of mobile phones by children for non-essential calls should be discouraged. We also recommend that the mobile phone industry should refrain from promoting the use of mobile phones by children** (paragraphs 6.89 and 6.90).

1.54 We have examined the value of mast sharing and roaming agreements. These can offer advantages in terms of providing a better service in rural areas and limiting environmental intrusion. **We recommend that operators actively pursue a policy of mast sharing and roaming where practicable** (paragraphs 6.69 and 6.70).

Health Related Research

1.55 The mobile phone industry has supported a substantial and ongoing programme of research internationally. The recent upsurge in the use of mobile phone technology in the UK has not been matched, in general, by the output of good quality relevant research supported by the public sector. Too many studies have been carried out at exposure levels and frequencies not directly related to the use of mobile phones or base stations.

1.56 In relation to present research findings, the following three areas deserve particular comment.

- First, the balance of the evidence available does not suggest that RF radiation from mobile phones or base stations causes cancer or other disease. However, there is now evidence that effects on biological functions, including those of the brain, may be induced by RF radiation at levels comparable to those associated with the use of mobile phones. There is, as yet, no evidence that these biological effects constitute a health hazard but at present only limited data are available. This is one reason why we recommend a precautionary approach.
- Second, concerns have been expressed that the pulsed nature of the signals from mobile phones and masts may have an impact on brain function. This is an intriguing possibility, which deserves further research, particularly if pulsed signals continue to be used in the third generation of phones and related technologies. Research should concentrate on signal modulations representative of present and future phone technology (paragraphs 5.4, 5.12–5.26 and 5.270).
- Third, we commend the World Health Organization (WHO) for encouraging the use of standard experimental protocols under realistic exposure conditions relevant to mobile phone technology (paragraph 5.284). This should allow experiments from different laboratories to be readily compared.

1.57 On the basis of the current state of knowledge **we recommend that priority be given to a number of areas of research related particularly to signals from handsets** (paragraph 5.270). These should include the following:

- effects on brain function,
- consequences of exposures to pulsed signals,
- improvements in dosimetry,
- the possible impact on health of sub-cellular and cellular changes induced by RF radiation,

- psychological and sociological studies related to the use of mobile phones,
- epidemiological and human volunteer studies (paragraphs 5.249–5.264), including the study of children, and individuals who might be more susceptible to RF radiation (paragraphs 4.37, 6.29 and 6.30).

1.58 **We recommend that a substantial research programme should operate under the aegis of a demonstrably independent panel**. The aim should be to develop a programme of research related to health aspects of mobile phones and associated technologies. This should complement work sponsored by the EU and in other countries. In developing a research agenda the peer-reviewed scientific literature, non-peer reviewed papers and anecdotal evidence should be taken into account (paragraphs 5.270–5.272).

1.59 **We further recommend that this programme be financed by the mobile phone companies and the public sector (industry departments, health departments and the research councils), possibly on a 50 : 50 basis.** The contribution from industry could be made on a voluntary basis or by a continuing levy reviewable every five years (paragraph 5.272).

1.60 It will be essential for further research in this area to be kept under review. **We recommend that the issue of possible health effects of mobile phone technology should be the subject of a further review in three years time, or earlier if circumstances demand it** (paragraph 5.273).

Public Information and Consumer Choice

1.61 We are concerned at the variability and the limited extent of the information made available to consumers on mobile phone products. **We recommend that Government circulates a leaflet to every household in the UK providing clearly understandable information on mobile phone technology and on related health aspects, including the use of mobile phones while driving** (paragraphs 5.201–5.208). **This leaflet should additionally be available at the point of sale. The leaflet should be developed in concert with industry, which has already produced some good leaflets** (paragraphs 3.48 and 3.49).

1.62 **We recommend that an Ombudsman be appointed to provide a focus for decisions on the siting of base stations when agreement cannot be reached locally, and on other relevant issues** (paragraphs 3.50 and 3.51).

1.63 There are various devices that seek to reduce exposure to RF radiation from mobile phones. These include shields and devices that attach to phones. We remain to be convinced of their effectiveness in reducing personal exposure in normal conditions of use of mobile phones.

1.64 Hands-free extensions, which allow the phone to be held away from the body, have the potential for reducing exposure, but some recent tests have cast doubt on their general level of effectiveness. For users wishing to reduce their exposure, we advocate the use of hands-free kits of proven effectiveness. A satisfactory design may involve the use of chokes or filters in the connecting lead. A standard testing procedure should be established.

1.65 The regulatory position on the use of shielding devices and hands-free kits, which may affect the phone's performance, is unclear. In addition, information available for the public on the use of such devices is limited to that provided by the suppliers of the devices and the mobile phone industry. **We recommend that Government sets in place a national system which enables independent testing of shielding devices and hands-free kits to be carried out, and which enables clear information to be given about the effectiveness of such devices. A kite mark or**

equivalent should be introduced to demonstrate conformity with the testing standard (paragraphs 6.86–6.88).

National Radiological Protection Board (NRPB)

1.66 We believe that NRPB is a valuable UK asset which should be built upon, and that it carries out scientific work which is well-regarded nationally and internationally.

1.67 Whilst there is no criticism of its science**, we recommend that NRPB gives greater priority to the execution of a more open approach to issues of public concern such as mobile phone technology and that it is proactive rather than reactive in its approach** (paragraph 3.44).

1.68 **We recommend that public concerns about risk be addressed by NRPB in a more sensitive and informative manner** (paragraph 3.45).

1.69 **We recommend that NRPB makes more use of specialist time-limited *ad-hoc* committees of experts and lay representatives to bring forward broadly based, well-considered advice** (paragraph 3.42).

1.70 **We recommend that in a rapidly emerging field such as mobile phone technology where there is little peer-reviewed evidence on which to base advice, the totality of the information available, including non-peer-reviewed data and anecdotal evidence, be taken into account when advice is proffered** (paragraph 3.46).

1.71 We note the paucity of resources available at NRPB for work on non-ionising radiation, including work on mobile phones, and related research on life sciences. **We recommend that work on non-ionising radiation and related life sciences work be strengthened at NRPB** (paragraph 3.47).

References

DETR (1998). Department of the Environment, Transport and the Regions and The National Assembly for Wales Code of Best Practice. Telecommunications prior approval procedures as applied to mast/tower development.

EC (1999). Council Recommendation of 12 July 1999 on the limitation of exposure of the general public to electromagnetic fields (0 Hz to 300 GHz). *Official Journal of the European Community* L1999, 59 (1999/519/EC).

Science and Technology Committee (1999). Third Report. Scientific advisory system: mobile phones and health. Volume 1, Report and Proceedings of the Committee.

Scottish Parliament Transport and the Environment Committee (2000). Third Report. Report on inquiry into the proposals to introduce new planning procedures for telecommunications developments.

2

Introduction

2.1 The telecommunications industry is experiencing rapid growth on a global scale. This is a direct consequence of technological development and has in turn facilitated the application of new technologies, and a consequent increase in economic activity. Within this sector, one of the greatest growth areas of recent years has been the development of mobile or wireless telecommunications.

2.2 The first land mobile services were introduced into the UK in the 1940s, but the significant expansion of services offered to the general public, including the introduction of mobile phones, began in the mid-1980s and rapidly attracted a small but significant number of subscribers. Developments in the early 1990s, such as the introduction of digital networks and the entry of additional service providers into the market, fuelled further increases in the numbers of subscribers.

2.3 It is now predicted that within a few years around half the population of the UK will be routinely using mobile telecommunications (see Figure 2.1) and that this will become the dominant technology for telephony and other applications such as Internet access. This wide use of a relatively new technology raises the question of whether there are any implications for human health.

2.4 There are conflicting reports relating to possible adverse health effects and these have understandably led to some concern. The Minister for Public Health recognised the importance of this issue and, following consultation with the Ministers at the Department of Trade and Industry, decided to seek the advice of an independent group as to the safety of mobile telecommunications technology, and asked the Chairman of the National Radiological Protection Board (NRPB) to establish an Independent Expert Group on Mobile Phones (IEGMP).

2.5 Following widespread consultation with interested parties, the Expert Group was set up under the chairmanship of Sir William Stewart FRS, FRSE. Membership of the Expert Group (Appendix A) represented a wide spectrum of expertise with leading figures from physics, radio engineering, biology, medicine, and epidemiology, in addition to lay members. The remit of the Group was

> *"To consider present concerns about the possible health effects from the use of mobile phones, base stations and transmitters, to conduct a rigorous assessment of existing research and to give advice based on the present state of knowledge. To make recommendations on further work that should be carried out to improve the basis for sound advice."*

2.6 The Expert Group held its first full meeting in September 1999 and determined from the outset that it must consult widely. To this end, advertisements were placed in national newspapers and scientific journals inviting individuals or organisations to submit evidence for consideration. Public meetings were arranged in Belfast, Cardiff, Edinburgh, Liverpool and London.

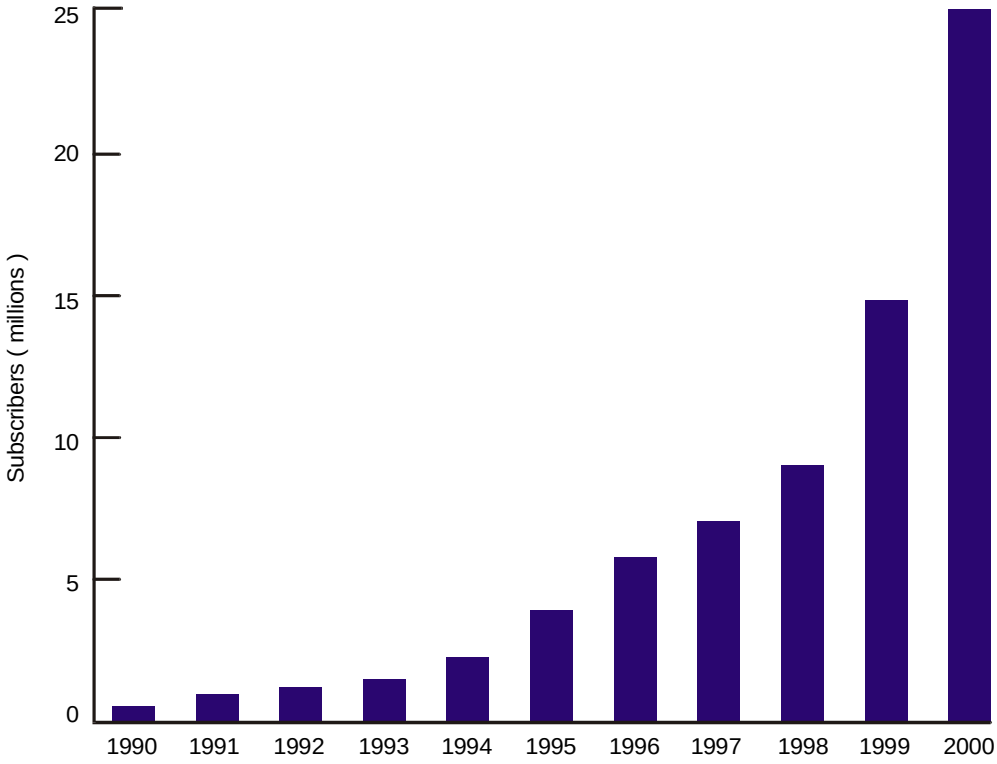

Figure 2.1 Growth in mobile phone subscribers in the UK between 1990 and 2000 (based on data from Federation of the Electronics Industry, FEI)

2.7 Those who submitted written evidence to the Expert Group are listed in Appendix B. A number of individuals and organisations accepted invitations to present evidence to closed meetings of the Group and these are indicated in Appendix C.

2.8 This report describes the work of the Expert Group. It presents the wide picture of mobile telecommunications as they impact on the general public, and recognises the contribution of mobile telecommunications to the quality of life and to the UK economy. It considers the underlying technology and the characteristics of the RF fields generated by present and near future (3–5 years) handsets and base stations, with particular reference to the magnitude of the fields. It provides an appraisal of the experimental and theoretical work that has been carried out which has a bearing on human health, and makes a number of recommendations to Government.

Background to the Introduction of Mobile Telecommunications

2.9 The UK telecommunications system was initially developed and operated as part of the General Post Office (GPO). In 1981, this situation changed with the passing of the British Telecommunications Act, which effectively separated the telecommunications and postal businesses of the GPO, and led to the creation of British Telecom (BT). The next stage in telecommunications development was the creation of a competitive marketplace governed by a new regulatory body, the Office of Telecommunications (OFTEL), which was established in 1984. These changes paved the way for the introduction of cellular telecommunications in a competitive environment.

2.10 Initially two companies were granted operating licences, Telecom Securicor Cellular Radio Limited (Cellnet) and a subsidiary of Racal Electronics plc (Vodafone). In January 1985, both these companies launched national networks based on analogue technology (see paragraph 4.10). However, in the late 1980s there was a move to develop standards for a second generation of mobile telecommunications throughout Europe in order to provide a seamless service for subscribers. This was achieved with the development and deployment of a new operating standard called the Global System for Mobile Telecommunications (GSM), which employs digital technology (see paragraph 4.11) and is now the operating system for 340 networks in 137 countries (Figure 2.2). Although this system is now used worldwide, the European geographical area is still the dominant user, with more subscribers than any other region. It has, however, been widely accepted in other areas such as the Asia Pacific region.

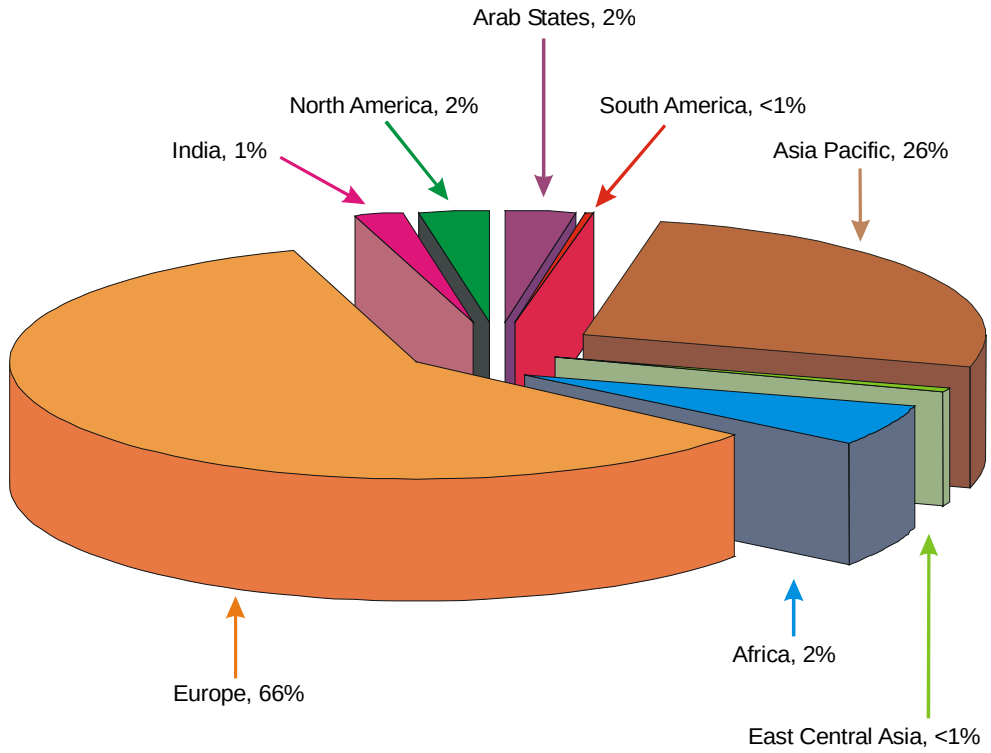

Figure 2.2 Distribution of GSM subscribers by geographical location (based on data from the GSM Association)

2.11 In the UK, the new GSM networks became operational in July 1992 (Vodafone), September 1993 (One 2 One), December 1993 (Cellnet), and April 1994 (Orange) the companies involved being referred to in this report as the network operators. The original analogue networks are still operational, but the Government has indicated that the analogue system should be removed from service by 2005.

2.12 On a worldwide scale, there has been a rapid growth in both the numbers of countries with operational networks and the number of mobile phone operators (Figure 2.3). There are a further 39 networks under construction for the GSM system alone.

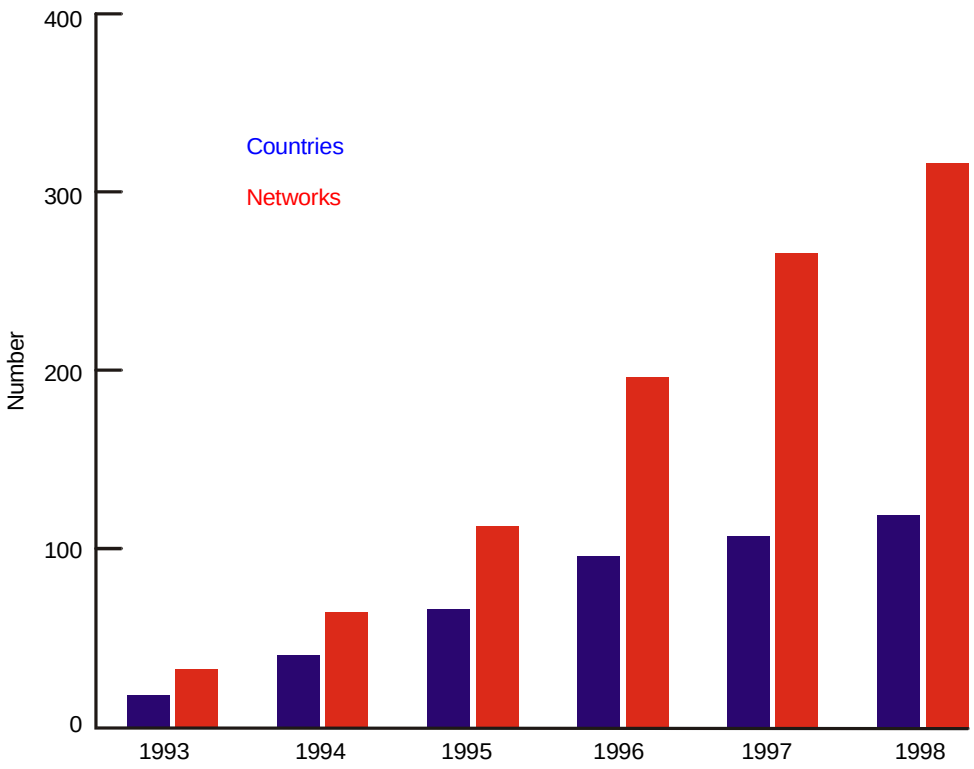

Figure 2.3 Growth of GSM networks throughout the world (based on data from the GSM Association)

Mobile Phone Networks and Communication

2.13 Individual mobile phones operate by communicating with fixed installations called base stations. These have a limited range (see paragraph 4.9) and mobile phone operators have to establish national base station networks to achieve wide coverage. It takes many years to establish a network that will provide both complete coverage and adequate capacity across the country and, even today, none of the UK networks provides complete coverage. However, since operators invest a great deal of money to purchase licences and establish networks and other infrastructure, they need to offer potential subscribers an effective communication system as quickly as possible. Moreover, operators were required, as a condition of their operating licences, to provide a minimum level of coverage within a given time frame. They established operational networks designed to allow most subscribers to access a base station most of the time. The initial phase of construction of such a network involves the installation of base stations in urban areas with high population densities, and along major transport routes such as motorways. These basic networks are then extended to provide coverage in more rural areas and increased capacity in urban areas. By developing networks in this way, operators can offer a functional system to the majority of the population. The more rural areas of the UK, particularly in the west of the country, still have rather poor coverage.

2.14 Base stations can be categorised into macrocells, microcells and picocells (see paragraph 4.9) depending on their size and power output. There are approximately 20,000 macrocells in the UK at present and, in general, all the major operators can now offer coverage to over 97% of the population. The number of macrocells is continuing to rise as operators seek to complete their geographical coverage and improve capacity. Since each base station can only handle a limited number of connections at any one time, operators need to install more base station units in densely

populated areas to cope with increasing demand. It seems likely that these will mainly be microcells and picocells. The overall number of base stations is likely to double within the next few years.

Present and Future Use of Mobile Phones

2.15 Initial market penetration by mobile phones was modest, with less than 1% of the UK population subscribing by the end of the 1980s. However, the advent of the more advanced GSM technology, in conjunction with greater competition in the market place, led to continuing growth in the number of subscribers throughout the 1990s (Figure 2.1).

2.16 At present there are approximately 25 million subscribers in the UK, which is equivalent to a market penetration of around 40%. Within the next five years it is expected that this will have increased to 75% market penetration or 45 million subscribers. At present it is estimated that around 45% of subscribers have a pre-paid mobile phone. Although it might be expected that many of these phones would not be used on a routine basis, the operators believe that around 90% of them are in regular use.

2.17 Within the next three years the "Third Generation" of mobile phones will be launched. This will employ a new operating standard called the Universal Mobile Telecommunication System (UMTS, see paragraph 4.15) and will enable operators to offer a full range of multimedia services. The introduction of these new services will require access to additional RF spectrum,

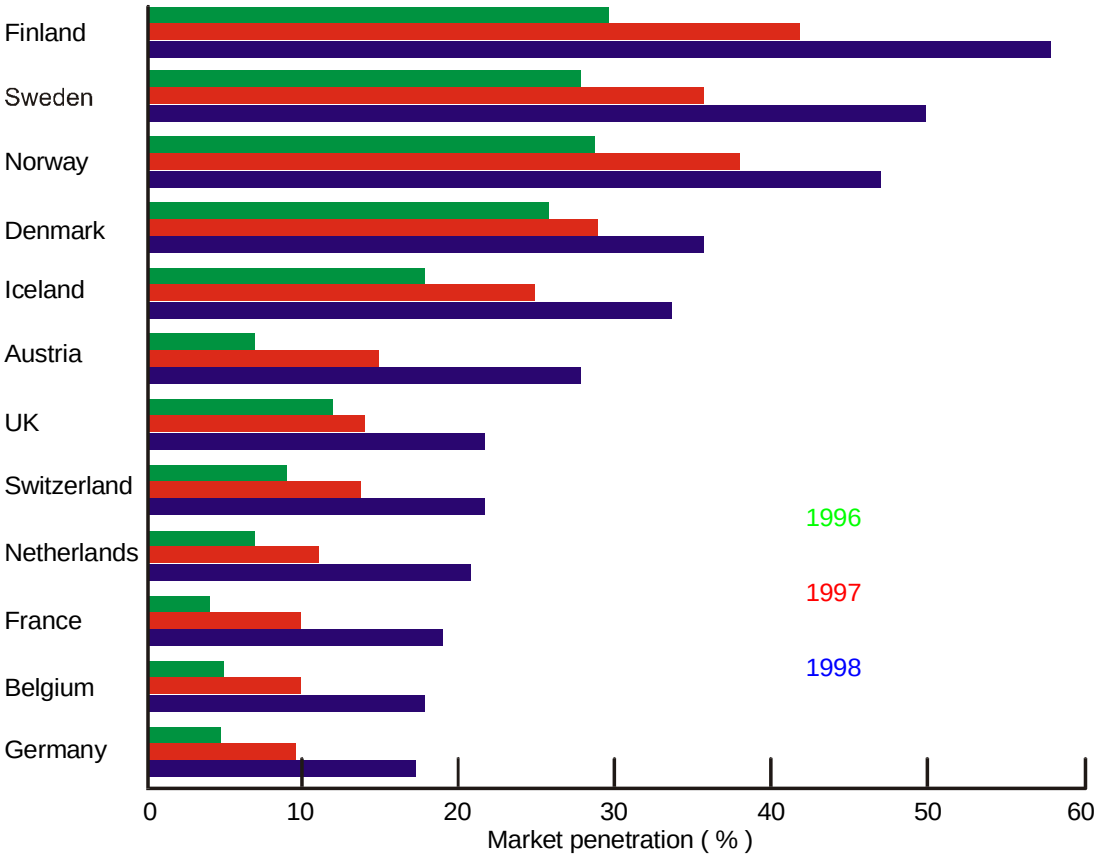

Figure 2.4 Increase in market penetration between 1996 and 1998 in Western European countries (based on data from MobilTeleBranschen)

and the UK Government has recently auctioned licences for the use of new spectrum. Five licences are to be issued.

2.18 The growth in the mobile phone market that has been observed in the UK reflects similar trends in Europe and elsewhere in the world. In Europe the greatest market penetration has occurred in the Scandinavian countries and in Finland is approaching 60%. However, all Western European countries have experienced a rapid growth in mobile phone use in recent years (Figure 2.4).

2.19 It is expected that the recent trends in the use of mobile phone technology will continue for the foreseeable future, with the number of GSM subscribers worldwide predicted to increase by a factor of three or more over the next five years (Figure 2.5).

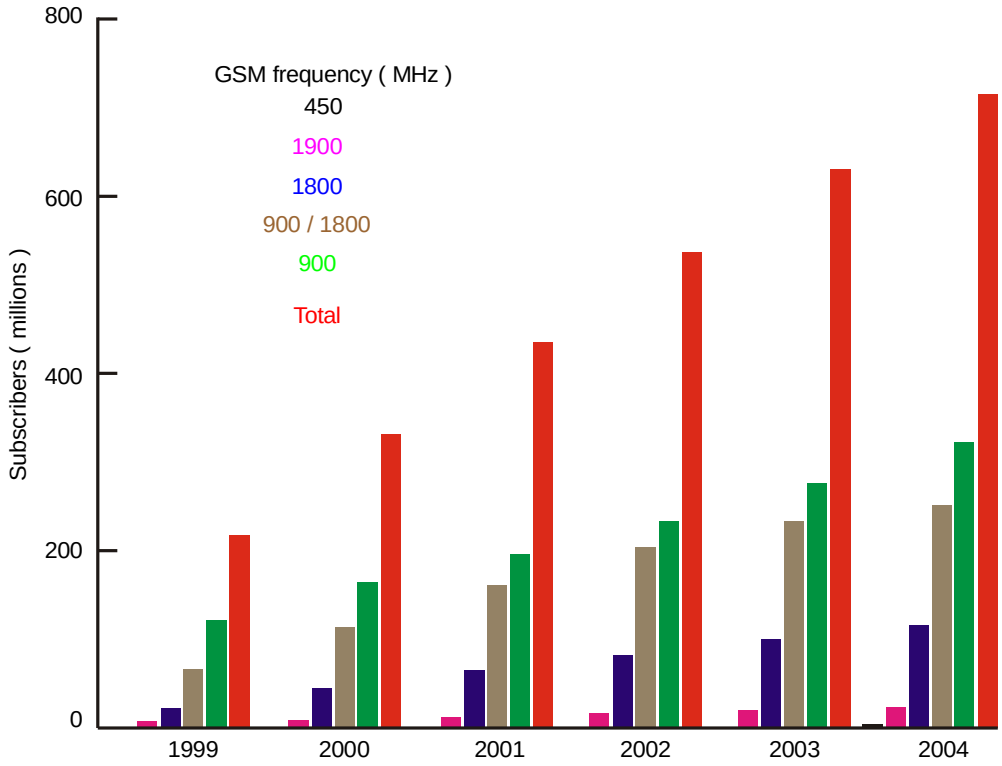

Figure 2.5 Predicted growth in the number of GSM subscribers worldwide. The different GSM frequencies (see paragraph 4.11) are used in different systems around the world.

Benefits of Mobile Telecommunications Technology

2.20 An active mobile telecommunications sector brings a number of economic benefits to the UK in terms of employment and tax revenue, which will be discussed in paragraph 2.22. There are also, however, a number of other advantages to be derived from application of this technology. Mobile telecommunications play an increasingly important role in general commercial activity and thereby make an indirect contribution to the national economy. This is difficult to quantify, but is likely to be significant.

2.21 It is already apparent that mobile telecommunications also offer benefits in emergency situations. For example, the use of a mobile phone may reduce the time taken to notify the emergency services of road traffic accidents and other dangerous situations including crimes. An assessment

of this aspect in Australia has recently been given by Chapman and Schofield (1998a,b). There have also been several accounts of individuals using mobile phones to alert rescue services following mountaineering or skiing accidents. Mobile phone availability may also be helpful during much rarer large-scale emergencies. For example, it is believed that many lives were saved following the earthquake in Kobe, Japan, because those trapped under rubble were able to use their mobile phones to alert rescue teams.

Economic Significance of the Mobile Phone Industry to the UK Economy

2.22 Information supplied by the Department of Trade and Industry indicates that the mobile phone industry is a major contributor to economic activity in the UK. Network operators had an estimated combined turnover of some £5.8 billion in the financial year 1998/99 (Figure 2.6).

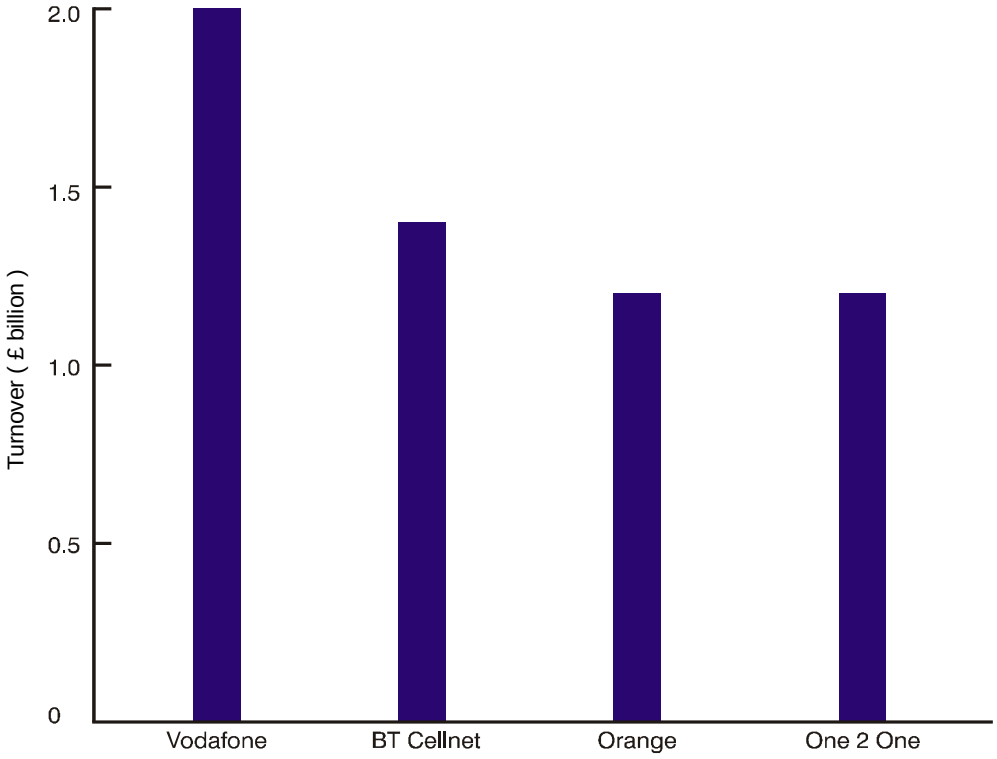

Figure 2.6 Annual turnover of UK network operators (data provided by the Department of Trade and Industry)

2.23 Vodafone has the largest turnover and, since its merger with the US company Air Touch, when it had a market capitalisation of £77 billion, and subsequent takeover of the German company Mannesmann, is now a major multinational. The other three operators (here we treat Orange as an independent company) are smaller, but nationally major companies. In late 1999, BT Cellnet and One 2 One were valued at around £8.4 billion, with Orange slightly more at that time. The Vodafone mergers emphasise the international nature of the mobile phone industry. All four UK operators have expanded into overseas markets in recent years; Vodafone and Orange have taken the lead in this respect. Between them, these two operators now have stakes in over 14 countries, including Australia, France, Germany, the Netherlands, Sweden, South Africa and Switzerland. The manufacture of mobile phone equipment is also an international industry and is dominated by

a few large multinationals. Although none of these is based in the UK, three of them, Nokia, Motorola and Ericsson, all have a significant presence through both manufacturing and research and development (R&D) facilities. Nokia and Ericsson bought out UK companies in the early 1990s and both have since expanded their operations. Other manufacturing companies that have invested in the UK include Lucent, NEC, Panasonic and Samsung. This is a rapidly changing sector and the above figures are indicative only.

2.24 The manufacturing base generates secondary manufacturing by companies such as Hewlett Packard and Racal, both of which make test equipment. In addition, there is some manufacturing of components by companies such as Filtronic Ltd. The latest available information on manufacturing turnover values the telecommunications sector at £3.5 billion in 1997, but it is growing rapidly. Mobile telecommunications represent a significant and increasing element of this sector.

2.25 The UK provides significant input into mobile telecommunications R&D through universities and their spin-off companies. A consortium of UK universities has formed a Virtual Centre of Excellence in this area to provide a focus for this work and ensure effective collaboration with industry. Funding for this Virtual Centre from industry and the Engineering and Physical Sciences Research Council totalled £3 million for the last three years and the budget for the next three is £4.5 million with industry providing 70%.

2.26 The mobile sector provides significant employment opportunities in the UK. It is difficult to obtain accurate data because the sector is developing so rapidly. However, taken together, the operators, manufacturers, and sales outlets probably employ about 100,000 people in the UK (industry estimate). This number seems likely to increase when mobile phones become more closely linked to the provision of Internet services.

3

Public Perceptions and Concerns

3.1 When the Minister for Public Health announced the formation of the Independent Expert Group on Mobile Phones (IEGMP) she stated:

> *"In recent years research interest in the effects of mobile phones has increased. To date there has been no consistent evidence suggesting risk to health, but there is continuing public concern about the possibility. It would be wrong to ignore that concern."*

3.2 As part of our inquiry we sought information on the specific aspects of mobile phone technology that worry the public, and on the information that is available to them about this issue from different sources. We examined the role of politicians, the media, specific interest groups, the telecommunications industry and the National Radiological Protection Board (NRPB).

The Public

3.3 In order to obtain input from the public we placed advertisements in the press, solicited information from various groups and held open public meetings in Belfast, Cardiff, Edinburgh, Liverpool and London. The Secretariat of the Group issued press releases in August and September 1999 explaining the role of the Group and inviting oral and written submissions. It also gave notice of dates and times of the public meetings and all information was featured on the Expert Group web site (www.iegmp.org.uk).

3.4 It is unlikely that those who attended the meetings or submitted evidence can be considered representative of the public at large, and it should be recognised that many members of the public are satisfied with all or most aspects of mobile phone technology. However, the relative frequency with which specific concerns were raised at the meetings provides an indication of those aspects of the technology that the concerned public finds most worrying.

3.5 People who attended the meetings described various symptoms which they attributed to base stations. Those mentioned most commonly were headaches, sleep disturbance, depression, stress and tiredness.

3.6 There was concern about the siting of base stations on or near schools, hospitals and residential areas; current planning processes; and particularly the fact that base stations less than 15 m do not have to follow the normal planning process in full. With regard to the siting of base stations, Orange, in particular, was criticised for inadequate public consultation. Operational practices such as erecting base stations in the middle of the night were an understandable cause for complaint. Other concerns related to possible reductions in the value of property caused by the proximity of base stations to residences, the negative visual impact of base stations, and possible health risks to farm animals.

3.7 The health problems most commonly attributed to the use of mobile phone handsets were impairment of short-term memory, headaches, brain tumours, other cancers, sleep disturbance, depression and tiredness.

Politicians

3.8 The Chairman of the Expert Group wrote to all Members of both Chambers of the Westminster Parliament. Out of 659 Members of the House of Commons, 11 acknowledged receipt of the communication and 6 responded in writing highlighting the concerns of their constituents, which related mainly to base stations.

3.9 No Member of the House of Lords replied to the letter.

3.10 The Chairman also wrote to Members of the Scottish Parliament (129), Members of the Welsh Assembly (60) and Members of the Northern Ireland Assembly (108). Responses were received from four Members of the Scottish Parliament and five Members of the Welsh Assembly. Two Members of the Northern Ireland Assembly acknowledged receipt of the letter, and a further three gave more detailed responses. One member of the Expert Group met four MPs to discuss the issues that concerned their constituents.

3.11 Regulatory powers in relation to telecommunications are reserved in the UK Parliament at Westminster. Therefore, although planning is seen as a local authority responsibility and, as a result of devolution, the Scottish Parliament now has a greater say in planning, contentious issues such as the General Permitted Development Order rights, which fall under the Telecommunications Act 1984, have not been devolved.

3.12 Devolved political power provides an opportunity for a less centralised approach to planning issues, and this may allow more areas of local and regional concern to be addressed in legislation and guidance. Already the devolved Assemblies and Parliaments have played a key role in raising public awareness regarding the potential health impact of mobile phone technology. In particular, we note with interest the work of the Scottish Parliament's Transport and Environment Committee, whose report on planning issues (see paragraphs 6.112–6.114) was published in March 2000 (Scottish Parliament, 2000).

3.13 In the past year UK Ministers received almost 600 letters from members of the public on health issues relating to mobile phone technology, and particularly base stations. In the House of Commons, the Health Ministers answered 85 letters on the same topic from MPs writing on behalf of constituents. There have also been parliamentary questions about possible health effects of mobile phones and base stations.

3.14 Of the 80 letters sent directly to the Department of Health by members of the public, 50 concerned base stations and 30 handsets. The Department of the Environment, Transport and the Regions received 350 letters relating to planning and environmental aspects of mobile phone technology, while the Department for Education and Employment received 157 letters about the location of base stations in or around schools.

3.15 Parliamentary debates on mobile phone technology have also centred on health and planning issues. An Early Day Motion on the subject of mobile phone masts calls for a more coherent planning policy and for the precautionary principle to be employed in relation to possible health risks. This is the most popular Early Day Motion currently tabled in the House.

3.16 In September 1999, the House of Commons Science and Technology Committee produced a report on "The Scientific Advisory System: Mobile Phones and Health". The Recommendations of this report were noted with interest by the Expert Group.

3.17 From the totality of evidence we received, it is clear that the role of politicians in this area extends further than legislation, and that their inputs have helped to inform the public.

The Media

3.18 The Expert Group reviewed 641 press cuttings published in the UK between January 1999 and February 2000 and 76 TV and radio programmes broadcast over the same period. In general, the same issues were covered as those cited above (paragraphs 3.3–3.7). Seventy-nine per cent of the media reports alleged adverse health effects from mobile phones and base stations, whereas nine per cent concluded that there was too little rigorous scientific evidence to arrive at a conclusion, or reported no adverse effect. Overall, the safety of mobile phone handsets achieved more coverage than the safety of base stations, although local newspapers tended to report more on issues relating to base stations. Other aspects of safety that were covered included concerns that driving while using a hand-held mobile phone was dangerous.

3.19 The adverse health effects most often linked with mobile phones in media reports were brain tumours, other cancers, headaches, and brain damage. However, possible positive effects were also reported (see also the Expert Group web site).

3.20 Most media reporting did not refer to specific scientific studies or discuss the biological mechanisms by which RF radiation from mobile phones might cause adverse effects.

3.21 Specific precautionary measures were suggested in the media including hands-free operation of mobile phones, use of shields, limiting the duration of mobile phone usage and choosing a mobile phone that emits a lower level of RF radiation.

3.22 The main concerns reported in the media about base stations were uncertainties about the distance at which they were "safe", and about their proximity to schools, homes, hospitals and residential accommodation for the elderly. Adverse aesthetic impacts were also noted. The health effects most often alleged were sleep disorders, fatigue, anxiety, stress, epileptic fits, burning sensations and shaking.

3.23 The media particularly targeted operators for ignoring medical evidence of adverse biological effects and for being irresponsible when choosing where to site base stations. Financial gain was sometimes alleged by the media to be more important to operators than the safety of people, particularly children. Operators were encouraged to remove base stations from populated areas. The reported responses from operators noted that the exposures from base stations were within NRPB guidelines.

Specific Interest Groups

3.24 About 175 specific interest groups declare an interest in the issue of mobile phone technology. Amongst these, three who have widely publicised their concerns are: Powerwatch, Friends of the Earth Scotland and NIFATT (Northern Ireland Families Against Telecommunications Transmitter Towers). Most of the other specific interest groups have been involved at a more local level.

3.25 Much helpful evidence was submitted to us by these interest groups – in writing, orally and through contributions to public meetings. It focussed mainly on possible adverse biological effects of mobile phone technology and the need for a precautionary approach.

3.26 There was criticism of the NRPB exposure guidelines for failing to take adequate account of uncertainties in current scientific knowledge, and the groups called for NRPB to be more open and proactive in communicating with scientists and the public.

The Telecommunications Industry

Mobile phone manufacturers

3.27 The Group received helpful inputs from mobile phone manufacturers in the UK and elsewhere and from the Federation of the Electronics Industry (FEI), an umbrella organisation.

3.28 The information provided by the FEI includes the following:

- general safety advice is provided with mobile phone handsets, usually in the accompanying operating guide or manual,
- the advice notes that all phones comply with the International Commission on Non-Ionizing Radiation Protection (ICNIRP) guidelines (ICNIRP 1998a, see paragraphs 6.27–6.31),
- all manufacturers emphasise their commitment to safety and report that there is no scientific evidence to show adverse health effects,
- in general, advice is provided that the mobile phone should be switched off when the user is in an aircraft, in a hospital, in the vicinity of medical equipment, or in hazardous environments such as petrol stations,
- there is support for the use of hands-free sets when in control of a car, and some manufacturers recommend that a hand-held phone should not be used when driving,
- all but one manufacturer warn that mobile phones may interfere with the functioning of pacemakers,
- most manufacturers advise that the antenna of the phone should not be in contact with the user when the phone is operating.

3.29 Information relating to the operation of mobile phones can be obtained on request from all the manufacturers. The Carphone Warehouse, an independent retailer, provides an important example of good practice in the way it makes information readily available at mobile phone outlets.

Network operators

3.30 The lines of communication between network operators and the general public do not appear to us to be as clear as those between mobile phone manufacturers and consumers. Each operator has opportunities for contact with its customers when they are billed or pay for calls in advance. However, communication with those who might be affected by the placement of base stations is less straightforward.

3.31 Overall, we conclude that more could be done in this area. In particular, there is substantial scope for the sharing of best practice in approaches to communication with the general public regarding the placement of base stations, as the public continues to distrust the operators in the area of public health.

National Radiological Protection Board

3.32 NRPB was given a range of responsibilities and powers under the Radiological Protection Act 1970. Amongst these was the responsibility:

> *"To provide information and advice to persons, including Government Departments, with responsibilities in the United Kingdom in relation to the protection of the community as a whole or of particular sections of the community from radiation hazards."*

3.33 NRPB as a recipient of taxpayers' monies should play a key role in informing the public. We were told by the Director of NRPB that it receives about 40,000 enquiries per year on a range of questions, and data provided by NRPB indicate that, during the period between August and October 1999, about a quarter of the enquiries related to mobile phones and base stations.

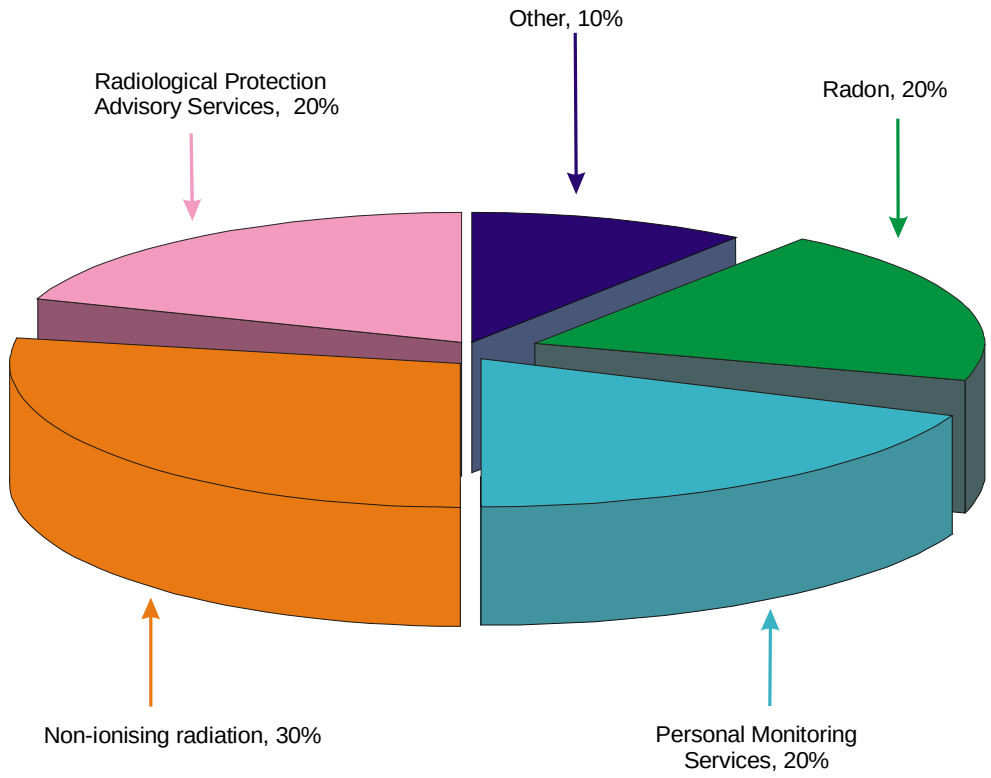

Figure 3.1 Enquiries received by NRPB on radiological protection matters by subject area (data are broad estimates for 1999 based on around 40,000 enquiries)

3.34 The NRPB's *modus operandi* is broadly as follows: members of the public who contact NRPB are normally sent a standard information pack about the topic on which they are seeking information, but where a more detailed response is required, the enquiry is directed to a member of the scientific staff with specialist knowledge in the relevant area. Members of the public who ask about planning issues are advised to contact their local authority, and for medical queries NRPB advises reference to a general practitioner.

3.35 Despite the fact that the public is currently most concerned about mobile phones and base stations, NRPB resources are largely targeted in other areas. Only about 10% of the total NRPB budget is allocated to non-ionising radiation, which includes work related to mobile phone

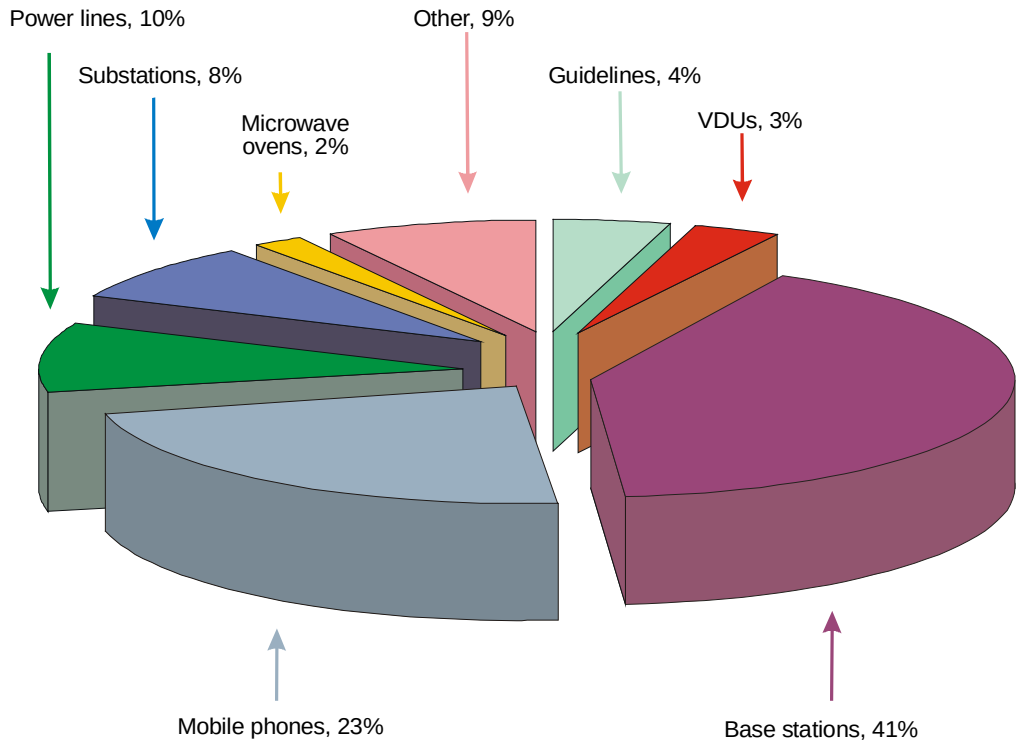

Figure 3.2 Enquiries received by NRPB on non-ionising radiation issues broken down into specific subject areas

technology, and the number of staff who have specialist knowledge in the mobile phone/base station area is in single figures.

3.36 We believe that effective communication on mobile phone technology should be a priority for NRPB and that the organisation should be more proactive in this area. The Director informed us that a new communications strategy is now being set in place by NRPB.

3.37 We note with interest that the Department of Trade and Industry has commissioned NRPB to produce an explanatory video on mobile phone technology suitable for the lay person.

3.38 As well as producing explanatory leaflets, NRPB publishes material on its web site (www.nrpb.org.uk) which was set up in February 1997. This includes pages giving NRPB views on the alleged health effects of mobile phones and base stations. The quality of the material produced is not universally user-friendly for the public and fails to address all of their current concerns. Nevertheless the site receives about 50,000 “hits” per month.

3.39 In summary, while a small minority of highly proactive individuals and groups has led the public debate on possible health hazards from mobile phone technology, NRPB has been mainly reactive. The Expert Group accepts that “scare stories” will always be the most popular focus of many sections of the media, but feels that NRPB should do more to inform the debate.

3.40 We note that many of the people who attended the public meetings that we organised expressed dissatisfaction with the advice given by NRPB. Their view was that operators should respond to suggestions of adverse health effects even if the evidence was not conclusive, whereas NRPB advises that current exposure guidelines should not be altered until there is “convincing and consistent” evidence of adverse biological effects at lower levels.

3.41 Currently, NRPB does not provide any advice beyond a strict definition of what is and what is not a "safe" level of non-ionising radiation. It has not made recommendations to Government on any need for precautionary policies to reflect uncertainties in current scientific knowledge. It has, however, recommended additional research that may help to resolve some of these uncertainties.

3.42 In general, we believe that NRPB has not adopted a sufficiently proactive approach to emerging public concerns about mobile phone technology, and has tended only to be reactive. That the impetus to set up this Expert Group came from Government rather than NRPB is in itself remarkable. NRPB does have its own independent Advisory Group on Non-ionising Radiation although it has not specifically addressed the issue of mobile phones. **We recommend that NRPB makes more use of specialist time-limited *ad-hoc* committees of experts and lay representatives to bring forward broadly based, well-considered advice.**

3.43 We further believe that additional resources should be made available within NRPB to address media and communications.

3.44 Whilst there is no criticism of its science**, we recommend that NRPB gives greater priority to the execution of a more open approach to issues of public concern such as mobile phone technology and that it is more proactive rather than reactive in its approach.**

3.45 **We recommend that public concerns about risk be addressed by NRPB in a more sensitive and informative manner.**

3.46 **We recommend that in a rapidly emerging field such as mobile phone technology, where there is little peer-reviewed evidence on which to base advice, the totality of the information available, including non-peer-reviewed data and anecdotal evidence, be taken into account when advice is proffered.**

3.47 We note the paucity of resources available at NRPB for work on non-ionising radiation, including work on mobile phones and related research on life sciences. **We recommend that work on non-ionising radiation and related life sciences work be strengthened at NRPB.**

Public Information

3.48 We are concerned that too much of the information that is currently presented to the public regarding the health aspects of mobile phone technology is misleading. We therefore believe that it would help if Government provided clear advice on this topic in the form of a leaflet circulated to all households.

3.49 **We recommend that Government circulates a leaflet to every household in the UK providing clearly understandable information on mobile phone technology and on health aspects, including the use of mobile phones while driving (see paragraphs 5.201–5.214). This leaflet should additionally be available at the point of sale. The leaflet should be developed in concert with industry, which has already produced some good leaflets.**

3.50 It would also help if an Ombudsman were appointed to whom issues relating to the siting of base stations and other aspects of the regulation of mobile phone technology could be referred where there was public dissatisfaction.

3.51 **We recommend that an Ombudsman be appointed to provide a focus for decisions on the siting of base stations when agreement cannot be reached locally, and on other relevant issues.**

General Conclusions

3.52 The continuing rapid growth in the use of conventional mobile phones, as shown in Figure 2.1, indicates that most people do not consider the possibility of adverse health effects to be a major issue.

3.53 Given the much lower exposures to radiation from base stations than from handsets (paragraph 4.32), the greater public concern from the people who gave evidence to the Expert Group about the former is paradoxical. It presumably arises because individuals can choose whether or not to use a mobile phone, whereas they have little control over their exposures from base stations. Furthermore, people derive a personal benefit from the use of a phone, but gain nothing directly from the presence of a base station close to their home or place of work. If anything they may suffer a loss of amenity and perhaps a reduction in the value of their property.

4

Radiofrequency Fields from Mobile Phone Technology

Radiofrequency Radiation Usage

4.1 Mobile phones and their base stations transmit and receive signals using electromagnetic waves (also referred to as electromagnetic radiation or fields, or radio waves). Electromagnetic radiation is emitted by many natural and man-made sources and plays a very important part in our lives. We are warmed by the radiation from the Sun or from an electric fire and we see using that part of the electromagnetic spectrum that our eyes can detect. All electromagnetic radiation consists of oscillating electric and magnetic fields and the frequency, f or ν (*nu*), which is the number of times per second at which the waves oscillate, determines their properties and the use that can be made of them. Frequencies are measured in hertz or Hz, where 1 Hz is one oscillation per second, 1 kHz or kilohertz is a thousand Hz, 1 MHz or megahertz is a million Hz, and 1 GHz or gigahertz is a thousand million Hz or 10^9 Hz. Frequencies between about 30 kHz and 300 GHz are widely used for telecommunication, including broadcast radio and television, and comprise the radiofrequency (RF) band.

4.2 In the UK, AM radio uses frequencies between about 180 kHz and 1.6 MHz, FM radio ranges from 88 to 108 MHz, and TV ranges from 470 to 854 MHz. Cellular mobile phone services operate within the frequency ranges 872–960 MHz and 1710–1875 MHz. Waves at higher frequencies but within the RF region, up to around 60 GHz, are referred to as *microwaves* and have a wide variety of uses. These include radar, telecommunications links, satellite communications, weather observations and medical diathermy; intense sources of 2.45 GHz microwaves confined within ovens are used for cooking. At even higher frequencies, radiation takes the form of infrared, then visible, ultraviolet, X-rays and eventually the γ-rays (*gamma* rays) emitted by radioactive material. Electromagnetic radiation is also characterised by its wavelength λ (*lambda*), which equals the velocity or speed of the wave (the speed of light) divided by its frequency.

Radiocommunication

4.3 An RF wave used for radiocommunication is referred to as a carrier wave. The information it carries – speech, computer data, etc – has to be added to the carrier wave in some way, a process known as modulation. The information can be transmitted in either analogue or digital form. For example, the electrical signal from a microphone produced by speech or music is an analogue signal at frequencies up to about 15 kHz. So the signal varies significantly with time on a scale of a few microseconds or μs (1 μs is a millionth of a second). At a particular time it might have any value within quite a large range. So if this signal is sent by analogue transmission, the size or amplitude of the RF carrier wave at any instant is made proportional to the size of the electrical modulating signal at that instant (this is called amplitude modulation and other forms of

modulation can also be used) (Figure 4.1). The carrier wave varies very much faster than the signal so that the modulation produces a relatively slow oscillation in the amplitude of the carrier wave. Information can also be transmitted in digital form. In this case only a small number of symbols are used. Printed language is an example of digital information since it only uses the symbols of the alphabet. Morse code is another and only uses two symbols, dots and dashes, so is called a binary system. Analogue signals are described by a number, which in general is not an integer (whole number), and the first step in digitising it is to round this to the nearest integer. For example, if the strength of an electrical signal from a microphone at a particular instant is 12793.56 microvolts or μV (1 μV is a millionth of a volt) the number 12793.56 is rounded to 12794. This can then be expressed in binary form in which it is represented by a series of zeros and ones, and these can be transmitted digitally to a receiver that converts them back to a signal of strength 12794 μV. Digital transmission, usually binary, offers many technical advantages over analogue transmission systems. It is, for example, less susceptible to distortion by interference and electrical noise, and it is replacing or has replaced analogue transmission in radio, TV, mobile phones, etc.

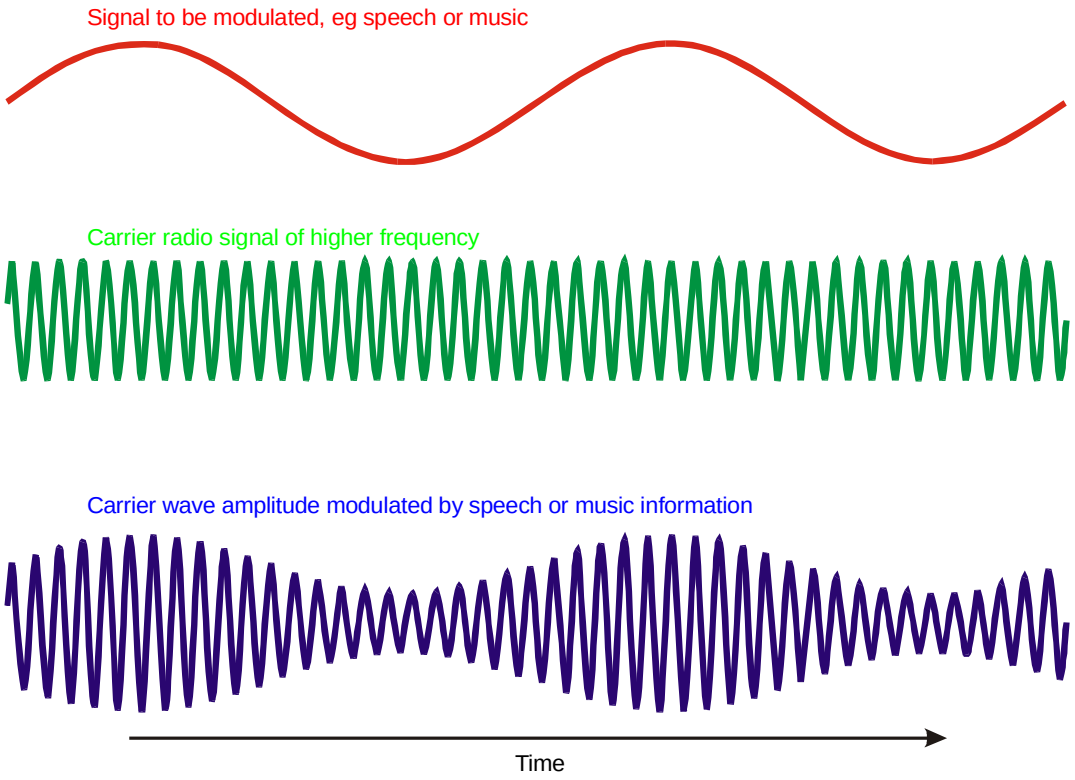

Figure 4.1 Amplitude modulation of a carrier wave

Electromagnetic Compatibility

4.4 The ability of electrical and electronic systems to operate in an electromagnetic environment without adverse effects is known as electromagnetic compatibility (EMC). The reality is that all electrical systems can be disturbed if subjected to sufficiently powerful emissions. For this reason, EMC is achieved by limiting or controlling electromagnetic emissions in addition to ensuring that electrical systems are sufficiently immune to electromagnetic interactions.

4.5 Mobile phones are intended to be electromagnetic emitters and as such their radiation characteristics (frequency, power, etc) are tightly regulated by standards set by organisations such as the European Telecommunications Standards Institute (ETSI). However, the distance between a mobile phone and an electrical system can vary considerably. A substantial research project recently concluded (DTI, 1999) that future mobile phone systems would have less adverse EMC effects than present systems, and suggested some techniques for reducing the effects still further.

4.6 EMC is of particular concern in hospitals because of the diversity of electronic equipment in use and safety-critical circumstances involved. The Medical Devices Agency issued a warning in 1994 and recommendations in 1997 (MDA, 1997) and many hospitals have imposed restrictions of varying degrees on the use of mobile phones in hospitals. Similarly, the use of mobile phones in aircraft is not permitted for EMC reasons.

4.7 EMC issues have not been considered in any detail as part of the work described in this Expert Group report.

Technology of Cellular Mobile Phones

Cellular radiofrequency networks

4.8 A mobile phone sends and receives information (voice messages, fax, computer data, etc) by radiocommunication. Radiofrequency signals are transmitted from the phone to the nearest base station and incoming signals are sent from the base station to the phone at a slightly different frequency. Once the signal reaches a base station it can be transmitted to the main telephone network, either by telephone cables or by higher frequency (such as 13, 23 or 38 GHz) radio links between an antenna (eg dish) at the base station and another at a terminal connected to the main telephone network. These microwave radio links operate at rather low power and with narrow beams in a direct line of sight between the antennas, so that any stray radiation from them is of much lower intensity than the lower frequency radiation transmitted to the phones (FEI, 2000)[*].

4.9 Signals to and from mobile phones are usually confined to distances somewhat beyond the line of sight. They can reach into buildings and around corners due to various processes including reflection and diffraction, that allows the radiation to bend round corners to some degree, but the coverage area from a base station is partly governed by its distance to the antenna's horizon. In the current GSM system (see paragraph 4.11), a timing artefact in the signal processing within the receivers limits the maximum distance over which a mobile phone can be used to about 35 km (22 miles). For such reasons an extensive network of base stations is needed to ensure coverage throughout the UK. An ideal network may be envisaged as consisting of a mesh of hexagonal cells, each with a base station at its centre (Figure 4.2), but in practice the coverage of each cell will usually depart appreciably from this because of the topography of the ground and the availability of sites for the base stations. The sizes of the cells are usually less than the 35 km maximum because obstruction by hills, buildings and other ground features reduces the effective range. Frequencies are reused several cells away and the capacity of a network (the number of simultaneous phone calls which may be made) depends on the extent of the frequency spectrum available, the cell diameter and the ability of the system to work against a background of interference from other cells. To accommodate the steadily increasing volume of users, cell sizes have to be progressively reduced (for example, by using base station antennas of lower height and reduced power) so that the frequencies may be reused more often. Indeed in large cities, base stations may only be a few hundred metres apart. The 20,000 or so base stations in the UK

[*] The maximum intensity on the ground 15 m from an antenna of a microwave link is stated to be 45 $\mu W/m^2$.

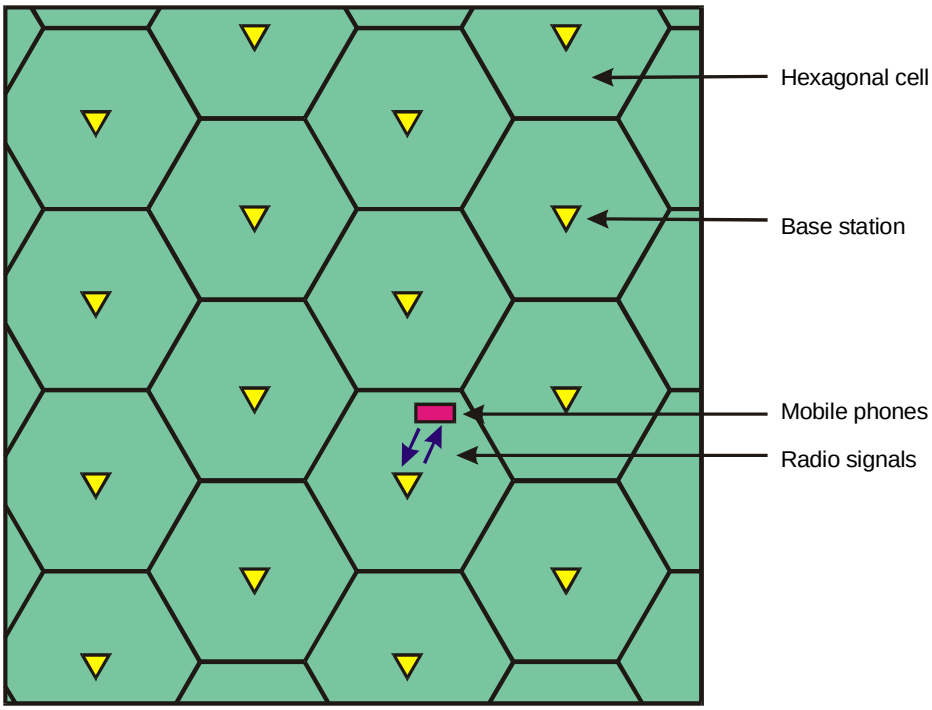

Figure 4.2 Network of base stations at the centre of hexagonal cells

mentioned in paragraph 2.14 each serve a "macrocell". Additional, smaller base stations* operating over even shorter distances are being installed in places such as railway stations where the density of users is particularly large ("microcells") and also within buildings such as office blocks ("picocells"). Cellular systems also include technology that ensures that the frequency channels employed by a user in a vehicle change automatically as the vehicle moves from one cell to the next.

Cellular phone technologies

TACS (analogue)

4.10 The first cellular system employed in the UK was the analogue TACS (Total Access Communication System) for which the phones have a nominal output of 0.63 W (FEI, 2000). This system is being phased out so that the frequency channels it uses around 900 MHz may be allocated to more recent systems. It uses frequency modulation† that results in only very small and essentially random changes in the amplitude of the carrier wave.

GSM (digital)

4.11 Systems using the TACS standard have largely, although not entirely, been replaced by the European digital phone standard, GSM, the acronym for Global System for Mobile Communications (Pederson and Anderson, 1999; Steele and Hanzo, 1999), and mostly operate in either the 900 MHz or 1800 MHz band. This standard is now widely used in many parts of the world. The digital processing uses phase modulation‡ that again results in only very small and essentially random changes in the amplitude of the carrier wave.

* For indoor propagation, it is possible that "leaky feeders" consisting of specially constructed cables may be used as sources of low intensity radiation.

† In frequency modulation, the frequency of the carrier is varied by an amount proportional to the size of the modulating signal. The amplitude of the carrier wave is not changed.

‡ Two electromagnetic waves may have exactly the same frequency but may be "out of phase". This means that one wave is displaced from the other so that, for example, when one has its maximum positive value, the other has its maximum negative value. When a wave is phase modulated, each digit that is transmitted introduces a phase change in the carrier wave and the change produced by a one is different from that produced by a zero.

4.12 In the GSM system, each user requires a frequency channel of bandwidth 200 kHz so there is a maximum of 174 channels (175 minus one needed for technical reasons) within the 35 MHz bandwidth of the 900 MHz band and 374 within the 75 MHz width of the 1800 MHz band available for allocation to network operators. The channels are distributed across the cells in a way that allows neighbouring cells to operate at different frequencies to avoid interference. Cells are very often divided into three 120° sectors with different frequencies for each. These considerations limit the number of frequency channels available to users in a particular sector. Since the wavelengths at 900 MHz are twice as long as those at 1800 MHz, they are better at reaching the shielded regions behind buildings, etc, as a result of diffraction (bending). So, to obtain the same coverage, fewer base stations and hence fewer channels are needed at 900 MHz than at 1800 MHz. One 2 One and Orange were in fact allocated 150 channels within the 1800 MHz band, and BT Cellnet and Vodafone were allocated 113 channels within the 900 and 1800 MHz bands.

4.13 To increase the number of users that can communicate with a base station at the same time, a technique called Time Division Multiple Access (TDMA) is employed that allows each channel to be used by eight phones. This is achieved by compressing each 4.6 ms chunk of information to be transmitted into a burst or pulse 0.58 ms long (1 ms or millisecond is a thousandth of a second). So the phones and base stations transmit for 0.58 ms, every 4.6 ms, which results in a 217 Hz pulse modulation* or variation in their output (217 Hz = 1/4.6 ms). For technical reasons, there is, in fact, additional data compression which leads to the phones and base stations transmitting 25 pulses but omitting every 26th, and so on. This produces further pulse modulation of the power output at the lower frequency of 8.34 Hz (= 217 Hz/26). There is, however, no detectable amplitude modulation at the frequency of 271 kHz (every 4 μs) at which the individual digits (zeros or ones) are transmitted since, as noted earlier (paragraph 4.11), this leads to a negligible change in amplitude.

4.14 The maximum powers that GSM mobile phones are permitted to transmit by the present standards are 2 W (900 Hz) and 1 W (1800 Hz)†. However, because TDMA is used, the *average* powers transmitted by a phone are never more than one-eighth of these maximum values (0.25 W and 0.125 W, respectively) and are usually further reduced by a significant amount due to the effects of adaptive power control and discontinuous transmission. Adaptive power control (APC) means that the phone continually adjusts the power it transmits to the minimum needed for the base station to receive a clear signal. This can be less than the peak power by a factor of up to a thousand if the phone is near a base station, although the power is likely to be appreciably more than this in most situations. Discontinuous transmission (DTX) refers to the fact that the power is switched off when a user stops speaking either because he/she is listening or because neither user is speaking. So if each person in a conversation is speaking for about half the time, he/she is only exposed to fields from the phone for that half of the conversation. In summary, the largest output from a phone occurs if it is mainly used at large distances from the base station or shielded by

* Pulse modulation is equivalent to amplitude modulation by several frequencies at once. Thus pulse modulation at 217 Hz is equivalent to amplitude modulation at 217 Hz (23.4%), 434 Hz (21.6%), 651 Hz (18.9%), etc; the figure in brackets shows the modulation amplitude compared with that of the pulse. At 8.34 Hz, the frequencies and sizes of the amplitude modulation are 8.34 Hz (1%), 16.68 Hz (1%), 25.02 Hz (1%), etc.

† To allow for small variations in performance that occur when a batch of phones is produced, standards require these powers to be met to within ±78% (±2.5 dB; a change in power from P_1 to P_2 can be described as a change of $10 \log(P_2/P_1)$ dB or decibels where the log is to base 10). Improvements in manufacturing techniques since the standards were set have, however, substantially reduced the variations in performance and it appears very unlikely that any of the more recently produced mobile phones would approach the upper limits allowed by the standards, particularly as an important aim of the manufacturers is to achieve the greatest possible battery life which requires the power used to be as small as possible. Advice from the Mobile Manufacturers Forum (an international association of seven major manufacturers) notes that members of the Forum are not aware of any phones operating above the standard. Nevertheless it seems possible that some older phones might still be in use whose maximum powers are 3.56 W (900 Hz) and 1.78 W (1800 Hz).

buildings, etc. In this situation, the *peak* powers could approach the values of 2 W (900 Hz) and 1 W (1800 Hz) and the *average* powers could approach the values of 0.25 W (900 Hz) and 0.125 W (1800 Hz).

UMTS/IMT-2000 (digital)

4.15 A third generation of mobile telecommunications technology has now been agreed and will be introduced in the next few years. In Europe this is called UMTS (Universal Mobile Telecommunication System) and worldwide it is known as IMT-2000 (International Mobile Telecommunications - 2000). The frequency bands identified for this system are 1885–2010 MHz and 2110–2200 MHz and the need for additional frequency spectrum to meet the future expected demand for capacity has also been recognised and will be debated at the World Radiocommunication Conference in May 2000. The specifications allow some choice in the modulation to be used but it is expected that the main choice will be CDMA (Code Division Multiple Access). The frequency channels will have 5 MHz bandwidths and, as in GSM, each can be used by a number of users at the same time. However, in CDMA, a transmission is "labelled" by a coding scheme that is different for each user. Since all the transmissions occur at the same time, the changes in amplitude of the carrier wave are essentially random (noise-like).

4.16 Two types of CDMA are likely to be implemented: FDD (Frequency Division Duplex), where separate 5 MHz channels are used for the two directions (to and from the mobile phone), and TDD (Time Division Duplex) where the same channel is used but in different time slots. Both types lead to pulse modulation because of the need to send regular commands from the base station to change the power level. In FDD the pulse frequency is 1600 Hz, while for TDD it can vary between 100 Hz and 800 Hz (Pederson and Anderson, 1999).

4.17 The expected demand for the use of UMTS both for speech and for data and Internet services is such that systems may be expected to employ macrocells and microcells, and also short-range picocells, to meet the various requirements for mobility and wide bandwidth services – for example, in the office environment.

DECT (digital)

4.18 Cordless phones are used at very short ranges between a base station located at the telephone socket outlet within the house or office and the cordless phone handset. Earlier cordless phones used analogue technology and are now being replaced by a digital system, DECT (Digital Enhanced Cordless Telecommunications) which has performance advantages in terms of privacy and protection against interference. DECT is now in widespread and increasing use and operates at similar frequencies, around 1850 MHz, to cellular mobile phones. There are ten channels with a spacing of 1.728 MHz. In each channel there are 24 time slots within a 10 ms frame and the transmission within a slot uses a form of frequency modulation. So a particular phone emits a pulse every 10 ms (100 Hz) during one of the time slots. Since the maximum power emitted is 250 mW, the average power emitted is about 10 mW. Possibly, DECT technology may form part of an overall UMTS system.

TETRA (digital)

4.19 The new TETRA (Terrestrial Enhanced Trunk Radio System) technology is not intended for public systems connected to the telephone network. It is designed for closed groups (eg for communication within an organisation or company) and is coming into use for the emergency services and some commercial applications. Frequency bands are available at about 400 MHz and 900 MHz. The modulation method is complex. The main features, however, are a 25 kHz band divided into four frequency channels, each of which is divided into 56.7 ms frames containing 4 time slots. So the transmission is pulsed at 17.6 Hz (1/56.7 ms).

Other radio systems

4.20 A modern environment contains many types of radiotransmitter. Broadcast radio and television transmitters usually have substantially higher powers than those of mobile phone base stations because they are designed to serve large areas of the countryside. For the same reason, their antennas are usually placed on taller masts located on higher ground at some distance from centres of population. Other high power transmitters are used for air traffic control and surveillance radar, which usually employ pulse modulation. Transmitters of much lower power, roughly comparable to those of the macrocell base station transmitters used in mobile telecommunications, are used for other communications purposes such as radiopaging and communications by the police, emergency services, local government, utility services, security personnel, amateur radio operators, and taxi services. They vary widely in the type of coverage needed but a large number of transmitters is needed for many of the services because of their relatively low power outputs. So it is important to recognise that the exposure from mobile phone base stations is just one component of the total RF exposure that people receive. Indeed, the exposure received by people living near to broadcast transmitters of high power output is likely to be appreciably greater than that received by people living near to mobile phone base stations, although less than that from a mobile phone near to the body.

4.21 Individuals may also be exposed to radiation from nearby low power transmitting devices such as wireless burglar alarms, toys, baby alarms, microphones, theft protection devices and car door openers. All of these types of equipment are of such low power that they do not need individual spectrum licences.

4.22 There are also RF amplifiers, which are used in such a way that they are not intended to radiate. These include RF heating – for example, in the plastics industry – microwave diathermy in physiotherapy and microwave ovens. Some of these sources, such as industrial heat sealers and medical diathermy equipment, give rise to exposures to patients, workers and physicians that are far higher than those to the public from mobile phone base stations, although the exposures are for far less time.

Electric and Magnetic Fields, Intensities

4.23 An electromagnetic wave consists of electric and magnetic fields that oscillate between their peak (largest) values (positive and negative) and zero. The size of a field can be indicated either by the magnitude of the peak value or by an average value. Since the field is positive for half the time and negative for the other half, its mean value is zero. So the average used is the rms or root mean square value (the square root of the average of the square of the field) which is equal to the peak value divided by 1.4 ($\sqrt{2}$). All fields in this report are quoted in rms values unless otherwise indicated. The electric (*E*) fields are measured in volts per metre or V/m and the magnetic (*B*) fields (or magnetic flux densities) in tesla or T or, more usually, in mT (a thousandth of a tesla) or μT (a millionth of a tesla). (The magnetic *H*-field, measured in amperes per metre or A/m, is sometimes stated rather than the *B*-field. In the materials of interest here, an *H*-field of 1 A/m corresponds to a *B*-field of 1.3 μT.) If an electrically charged object such as an ion (an atom or group of atoms which has lost or gained one or more electrons) or a cell is exposed to an electric field, it feels a force of magnitude proportional to the field. If, however, it is exposed to a magnetic field it only feels a force if it is moving at an angle to the field. The size of the force is proportional to the magnetic field and to the speed at which the object is moving across the field. Magnetic fields can also interact strongly with magnetic material such as iron. The intensity *I*, or power density, of an electromagnetic wave is the power passing through 1 m^2, as illustrated in Figure 4.3. The power is usually measured in watts (W), milliwatts (mW) or microwatts (μW), where 1 W = 1,000 mW = 1,000,000 μW, and the intensity is measured in watts per square metre

or W/m^2 (or in mW/m^2 or μW/m^2). Since the area of a sphere surrounding a source increases as the square of its radius, then in an ideal case (in the absence of any nearby objects including the ground) the intensity falls off as 1/(distance)^2, the inverse square law.

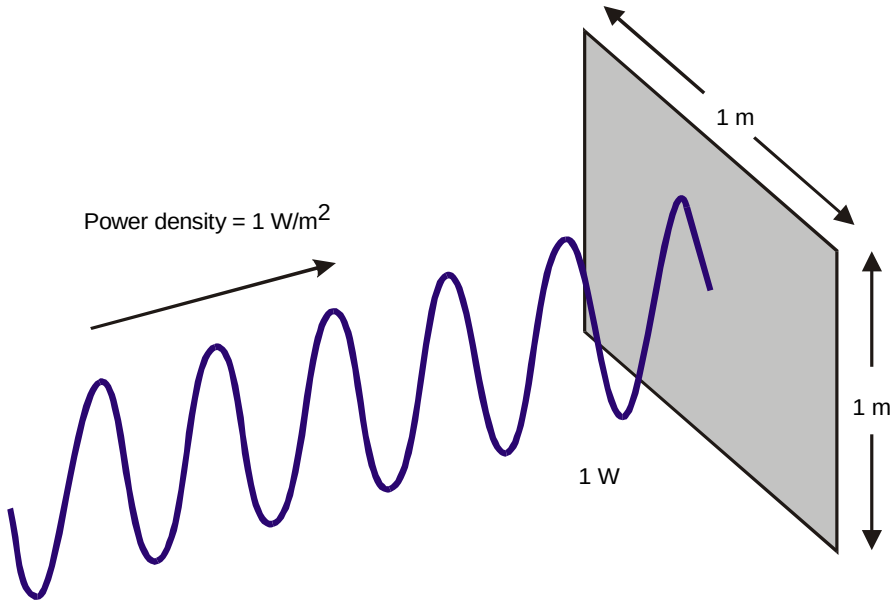

Figure 4.3 Electromagnetic wave passing through 1 m^2. If the power passing through the area is 1 W, the wave has an intensity or power density of 1 W/m^2

4.24 The properties of an electromagnetic field change with the distance from the source. They are simplest at distances of more than a few wavelengths – around a metre or more at the frequencies of interest here – which is referred to as the far-field region. In this region, the electromagnetic wave consists of an electric field *E* and a magnetic field *B* oscillating at right angles both to each other and to the direction in which the power of the wave is travelling (the direction of the intensity). The fields are in phase, so that the point at which *E* is greatest coincides with the point at which *B* is greatest, and their magnitudes are related to the intensity *I* (in W/m^2) by the expressions:

$$E = 19\sqrt{I} \text{ V/m}$$

$$B = 0.06\sqrt{I} \text{ μT}$$

4.25 In the near-field region, however, the situation is more complicated. The amount of power being radiated outwards is the same as that in the far-field region, but near to the antenna a considerable amount of electromagnetic energy is also being stored. So as well as the net radiated energy flowing outwards, there is additional energy that oscillates to and fro. These oscillating flows occur perpendicularly to the outward direction from the antenna as well as along it so the net energy flow is tilted with respect to the outward direction. The *E*-field and *B*-field are still at right angles to each other and to the direction in which the energy is being carried, but they are no longer in phase and their values can differ appreciably from the simple expressions that apply in the far-field region.

4.26 The difference in these properties near and far from an electric dipole antenna is illustrated in Figure 4.4, which shows the directions in which most of the energy flows. (The electric field directions are in the plane of the paper and perpendicular to the directions of energy flow, while the magnetic field directions are perpendicular to the paper.) Far from the antenna, the energy flows outwards. However, near to the antenna, most of the energy is stored around the antenna, flowing to and fro along its length, and only a small proportion is radiated outwards.

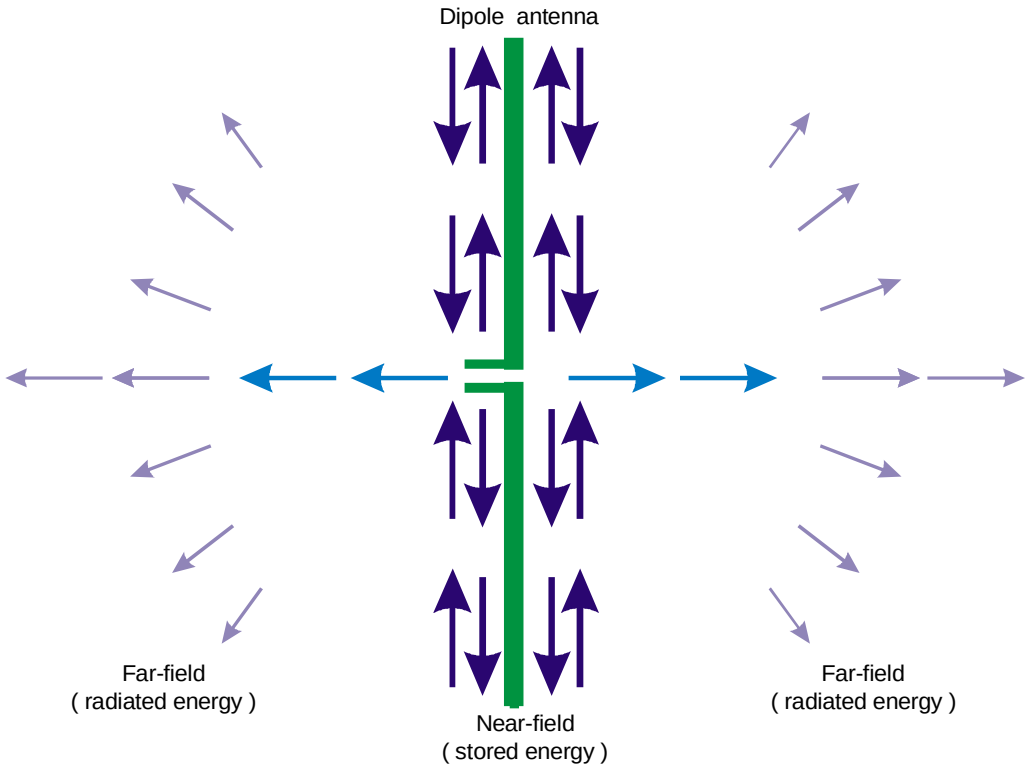

Figure 4.4 Electric dipole antenna showing the directions in which most of the electromagnetic energy flows

Fields from Mobile Phone Systems

4.27 The considerations in this section are restricted to the fields produced by GSM mobile phones and base station antennas since these form the large majority of those presently in use in the UK and Europe.

Output from mobile phones

4.28 The RF power from a phone is mainly transmitted by the antenna together with circuit elements inside the handset. The antenna is usually a metal helix or a metal rod a few centimetres long extending from the top of the phone. Neither type is strongly directional, although more power is radiated in some directions than others. At points 2.2 cm from an antenna (the distance at which calculations were made), the *maximum* values of the electric field are calculated to be about 400 V/m for a 2 W, 900 MHz phone and about 200 V/m for a 1 W, 1800 MHz phone and the

maximum magnetic field is calculated to be about 1 μT for both phones*. For both 2 W, 900 MHz phones and 1 W, 1800 MHz phones the *maximum* intensity 2.2 cm from the antenna is very roughly about 200 W/m^2 (this is about one-quarter of the intensity of the Sun's radiation on a clear summer day, although the frequency of the emission from a phone is a million or so times smaller). These are the fields and intensities when the antenna is a long way from the head or body. When the antenna is near the body, the radiation penetrates it but the fields inside are significantly less, for the same antenna, than the values outside. For example, the largest *maximum* fields inside the head when its surface is 1.4 cm from the antenna are calculated to be about three times smaller than the values given above. (The *average* field values are all appreciably less than these *maximum* values for the reasons explained earlier.) As well as these RF fields, that are pulsed at 8.34 Hz and 217 Hz, there are magnetic fields near to the phone that oscillate at these same frequencies, and are a few μT in magnitude. These are generated by currents flowing from the battery which are switched on and off at these frequencies as a result of TDMA.

4.29 An indication of the size of these fields (although not of course any effect they may have) may be obtained by noting that the *maximum* values of these low and high frequency oscillating magnetic fields are about one-tenth the size or less of the Earth's static magnetic field, 50 μT, while the *maximum* values of the oscillating electric fields outside the body are a few times greater than the electric field at the surface of the Earth due to its static charge. This is directed towards the ground and on a fine day has a constant value of about 100 V/m.

Output from base stations

4.30 The base station antennas serving macrocells are either mounted on free-standing towers, typically 10–30 m high, on short towers on top of buildings, or attached to the side of buildings. In a typical arrangement, each tower supports three antennas, each transmitting into a 120° sector. A large proportion of the power is focussed into an approximately horizontal beam typically about 6° wide in the vertical direction and the rest goes into a series of weak beams (called side lobes) either side of the main beam. The main beam is tilted slightly downwards (Figure 4.5) but does not reach ground level until the distance from the tower is at least 50 m (usually 50–200 m).

4.31 The base station antennas transmit appreciably greater power than the phones. The limit to the power is formally set by the need to avoid RF interference and defined by a licence issued by the Radiocommunications Agency. This does not directly limit the total power emitted but does so indirectly by fixing the maximum intensity that an antenna can transmit into the main beam. This is done by defining the maximum "equivalent isotropically radiated power" (EIRP) that can be transmitted. The EIRP is the power that would have to be emitted equally in all directions to produce a particular intensity. In fact, as already noted, the antennas used are very far from isotropic, with most of the power being emitted into the main beam, and the ratio of the EIRP to the total power output is called the gain of the antenna. For a 120° sector antenna the gain is usually between about 40 and 60.

* It has already been noted that the electric and magnetic fields vary in rather complicated ways at distances from an antenna that are small compared with the wavelength λ of the radiation (33.3 cm at 900 MHz and 16.7 cm at 1800 MHz). Therefore detailed calculations are needed to obtain accurate values for the intensities and fields near to a phone and the approximate values given here are only intended to give indications of their size. The field values given above, when the antenna is a long way from the head or body, are from computed values for a particular antenna (Dimbylow and Mann, 1994; Mann *et al, in press*). The largest values of electric field *E* inside a model of a head whose surface is 1.4 cm from the antenna were also computed and are about 120 V/m for a 900 MHz antenna radiating 2 W and 70 V/m for a 1800 MHz antenna radiating 1 W. (These are obtained from their published figures of the specific absorption rates (SARs) inside the head; SAR is defined in paragraph 4.37). The value for the intensity was obtained by assuming the antenna to be a rod of length $l = \lambda/4$. The average value a distance r from the antenna is very roughly equal to $P/2\pi rl$ since nearly all the radiated power, P, has to pass through a cylinder of area $2\pi rl$. The intensities are the same for both 900 MHz and 1800 MHz GSM phones since P/l is the same for both.

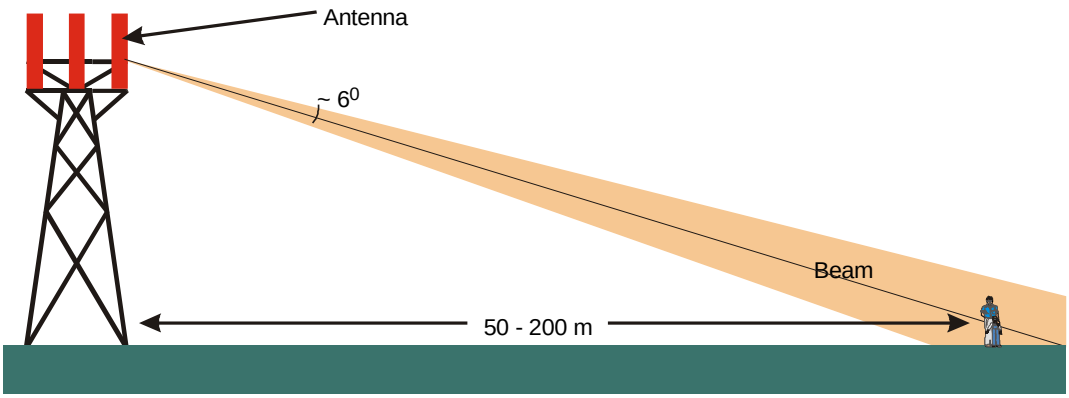

Figure 4.5 Main beam from an antenna mounted on a tower. The beam is in fact less well defined than that shown here and there is a series of weak side lobes either side of it

4.32 The licence sets the maximum EIRP at 1500 W per frequency channel corresponding to a maximum total radiated power of about 30 W per channel (= EIRP/gain). It also limits the number of channels per antenna to 16 (for 1800 MHz) and 10 (for 900 MHz). However, we have been told that in practice the number of channels is typically less than 4 for 1800 MHz and 2 to 4 at 900 MHz (FEI, 2000), which would correspond to maximum radiated powers of less than 120 W and 60–120 W, respectively. Similarly, the total radiated power emitted from an antenna is generally limited by the characteristics of the equipment to somewhat under 70 W (FEI, 2000), and a figure of 60 W will be assumed in this report. It needs to be stressed that the number of channels used, and hence the total radiated power, is limited by technical rather than legal requirements, which would in fact permit significantly larger powers to be radiated. As with a phone, and for largely the same reasons, the *average* power transmitted by a base station is normally less than the *maximum* power, although in this case it could rise to the maximum at times (rather than to one-eighth of the peak power in the case of a phone). By the inverse square law, the *maximum* intensity in the main beam at a point on the ground 50 m from a 10 m tower carrying an antenna transmitting 60 W into a 120° sector is about 100 mW/m^2 [*]. This corresponds to oscillating electric and magnetic fields of about 5 V/m and 0.02 μT, respectively, very roughly about 50 to 100 times smaller than those 2.2 cm from the antenna of a phone. The heating effects that these fields would produce will vary with the intensity and are about 5000 times smaller than the maximum value 2.2 cm from the antenna of a mobile phone.

4.33 The RF intensity on the ground is not zero outside the main beam, because of the power emitted into the side lobes. Its value will depend on the design of the antenna but it seems unlikely that it could ever be significantly more than that within the beam. So the values given above should be reasonable indications of the maximum intensity and fields that would be present on the ground around a base station. The intensity will, however, become appreciably larger as the antenna is approached, as it might be by maintenance workers.

[*] If the power P were transmitted equally into all directions, the intensity at a distance r would be $P/4\pi r^2$ (the power passes through a sphere of area $4\pi r^2$) which is about 2 mW/m^2 at a point on the ground 50 m from the bottom of a 10 m tower transmitting P = 60 W. For a 120° sector antenna with a gain of 50 the intensity at the centre of the beam rises to around 100 mW/m^2. However, even though the intensity *at a particular distance* is greatest at the centre of the beam, the intensity on the ground is somewhat larger at angles just away from the centre since the distance from the antenna is less (see Figure 4.5). So the region of greatest intensity lies between the points where the centre of the main beam hits the ground and the points where the nearest edge of the beam hits the ground (for this purpose we define the edge as occurring at the angle at which the intensity falls by half, 3 dB). In this report we refer to the part of the beam producing this region as the *beam of greatest intensity*.

4.34 In the last year or so NRPB has made spot checks on the average intensities around base stations. Eight of these stations were mounted on the roofs of schools; four were on tower blocks and five on other buildings. Measurements were made at various points within the buildings, at ground level or at other locations of public access (Mann *et al, in press*). The measured intensities were typically between 0.01 and 1 mW/m^2 and the maximum was never more than 10 mW/m^2. These values are then very much less than the calculated values in the beam given above, although the sample is small. It is also of note that the calculations and most of the measurements were for towers used by one operator only. The average intensities would be expected to be larger near to a tower used by more than one operator.

4.35 We note that these measurements by NRPB were spot checks made under contract at the request of a client such as a local authority. Neither NRPB nor any other independent agency has made any systematic experimental study in the UK of, for example, how the intensity changes with distance from a base station, although such studies have been reported in the USA. The NRPB report also includes the measurements made during these spot checks of the intensities due to radio and TV transmissions but again there have been no systematic studies which would have allowed us to make a useful comparison of the intensity of typical exposure levels received by individuals from mobile phone transmitters compared with those from other RF sources. Surveys of this sort have been conducted in the USA but they are several years old and have been made obsolete by the rapid development of wireless technologies. This is, indeed, a very complex problem given the great diversity of RF sources that are presently in operation.

4.36 Two further properties of the electromagnetic waves emitted by both mobile phones and base stations, that might be of significance in their interaction with biological tissue, are their frequency spectrum and coherence time. The emission from a mobile phone is essentially at one frequency and that from a base station is at several specific frequencies and, in both cases, the waves have the relatively long coherence time of around 4 μs (the coherence time is the average time between random phase changes, which in this case are the result of phase modulation, see paragraph 4.11). Both these properties are very different from those of, say, the radiation from the Sun which consists of a broad spectrum of frequencies and electromagnetic waves with coherence times which are shorter by a factor of around a hundred-thousand.

Field penetration into the body: dosimetry

4.37 Radiofrequency fields penetrate the body to an extent that decreases with increasing frequency. To understand the effects this might have on biological tissue, the magnitude of the fields needs to be determined within the various parts of the body that are exposed. This requires a knowledge of the electrical properties of the different types of tissue and, once this has been determined, it is possible to calculate E and B at every part of the body caused by a particular source of radiation such as a mobile phone. The rate at which the energy is absorbed by a particular mass of tissue m, is $m\sigma E^2/\rho$, where σ and ρ are, respectively, the conductivity and density of the tissue and E is the rms value of the electric field. The quantity $\sigma E^2/\rho$ is called the specific energy absorption rate or SAR and is measured in watts per kilogram (W/kg). It varies from point to point in the body both because the electric field changes with position and because the conductivity is different for different types of tissue. (The density is much the same for all tissues apart from bone.) Since the average values of the conductivity at 900 MHz and the density of body tissue are 1 S/m and 0.001 kg/m^3, respectively, the typical value of electric field needed to produce an SAR of 1 W/kg is about 30 V/m. (The average value of conductivity is somewhat higher at 1800 MHz so lower electric fields, about 25 V/m, are needed.) The SAR produced by a particular value of electric field is somewhat larger in children than in adults because their tissue normally contains a larger number of ions and so has a higher conductivity (Gabriel, 2000). We understand that an internationally agreed standard testing procedure that will allow the SAR from mobile phones to be compared is being developed and will be finalised this year (2000).

4.38 It is important to stress that these are the electric fields *inside* the body. The fields outside the body that correspond to these internal fields are typically around three times larger; this was discussed in paragraph 4.28.

4.39 It is very well established that electromagnetic radiation can only be absorbed in quanta of energy $h\nu$, where h is Planck's constant. Now the energy needed to remove an electron from (ionise) an atom or molecule is a few electron volts (eV)[*] (an eV is the energy needed to move an electron of charge e from an earthed plate to one at a negative voltage of one volt). So if the quantum of energy is less than about 1 eV, it is essentially impossible for ionisation to occur[†]. The quantum of energy of RF radiation is in fact many thousand times less than 1 eV so RF radiation cannot ionise atoms or molecules and is described as non-ionising radiation (NIR). However, higher frequency radiation, such as far-ultraviolet radiation and X-rays, has energy quanta bigger than 1 eV and so can readily ionise atoms and molecules, and produce some damage to biological tissue even at very low intensities. This is referred to as ionising radiation. The intensity determines the number of quanta striking the body per second and, even though this is small at low intensities, each quantum still has a certain probability of ionising and so damaging biological molecules such as DNA. Non-ionising electromagnetic radiation, however, is believed to be harmless at very low intensities, although it can be damaging at high intensities. For example, light at modest intensities produces useful biological effects which allow us to see illuminated objects. However, if the intensity of the light becomes too large, the eye can be seriously damaged. Very high intensity RF radiation can also be damaging as is clear from the strong heating effects produced in a microwave oven. So we need to know at what intensity the radiation starts to produce damage; this might usually be expected to be higher than the lowest intensity at which biological effects can be detected. The current guidelines that are in force to protect people from harmful exposures are discussed in paragraphs 6.19–6.32.

[*] The ionisation energy has been quoted as a few electron volts, although the usual boundary between ionising and non-ionising radiation in biological material is taken as around 10 eV, reflecting the fact that the atoms that are present have ionisation energies that are greater than average.

[†] It is possible for two or more quanta to be absorbed simultaneously. However, the probability of this happening falls rapidly with the number of quanta and becomes minute if thousands of quanta are to be absorbed at the same time.

5

Scientific Evidence

5.1 Assessment of any health risk resulting from exposure to radiofrequency (RF) fields depends on the results of well-conducted and reproducible scientific studies, and the need for these is all the greater because any effect of exposure to RF fields at the levels encountered from mobile telecommunications is likely to be subtle. Reports of such studies are mainly found in peer-reviewed journals, although the Expert Group considered evidence from all sources of information available to it.

5.2 The World Health Organization (WHO) defines health as the state of complete physical, mental and social well-being, and not merely the absence of disease or infirmity. Thus the Expert Group considered ways in which mobile phones and their base stations might jeopardise any of these aspects of the health of people.

5.3 We are aware of the concerns about the biological effects and possible health risks of exposure to RF fields expressed by a group of scientists who met in Vienna in October 1998. The evidence reviewed in this chapter addresses the issues in detail.

5.4 New telecommunications technologies have been introduced without full provision of information about their nature and without prior discussion within the scientific community about possible consequences for health. The average output power from the antennas of digital mobile phones is lower than that from earlier analogue models, but the maximum powers are greater, the exact patterns of radiation are different and these differences might influence their effects on people. As the costs of mobile phone technology have fallen, their use has increased dramatically and the overall levels of exposure of the population as a whole have therefore increased.

Interaction of Radiofrequency Fields with Tissues

Mechanisms

5.5 While risk assessment is generally based on experimental data from biological systems, a consideration of possible mechanisms is nevertheless pertinent, for two reasons. Firstly, as discussed below, the experimental data regarding biological effects of RF fields are fragmentary and inconsistent in many respects, and an understanding of the biophysical mechanisms for reported effects can help to rationalise and understand the data. Secondly, it is necessary to extrapolate data from one exposure condition to others, and for reliable extrapolation some understanding of the underlying mechanisms is needed.

5.6 The electric and magnetic fields produced in the body by a nearby electromagnetic source may cause both thermal and non-thermal biological effects. The effects of magnetic fields vary with frequency, and are probably greatest in biological tissue containing small amounts of magnetite. Magnetite (Fe_3O_4) is a naturally occurring oxide of iron. It is a ferrimagnet but behaves similarly in magnetic fields to a ferromagnet such as iron. Magnetite is found in certain bacteria and in the

cells of many animals, including human beings. It is believed to be used by some species of birds and fish to provide magnetic sensitivity, which they employ in navigation. However, no other effects associated with the interactions of electromagnetic fields with magnetite have been demonstrated in animals. It has been calculated that the interaction resulting from the largest RF magnetic fields generated by mobile phones is extremely small (Adair, 1994), and that any other effects of magnetic fields at these frequencies should be even smaller. Indeed, it seems to be generally agreed that any biological effects from mobile phones are much more likely to result from electric rather than from magnetic fields.

Thermal effects

5.7 Thermal effects are those caused by the rise in temperature produced by the energy absorbed from oscillating electric fields. The force produced by an electric field on charged objects, such as the mobile ions present in the body, causes them to move, resulting in electric currents, and the electrical resistance of the material in which the currents are flowing results in heating. This heat input causes the temperature to rise and it continues to do so until the heat input is balanced by the rate at which it is removed, mostly by blood flowing to and from other parts of the body. It is estimated that it takes several minutes from the moment RF exposure occurs for the irradiated parts of the body to reach their final equilibrium temperatures. In view of this slow response, the equilibrium temperature arising from the pulsed fields of mobile telecommunications will essentially be determined by the *average* power absorbed. There will, however, be small oscillations about that temperature at the pulse frequency or frequencies.

Heating in the head

5.8 It has not yet proved possible to measure these small changes in temperature directly, except those at the outer skin (Adair *et al*, 1999) and, although temperature is a more direct determinant of thermally induced tissue damage, the majority of theoretical studies up to the present time have restricted themselves to the computation of SAR alone (paragraph 4.37).

5.9 The relationship between the SAR and the resulting temperature rise is complex, and significantly dependent on antenna configuration, location and frequency. The most problematic feature of a temperature calculation is modelling the effect of blood flow on heat transfer. The traditional continuum heat-sink model developed by Pennes (1948) has been found to give remarkably accurate results in many circumstances, but numerous modifications have been suggested more recently (Arkin *et al*, 1994).

5.10 In a recently published study (Van Leeuwen *et al*, 1999) the heat deposition within the head was computed by coupling a finite difference time domain model for SAR with a new thermal model. The thermal model includes the convective effects of discrete blood vessels, whose anatomy was determined using magnetic resonance angiography of a healthy volunteer. For a 915 MHz dipole antenna with a time-averaged power output of 0.25 W (equivalent to a typical mobile phone), this study results in an SAR of about 1.6 W/kg and predicts a maximum brain temperature rise of 0.11˚C in the steady state. There is general agreement between the brain temperatures calculated using the Pennes equation and that using the new discrete vessel model, which suggests that the sensitivity of the results to the exact blood flow model may not be critical. However, further work should be done to apply this model to more realistic simulations of mobile phone configuration, and to investigate the effect of different antenna positions and frequencies (particularly in the 1800 MHz band also used by mobile phones).

5.11 A recent NRPB study (Wainwright, *in press*) has applied the traditional Pennes thermal model to the SAR patterns predicted by earlier work (Dimbylow and Mann, 1994). The radiation source was modelled as a monopole antenna on a metal box, and both horizonal and vertical orientations

of the antenna were considered. Computations of the final steady-state temperature rise were carried out for a 0.25 W antenna at frequencies of 900 and 1800 MHz. The highest temperature rises found in the brain were around 0.1˚C.

Non-thermal effects

5.12 The energy quanta of radiation at 0.9 and 1.8 GHz equal 4 and 7 μeV, respectively (1 μeV is a millionth of an eV). Both these values are extremely small compared with the energy of around 1 eV needed to break the weakest chemical bonds in genetic molecules (DNA). As already noted, it seems impossible, therefore, that RF radiation could damage DNA directly, which might start cells on the path to cancer.

5.13 Radiofrequency radiation could, however, produce other effects. In general, detectable changes can arise only if the effect of the electric field within the biological system exposed to RF fields is not masked by *thermal noise*. Thermal noise or random motion, also known as Brownian motion, is due to the thermal energy that all objects possess at temperatures above absolute zero. In solids, the atoms vibrate and in gases and liquids they move erratically to and fro following very frequent collisions with other atoms. So all components of biological tissue – ions, molecules and cells – are in constant motion. The thermal energy of each component has an average value of about kT, where k, Boltzmann's constant, is 86 μeV per degree and T is the absolute temperature measured in kelvin, K ($T = 273 + t$, where t is the temperature in degrees centigrade). The value of T is about 300 K at body temperature so that kT is 26 meV and, if this is much larger than the energy of the motion produced by the electric field, any effect of the field will be completely masked (not detected by any component of the biological tissue). This comparison with thermal noise should then provide a good measure of the minimum electric field necessary to produce detectable biological effects. It should be noted, however, that if there was a special case in which the biological system were resonantly sensitive at the frequency of the electric field and rather insensitive to fields at other frequencies, the comparison would need to be made with the thermal motion taking place at frequencies close to the resonant frequency. If the resonance was very sharp, this would be very much smaller than the total thermal noise, so that quite small electric fields might produce detectable effects in resonant systems of this type, should they exist in biological tissue.

5.14 This argument can be used, for example, to see whether non-thermal effects could arise from the motion of the ions discussed above. The ions are driven to and fro by an oscillating electric field, although the extent of the motion is severely reduced by the viscosity of the surrounding liquid. For a field of 100 V/m the movement is in fact less than 10^{-14} m – the diameter of an atomic nucleus – and the energy associated with this motion is less than that of the thermal motion of the ion by about a factor of 10^{15}*. This is so small that it can safely be concluded that this ionic motion could not result in any non-thermal biological effects. The expression (in the footnote) shows that the energy increases with the mass of the charged object, although, for E = 100 V/m, it would still appear to be small at these frequencies compared with thermal noise for objects such as cells of average size which have radii around 10 μm (Adair, 1994). Adair notes, however, that it could become significant for larger cells with correspondingly greater masses.

5.15 Another mechanism involving cells concerns the attraction between them in the presence of an electric field (Schwan, 1985; Adair, 1994). The electric field polarises the cell, that is to say

* The velocity of the ion is $\mu E = \mu E_0 \sin(2\pi\nu t)$, where μ, the mobility, is about 10^{-7} m^2/(V/s) for chloride, the ion of highest mobility, and ν, the frequency, is 0.9 or 1.8 GHz. This leads to a maximum displacement of $\mu E_0/2\pi\nu$ which, for an electric field E_0 = 100 V/m, equals 2 x 10^{-15} m and 10^{-15} m at frequencies of 0.9 and 1.8 GHz, respectively. So the average kinetic energy of an ion of mass m in this field is $m\mu^2E_0^2/4$ or $m\mu^2E^2/2$, where E is the rms value of the electric field. For a chloride ion this energy is equal to about 10^{-17} eV, or about 10^{-15} kT.

charges in the cell move so that one side of it becomes positive with respect to the other. The cell is then an electric dipole (like a tiny torch battery) and attracts similarly polarised cells. For typical cells and frequencies below about 100 MHz, the energies involved are calculated to become comparable to thermal noise in electric fields of $E = 300$ V/m. The energies are calculated to become appreciably less for RF fields, but Adair (1994) suggests that, since these values would depend on the detailed structure of the biological elements involved, the possibility of biological effects for fields of this size cannot be excluded.

5.16 Other possible biological effects are associated with cell membranes and the movement of currents through the membrane in either direction. Membranes are known to have strongly non-linear electric properties (Montaigne and Pickard, 1984). When a voltage is applied across the membrane, the current that flows is not always proportional to the voltage. Part of this non-linearity may, in fact, be due to the effect of the electric field on the proteins in the membrane or nearby, which assist the flow of the product currents through the membrane. The membrane also acts as a rectifier. If a voltage is connected across the ends of a wire, the size of the current that flows depends solely on the magnitude of the voltage: if the polarity of the voltage is reversed, the current changes direction but its size is unchanged. However, if the polarity of the voltage applied across a rectifier is reversed, the current changes direction but now its size also changes. So, if an oscillating voltage (electric field) is applied across a rectifier, the total current that flows when the field is in one direction is not balanced by the current when the field is in the other: an AC field produces a net DC current and hence a net flow of products through the membrane. However, the response times of the ion gates are very much slower than the period of microwave frequencies and, using data obtained from measurements on membranes (Montaigne and Pickard, 1984), it has been shown that, for electric fields of 200 V/m, the relative change in the membrane potential is very small (Adair, 1994; see also Foster, 2000a). Therefore no biological effects seem likely from this mechanism.

5.17 Many other mechanisms have been proposed by which significant biological effects from RF fields might arise, but very few, if any, appear to stand up to critical analysis of the sort presented above (Foster, 2000b). One, for which there is recent experimental support (Bohr and Bohr, 2000), is that microwave radiation might cause proteins to unfold (denature). The experiments were carried out in a modified microwave oven at 2.45 GHz, a frequency comparable to the likely torsional modes of the protein. The intensity was not specified, but seems likely to have been above ICNIRP guidelines. The experiments were very recent and have not yet been replicated. Another mechanism that has continued to create interest is based on the assumption that biological systems might interact resonantly with microwave fields. This possibility was initially discussed by Fröhlich (1968, 1980) and his work has had a considerable impact (see, for example, Penrose, 1994; Pokorny and Wu, 1998).

5.18 Fröhlich was interested in the mechanism through which the chemical energy taken into the body (food) was channelled into highly ordered processes, such as cell building, rather than into heat. His model involves the mechanical vibrations of large molecules or components of biological tissue and the way they interact with each other, which he argued could lead to the existence of a band of frequencies into which energy could be absorbed, plus a particular "coherent state" of vibration. He also considered whether quite small oscillating electric fields might put energy into this state and hence trigger significant biological changes; that it is to say, whether a living biological system might behave in a manner roughly similar to a radio receiver. A radio can detect and amplify an extremely small signal against a background of very much larger signals. It does this when the operator tunes a resonant circuit to the frequency of the carrier wave. The resonant circuit essentially responds only to electromagnetic waves of frequencies (including those generated by thermal noise – see paragraph 5.13) within a narrow bandwidth. The power

needed to amplify these waves comes from the power supply of the radio. A number of solid state systems behave in similar ways, such as narrow-band optical amplifiers, which are the basis of lasers.

5.19 The Fröhlich model has stimulated a range of other work. However, so far there appears to be no direct experimental evidence, and no convincing indirect experimental evidence, for the existence of Fröhlich's coherent state in biological systems. Moreover, the present theoretical treatments of the model do not provide estimates for the magnitude of the electric fields needed to produce biological effects. Fröhlich suggested that the findings of a number of experiments carried out at frequencies of 40 GHz and above on systems such as *E coli* bacteria and yeast cultures (see Fröhlich, 1980) might be (indirect) evidence for his model, since these frequencies lie in the range where cell membranes are expected to resonate mechanically. Four recent attempts to reproduce some of this work have failed to do so (*E coli*: Athey and Krop, 1980; Santo, 1983; yeast cultures: Furia *et al*, 1986; Gos *et al*, 1997), although there have been further reports from Balyaev and colleagues that appear to endorse the earlier research (*E coli*: Balyaev, 1992). A recent appraisal of all this work (Foster, 2000a) notes that the experiments present formidable technical problems and that, while their results may be statistically significant, it may not always be possible to eliminate systematic errors. In view of this appraisal, it is not possible to conclude that this work provides support for the existence of resonant absorption by biological tissue.

5.20 Hyland (1998) has suggested that the mechanism proposed in Fröhlich's model might lead to biological effects from electromagnetic fields at the appreciably lower frequencies of mobile phones. This would require the presence of components in the biological tissue with sharp resonant vibrational modes in this frequency range. The frequencies are lower than those expected for most components, although theoretical work (Kohli *et al*, 1981; Van Zandt, 1986; Porkny and Wu, 1998) suggests that DNA polymers and elements of fibre structures (cytoskeletons), such as microtubules and actin filaments, could have modes in this range. However, since these components are surrounded by relatively viscous fluids, their mechanical vibrations would normally be expected to be very highly damped. Thus, resonances they might have out of solution would be almost completely smeared out when they are immersed[*]. Certainly no evidence of resonant absorption[†] was found from DNA in solution (Gabriel *et al*, 1987), although this might not rule out the possibility that it occurs under the conditions in which DNA exists in tissue.

5.21 *CONCLUSION This work on DNA should be repeated under conditions more closely matched to those in tissue and similar measurements should be made on microtubules and actin filaments.*

5.22 Another hypothesis is that the interaction with biological tissue depends on the coherence of the electromagnetic fields (see paragraph 4.36). Experimental evidence in support of this idea has been given by Litovitz *et al* (1993, 1997a,b) but not yet independently replicated.

5.23 In summarising the physical basis for non-thermal effects, it is convenient to consider separately the situations near to the antenna of a mobile phone and near to a base station.

[*] Water provides an example of this effect. Water vapour shows strong resonant absorption but the resonances are smeared out in liquid water and absorption occurs over a wide range of frequencies. Scott (1984) and Van Zandt (1986) have, however, proposed models to explain why this might not happen for DNA in solution.

[†] Earlier work on DNA in solution appeared to show strong resonant absorption in this range (Edwards *et al*, 1984). It was shown, however, that this could have been the result of an experimental artefact (Foster *et al*, 1987) and, as noted, the work of Gabriel *et al* (1987), carried out on samples chosen to be as close as possible to those of Edwards *et al*, failed to see any such effects. In this work, three different techniques were used in two different laboratories and the results were essentially identical. It can be concluded, therefore, that DNA in solution does not have resonant modes that couple to microwaves in this range.

Mobile phones

5.24 In Chapter 4 it was noted that the *maximum* size of the electric fields produced in the head by the antenna of a mobile phone is around 100 V/m, although the fields inside the brain would be appreciably less. For fields of this size the mechanisms most likely to produce non-thermal biological effects would be through the movement of large cells (paragraph 5.14) or through the attraction between neighbouring cells (paragraph 5.15). At this stage, although there is no experimental evidence to support these mechanisms, the possibility that both of these could produce effects cannot be excluded (Adair, 1994).

Base stations

5.25 The maximum size of the electric fields resulting from base stations to which the general public is exposed is around 5 V/m, although the largest field measured to date by NRPB is 2 V/m (Mann *et al*, *in press*). (The corresponding field strengths inside the body will be appreciably smaller.) One mechanism that could lead to biological effects at these relatively low fields is that proposed by Fröhlich and which relies upon the existence in biological tissue of a particular coherent state of mechanical vibration. The absence, even after 30 years, of any convincing evidence for this state, or indeed for any resonant behaviour, would seem to cast considerable doubt on its existence, although it cannot totally be ruled out. It should be noted, however, that Adair (1994) has considered more generally the possibility of energy transfer through a resonant mechanism and his conclusion is that it would be too small to lead to measurable biological effects at any value of electric field.

5.26 We conclude that there is little evidence to support resonant behaviour, but further work to investigate this proposed mechanism could be worthwhile.

Experimental Studies

5.27 This section considers and interprets research on *in vitro* preparations and on animals (*in vivo*) relevant to the possibility that exposure to RF fields used in mobile telecommunications is associated with adverse health outcomes.

Significance of experiments on molecules, cells and animals

5.28 Our principal remit was to report on the possible risks to human health of mobile phones and their base stations. The most direct demonstration of such risks comes from research on people, either through epidemiological studies, which could identify an increased incidence of health problems as a result of exposure to RF fields, or by means of experiments on human volunteers aimed at revealing physiological or behavioural abnormalities resulting from such exposure. However, because of the obvious limitations of human experimentation, more direct methods are necessary to show the underlying biological basis of any health hazards. These involve laboratory studies of biological systems and animals.

5.29 Experimental observations on biological molecules (eg solutions of enzymes), and on *in vitro* preparations of isolated cells or tissue samples, could define the mechanism of any action of radiation on living organisms. A great deal of research has been performed on such preparations, particularly on cultured animal or human cells. Such work could, in principle, give insight into the basic mechanisms responsible for more complex effects seen in whole animals or people (eg tumour formation, changes in brain activity and even alterations of behaviour). The results of work on simple molecular and cellular preparations are often easier to interpret, since the nature of any effects can be more precisely defined and the conditions of the preparation (including its

temperature) can be more accurately assessed and controlled than in whole animals. However, even when clear effects are defined *in vitro*, it is often difficult to extrapolate from them to a health risk for people.

5.30 Laboratory studies on animals play an essential role in evaluating the integrated reactions of various, intact systems of the body, particularly the nervous, endocrine (hormone) and immune systems. These systems are largely responsible for homeostasis – the essential maintenance of the internal environment. The complex, co-ordinated, interdependent response of these systems when challenged by potentially damaging stimuli cannot be defined through experiments on molecules or isolated cells.

5.31 The environment and parameters of exposure to RF radiation can be made virtually the same for all animals in each group; well-matched "control" animals can be identically treated except that they receive no exposure. Valid comparisons can then be made between the exposed and control groups to determine if exposure causes any effects. The precision of this type of study also allows dose–response relationships to be determined.

5.32 Of course, phenomena seen in experimental animals do not necessarily imply a health risk for people. In particular, an effect found in only one animal species may be specific to that type of animal and not relevant to people.

5.33 Although rodents differ from people in some aspects of their physiology, mice and rats are frequently used in biomedical research. This is mainly because genetically homogeneous strains of rodents are available for research and because they are easy to breed and maintain. Another advantage of rodents accrues from the fact that they have been so widely used for research in the past and consequently much is known of their normal physiology. Moreover, mice have been the preferred species for research on genetic mechanisms and for experimental work involving transgenesis (the modification of genes).

5.34 Appropriate animal studies provide the opportunity to test whether lifetime exposure to well-characterised RF radiation causes cancer, something that is obviously impossible using human volunteers. While epidemiological studies do allow human populations to be studied, they are generally not able to assess RF exposure accurately. Research on animals can also demonstrate influences of RF exposure on susceptibility to cancer promotion and progression, as well as on various physiological functions, including behavioural performance in tasks involving learning, memory, etc. It is important to note, however, that the interpretation of any such behavioural effects in small animals is not straightforward. For instance, if radiation affects the performance of mice in tests of learning (as has been claimed: see paragraphs 5.80–5.92), one might imagine that it could influence this aspect of brain function in people. However, given the small size and somewhat different functional and anatomical organisation of the mouse brain, such a result could not easily be extrapolated to suggest a similar effect on memory in people.

Stimulus conditions

5.35 As described above, high levels of RF radiation cause heating by inducing small electric currents and increasing the movement of molecules. However, the rise in temperature in the brain caused by the use of a mobile phone for more than a few minutes (the time taken to reach thermal equilibrium) is estimated to be only about 0.1˚C (Van Leeuwen *et al*, 1999). Moreover all cells in the body have a mean thermal energy proportional to the absolute temperature T (paragraph 5.13) and the energies of individual cells fluctuate about this mean energy as they interact with other cells, extracellular fluids etc. Any increase in energy caused by radiation is

superimposed on this background of fluctuating energies and would have to begin to be comparable in size to these to cause any additional biological hazard.

5.36 Experimental studies have employed a bewildering variety of exposure conditions, with respect to the RF carrier frequency, whether it is continuous, amplitude modulated or pulsed, and, particularly, its intensity. High intensities, above present guidelines (see paragraphs 6.19–6.31), can cause significant heating (ICNIRP, 1998a,b), which itself can lead to a variety of pathological effects in cells and tissues.

5.37 We have considered the whole range of research reports, including those that employed intensities that caused heating, since they might provide clues to the mechanism of effects at more modest levels of exposure. However, we have concentrated on studies that used intensities below 50 W/m^2 (5 mW/cm^2) and/or SARs below 1 W/kg, which are less likely to have involved significant heating (see paragraph 5.8). We have also taken note of studies in which investigators used higher intensities but have attempted to prevent what they considered to be significant rises in temperature. It must be noted, however, that it is difficult to be sure of the absence of thermal effects. The absorption of RF energy is necessarily accompanied by temperature increases, which can be minimised, but not prevented. Whole-body SARs less than 1 W/kg have been shown to cause thermoregulatory responses in animals. In the case of pulsed fields, the average SAR is obviously much lower than the peak SAR during pulses, which can generate quite rapid, transient heating and quite large electric fields. This probably accounts for many of the effects, such as 'microwave hearing' (see paragraphs 5.76–5.79), that have been described at low average SARs.

5.38 We were struck by certain inconsistencies and inadequacies in the scientific literature on the biological effects of RF radiation. Many studies in this field have been exploratory and preliminary in nature, and claims of effects have sometimes been based on single experiments rather than a consistent series of hypothesis-driven investigations. In some cases, study design and statistical analysis have been inadequate, and apparent effects may have been artefactual or due to random variation. Indeed, the field is troubled by failures to replicate previous studies and by a lack of theoretical explanation of some effects that have been claimed. There may also be biases arising from selective publication and non-publication of results. Finally, even for effects that appear to be well substantiated, the biological significance and the implications for health are often unclear.

5.39 A considerable problem in the interpretation of experimental reports is that many of them have given insufficient detail concerning exposure conditions, including the important SAR value. Moreover, in the case of pulsed fields, when SAR values are quoted it is often unclear whether these refer to the average SAR or to the peak SAR during pulses. It is very important to make this distinction, since the peak SAR can be 1000 or more times the mean value.

5.40 Current mobile phone systems utilise RF radiation between about 800 MHz and 2 GHz, which fall in the microwave part of the spectrum. Emerging telecommunications technologies under development may use up to 60 GHz. Paging systems and two-way radios employ frequency bands down to about 150 MHz. Thus, we have focussed on experimental studies involving RF radiation between 100 MHz and 60 GHz.

5.41 *CONCLUSION We consider that, in future, researchers in this field should provide full details of the experimental conditions used, including the maximum specific absorption (SA) per pulse, for pulsed radiation.*

Biological issues

5.42 With regard to possible influences on the head, we have concentrated primarily on functional changes in the brain and consequent changes in behaviour. With regard to whole-body exposure, we have concentrated on possible effects on carcinogenic processes, on reproduction and development, on immune responses, on the cardiovascular system and on overall longevity.

Nervous system: can RF exposure cause functional changes in the brain and affect behaviour?

5.43 There is particular concern about the possible effects on the brain and behaviour of repeated, acute exposure to RF fields, largely because mobile phones are conventionally held close to the head. Recent reports in the media, for instance, have implied that the use of mobile phones can cause memory loss, changes in attention, and variation of blood pressure (see Chapter 3).

5.44 There is a vast literature on the effects of RF fields on isolated nerve cells (neurons), on cultured nervous tissue, on living brain slices, on brain function in experimental animals, on the blood–brain barrier and on behavioural measures of brain function (see, for example, UNEP/WHO/IRPA, 1993; Cleary, 1995; Hermann and Hossmann, 1997; Repacholi, 1998; D'Andrea, 1999; Jokela *et al*, 1999; Royal Society of Canada, 1999). The behaviour of animals, in particular, can be a very sensitive indicator of adverse health consequences. Early signs of potential insult are often behavioural rather than anatomical (Salzinger, 1994). Behavioural experiments on animals are used to investigate the biological basis of memory, and studies with non-human primates can serve as a model of human cognitive functions. Work on people is reviewed below (paragraphs 5.176–5.200) and here we deal specifically with *in vitro* studies and work on animals. Much of the research, especially the early studies, was conducted with high levels of RF exposure, or low average levels using high peak-power pulses (which are characteristic of radar and quite unlike emissions from telecommunications systems). Few relevant experiments have used low level fields with characteristics similar to those used in telecommunications systems (UNEP/WHO/IRPA, 1993; Repacholi, 1998; Royal Society of Canada, 1999).

Effects on cell membranes: do RF fields affect the movement of substances across membranes?

5.45 The lipid bilayer membrane that surrounds cells and the internal membranes within cells are vitally important for normal cellular function. Embedded in the external membrane are important protein molecules. Some act as receptors, detecting extracellular molecules (such as growth factors, hormones and neurotransmitters) and triggering changes in the conductivity of associated ion channels (see below) or activating signalling pathways within the cell. Other membrane proteins, called pumps, actively transport ions across the membrane, using energy derived from the energy source ATP. Yet others are channels that serve as conduits through which ions (eg sodium, potassium, chloride and calcium ions) can move across the membrane. Changes in the conductivity of ion channels, which increase or decrease the flow of ions across the membrane, cause changes in the intracellular potential of the cell. In the case of a neuron, this can affect its excitability and the amount of transmitter substance that it produces at the end of its fibre (or axon) where it makes contact with another nerve cell. Calcium acts as a signalling molecule, influencing a variety of intracellular molecular pathways, as well as having direct electrical effects within or around the cell.

5.46 Inside neurons, calcium is held in internal stores, from which it can be released into the cytoplasm. It can enter neurons from the extracellular space through selective ion channels associated with one class of neurotransmitter receptor (the NMDA-receptor). It can also move in

or out of the cell (depending on the internal concentration and electric potential) through calcium channels, the permeability of which is affected by the intracellular potential. In the terminals of nerve fibres, where they form junctions (called synapses) with other neurons, the amount of transmitter substance released when a nerve impulse arrives depends on the entry of calcium caused by the change of intracellular potential.

5.47 In neurons in certain parts of the brain (especially a structure called the hippocampus, and in the cerebral cortex, particularly in young animals), changes in the level of intracellular calcium resulting from incoming synaptic activity can lead to long-term alterations in the "strength" of synaptic inputs on to the neurons. Such long-term potentiation and long-term depression are thought to be involved in the mechanisms of memory and learning (see Kandel *et al*, 2000).

5.48 Repacholi (1998) has recently concluded from a World Health Organization review of the literature that RF fields, continuous or pulsed, can affect membrane channels, mainly at fairly high intensities, but even at levels that do not cause significant heating. There have been reports of decreased rates of channel formation, decreased frequency of channel openings, and increased rates of rapid, burst-like firing (see UNEP/WHO/IRPA, 1993). However, there is no clear understanding of how low intensity RF fields have such effects.

5.49 Cleary (1990a,b 1995) has reported, in reviews of the literature, that the flux of positively charged sodium and potassium ions across cell membranes can also be affected by RF exposure, over a wide range of frequencies (27 MHz to 10 GHz). Although most of these experiments involved very high SARs (up to 200 W/kg), he reported that effects can be produced at much lower intensities, without what he considered to be a significant rise in temperature. However, they seem to occur over a temperature "window" from 17.7 to 25˚C, which might imply that the RF energy facilitates lipid phase transitions in the membrane near the phase transition temperature (Tenforde and Liburdy, 1988). In the human body, cells are always well above this temperature window and therefore the effect is unlikely to occur *in vivo*.

5.50 RF exposure has been reported to influence the ATP-dependent sodium/potassium pump in the membranes of human red blood cells, and this effect might also perhaps be mediated by membrane phase transitions (Allis and Sinha-Robinson, 1987; Liu *et al*, 1990).

5.51 Effects on receptor proteins and their associated ion channels have also been described. For instance, Philippova *et al* (1994) found that 900 MHz radiation, at SARs of 1 and 100 W/kg, specifically affects the binding of odorant molecules to receptor protein in the membranes of olfactory receptor neurons in the rat. They attributed this to shedding of this particular protein from the membrane, probably because of increased peroxidation of membrane lipids (see Phelan *et al*, 1992). Liburdy and Vanek (1987) have also reported protein shedding from membranes as a result of RF exposure. Radiation at very low power densities can affect the ion channels associated with transmitter receptors: D'Inzeo *et al* (1988) reported a decrease in the frequency of opening of sodium channels associated with acetylcholine receptors in muscle membranes as a result of exposure to 9.75 GHz radiation at only 10–20 $\mu W/m^2$, which might cause a decrease in the excitability of the muscle.

5.52 *CONCLUSION There is evidence that RF fields can affect membrane proteins and can change the movement of ions across membranes. Some of these effects seem to occur in cells only at temperatures well below normal body temperature or with RF intensities that cause significant heating. However, some evidence suggests that RF radiation at levels produced by mobile phones might influence ion channels and other membrane proteins of neurons in the brain under normal conditions. This might cause subtle changes in cell function, but the significance of such effects*

for human health is uncertain. Moreover, these effects have not been independently confirmed, which is important given the frequent lack of reproducibility of RF biological effects.

Calcium efflux

5.53 In view of the vital role of calcium in the function of neurons and other cells, considerable work has been done on the effects of RF fields on calcium movement in brain tissue (see Adey, 1981; UNEP/IRPA/WHO, 1993; Repacholi, 1998; Jokela *et al*, 1999; Royal Society of Canada, 1999; Table 5.1).

5.54 At high intensities, 2.8 GHz radiation pulsed at 350 pulses per second (pps) – with peak SARs of several tens of thousands of watts per kilogram – can cause an increase in the incorporation of radiolabelled phosphorus into important phosphoinositide signalling molecules (Gandhi and Ross, 1989). Phosphoinositides mediate the release of calcium from internal stores, which could alter calcium-dependent processes inside the cell. In turn, this could lead to a rise in extracellular calcium because of movement through the cell membrane, which could electrically stabilise the membrane by charge-screening the surface. This would be expected to reduce the excitability of neurons.

5.55 Several studies, starting with the work of Bawin *et al* (1975), have involved measurement of the efflux of calcium out of large explants of brain tissue, prelabelled by incubation in medium containing radioactive calcium. Bawin *et al* (1975) reported that exposure to 147 MHz fields at intensities too low to cause heating increased the efflux of calcium from chick brain, but only if the field was amplitude modulated at 16 Hz. The RF carrier frequency alone had no obvious effect. This observation was confirmed by Blackman *et al* (1979, 1980a,b), who used a number of different frequencies of amplitude modulation (3–30 Hz) and found that the effect was maximal at 16 Hz. This led to the view that modulation at or near 16 Hz might be critically important and a number of other studies using this frequency of amplitude modulation have also reported increases in the diffusion of calcium out of isolated fragments of nerve cells and cultured human neuroblastoma cells (see Table 5.1). Such calcium efflux may partly reflect movement of calcium out of neurons. Indeed, Kittel *et al* (1996), using electron microscopy to identify labelled calcium in a particular part of the brain (the medial habenular nucleus), found that exposure of mice *in vivo* to 2.45 GHz RF fields, amplitude modulated at 16 Hz, caused a reduction in the number of calcium-containing vesicles inside nerve cells and an increase in the amount of calcium precipitated on the surface of the cells. However, calcium efflux from brain explants almost certainly involves a number of other factors, including the release of calcium bound or adherent to membranes and simply trapped in the interstices of the tissue. It is also likely to be influenced by temperature.

5.56 Adey (1989, 1993) has suggested that changes in calcium efflux may be due to an amplification process in which weak electric fields might be set up in the tissue at the extremely low frequency of amplitude modulation, and that these might act as a "trigger" for the initiation of long-range co-operative events within the cell membrane. However, there is no obvious theoretical basis for such effects which would seem to require the presence of a non-linear mechanism operating on the timescale of the carrier frequency. This is not the case for ion-gating mechanisms.

5.57 A number of subsequent studies in other laboratories have failed to detect an increase in calcium efflux from brain explants *in vitro* (see UNEP/WHO/IRPA, 1993), but they generally used different conditions of stimulation (see Table 5.1).

5.58 There have been only two attempts to determine if such efflux of calcium occurs *in vivo*. Adey *et al* (1982) exposed cats to 16 Hz amplitude-modulated 450 MHz fields (SAR of 0.29 W/kg) and

Table 5.1 Effects in calcium efflux on nervous tissue

Model	Exposure conditions	Result	Reference
Chick brain, *in vitro*	147 MHz CW; 0.5–35 Hz AM; 10–20 W/m^2	Increase in efflux, maximum effect at 16 Hz AM. No effect of carrier frequency alone	Bawin *et al*, 1975
Chick brain, *in vitro*	147 MHz or 50 MHz ; 3–30 Hz AM	Increase in efflux at 16 Hz AM, dependent on intensity	Blackman *et al*, 1979,1980a,b
Chick brain, *in vitro*	450 MHz; 16 Hz AM	Increase in efflux dependent on intensity	Sheppard *et al*, 1979
Human neuroblastoma cells	147 or 915 MHz CW; 16 Hz AM	Increase in efflux dependent on intensity and modulation	Dutta *et al*, 1984, 1989
Synaptosomes (isolated fragments of nerve cells)	450 MHz; 16 Hz AM	Increase in efflux	Lin-Lui and Adey, 1982
Mouse brain: medial habenula nucleus, *in vivo*	2.45 GHz; 16 Hz AM	Number of calcium-containing vesicles reduced, level of calcium precipitation on surface of neurons increased	Kittel *et al*, 1996
Cat cortex, *in vivo*	450 MHz; 16 Hz AM	Sustained increase in efflux	Adey *et al*, 1982
Rat brain, *in vitro*	1 GHz pulsed; 10 or 20 ms pulses; 16 or 32 pps for 20 min;5–150 W/m^2	No effect on efflux	Shelton and Merritt, 1981
Rat brain, loaded with radiolabelled calcium *in vivo*, exposed *in vitro*	1 GHz or 2.45 GHz pulsed; 10 ms pulses at 16 pps for 20 min; 0.29–2.9 W/kg	No effect on efflux	Merritt *et al*, 1982
Rat brain, loaded with radiolabelled calcium *in vivo*, exposed *in vivo*	2.06 GHz pulsed; 10 ms pulses at 8, 16 or 32 pps, OR 2.06 GHz CW; SAR 0.12–2.4 W/kg; 20 min	No effect on efflux	Merritt *et al*, 1982
Chick cerebral hemispheres, *in vitro*	147 MHz; 16 Hz AM; 7.5 W/m^2	No effect on efflux	Albert *et al*, 1987

AM = amplitude modulated CW = continuous wave

reported changes in calcium ion-exchange in the cerebral cortex. However, Merritt *et al* (1982) did not find such an effect in the brain of anaesthetised rats.

5.59 *CONCLUSION Although the weight of evidence suggests that RF exposure at average levels, too low to cause significant heating, does increase the release of calcium from brain tissue, there are contradictory results. The suggestion that these effects occur specifically with fields that are amplitude modulated at extremely low frequencies is intriguing but difficult to interpret. Further, this finding is of no obvious relevance to mobile phone technology, where the amplitude modulation within the critical frequency band is very small (see paragraph 4.13). If such effects occur as a result of exposure to mobile phones, their implications for cell function are unclear and no obvious health risk has been suggested. Nevertheless, as a precautionary measure, amplitude modulation around 16 Hz should be avoided, if possible, in future developments in signal coding.*

Neuronal excitability

5.60 Any tendency for calcium to move out of neurons and to accumulate on the surface of membranes would be expected to stabilise them electrically and hence to decrease the general excitability of neurons. Such effects have been described by Arber and Lin (1984, 1985), who reported an increase in membrane conductance and a decrease in the spontaneous firing of

impulses in neurons of the snail *Helix aspersa* when exposed for an hour to continuous and amplitude modulated 2.45 GHz radiation. The effects were abolished by the application of ethylenediamine tetraacetic acid (EDTA), which chelates calcium. However, they occurred at a high RF intensity and clearly depended on a rise in tissue temperature. McRee and Wachtel (1980) described a decrease in the electrical amplitude of impulses and a reduction in the excitability of the frog sciatic nerve when exposed to 2.45 GHz radiation, but only at high levels. Wachtel *et al* (1975) and Seaman and Wachtel (1978) also described a decrease in spontaneous activity of neurons isolated from the marine gastropod *Aplysia* at relatively high intensities.

5.61 On the other hand, Chou and Guy (1978) found no obvious electrophysiological changes in the frog sciatic nerve exposed to 2.45 GHz (continuous or pulsed) at modest intensities. A slight increase in conduction velocity was found at a very high level and was probably dependent on the temperature rise, since it could be mimicked by a 1˚C rise in temperature. Wang *et al* (1991), found no change in the membrane resting potential, electric capacitance, or the properties of impulses in dorsal root ganglion cells exposed *in vitro* to continuous-wave 2.45 GHz radiation under temperature-controlled conditions; recordings were made using sensitive patch-clamp techniques. Linz *et al* (1999), who also employed whole-cell patch-clamping, found no effect of low intensity continuous or pulsed RF fields on the membrane potential, action potentials or calcium and potassium currents of isolated heart muscle cells.

5.62 *CONCLUSION There is good evidence that exposure to high intensity RF fields, sufficient to cause a significant rise in tissue temperature, reduces the excitability of neurons. Exposure that does not cause an increase in temperature produces no obvious effects.*

Neurotransmitter systems

5.63 Changes in the amount of neurotransmitter substance released by nerve terminals could alter brain function. Since release is dependent on intracellular calcium levels, there has been concern that it could be affected by RF radiation.

5.64 Modak *et al* (1981) reported that RF exposure caused a decrease in the concentration of the important transmitter acetylcholine in the mouse brain, but they employed a very intense 2.45 GHz single pulse, causing a 2–4˚C rise in temperature. The rate-limiting step in the synthesis of acetylcholine is the uptake of choline by nerve cells. In an extensive series of experiments, Lai and colleagues (Lai *et al*, 1987, 1989a,b, 1990, 1991, 1994) have reported that 20 minutes of exposure of rats to pulsed 2.45 GHz radiation at low intensities causes an increase in choline uptake and a reduction in the concentration of acetylcholine receptors, whereas exposure for 45 minutes has the opposite effects (Table 5.2). These effects were found to be prevented by pretreatment of the animals with naltrexone (which blocks opioid receptors) or with corticotrophin-releasing hormone. Although the average intensities used in these studies were relatively low, the findings might depend on thermal effects, especially since acetylcholine is known to be involved in transmission in the parts of the hypothalamus responsible for temperature regulation, which is acutely sensitive to temperature change. Moreover, as discussed below, the studies by Lai *et al* used radar-like pulses of quite high peak intensity that are capable of eliciting auditory responses in animals, which themselves might have behavioural effects.

5.65 Dutta *et al* (1992) detected an increase in the activity of the enzyme acetylcholinesterase (which hydrolyses acetylcholine) in cultured human neuroblastoma cells exposed to low intensity RF fields, amplitude modulated at 16 Hz. Curiously, these effects were reported to occur over an SAR "window" – ie within a narrow range of SAR values, but not at lower or higher intensities. On the other hand, Galvin *et al* (1981) and Millar *et al* (1984), who examined the effects of continuous and pulsed 2.45 GHz RF fields on purified solutions of acetylcholinesterase, detected

Table 5.2 Effects on cholinergic systems

Model	Exposure conditions	Results	References
Rat brain	2.45 GHz pulsed; 2 µs pulses at 500 pps; 0.6 W/kg	Exposure for 45 min decreased choline uptake and concentration of acetylcholine receptors. Exposure for 20 min opposite effect seen. Effects blocked with naltrexone	Lai *et al*, 1987, 1989a,b, 1990, 1991, 1994
Human neuroblastoma cells	147 MHz; 16 Hz AM	Increase in acetylcholinesterase activity at 0.02 and 0.05 W/kg. No effect at 0.005 or 0.1 W/kg	Dutta *et al*, 1992
Solution of acetycholinesterase	2.45 GHz CW or pulsed; 16.7 ms pulses, 10–90 pps; up to 100 W/kg	No effects	Galvin *et al*, 1981; Millar *et al*, 1984
Guinea pig	3 GHz CW or pulsed; 400 pps (no pulse width specified); 35–250 W/m^2	Decrease in acetylcholinesterase activity	Baranski *et al*, 1972

AM = amplitude modulated CW = continuous wave

no influence on the activity of the enzyme, even with SARs up to 100 W/kg. The use by Dutta *et al* of 16 Hz amplitude modulation, the frequency of modulation reported to produce calcium efflux (see paragraphs 5.53–5.59) might have been critical, although it is not clear how such modulation can have a specific effect on tissues. Without a theoretical basis, the non-linear dependence of effects on intensity (the SAR window phenomenon) is very surprising: the possibility that such window effects are merely due to chance variation, or some undefined experimental artefact should be kept in mind.

5.66 In contrast to the above, Baranski *et al* (1972) reported a decrease in the activity of acetylcholinesterase in guinea pigs exposed to pulsed RF fields at high power densities, but they attributed this to thermal effects.

5.67 Attention has also focussed on the amine transmitters norepinephrine (noradrenaline) and serotonin (5-hydroxytryptamine), which may determine arousal and mood, but, more pertinently, are involved in the hypothalamic mechanisms that regulate body temperature (Brück and Hinckel, 1990). Amine transmitters are released in many parts of the brain by the terminals of axons from cell groups in the brain stem. The various changes in levels of these transmitters and their metabolites after acute and long-term exposure to RF (see, for example, Snyder, 1971; Grin, 1974; Merritt *et al*, 1977; Inaba *et al*, 1992) may well be due to short-term and adaptive responses to thermal effects, since most of the experiments involved quite intense stimulation (see Hermann and Hossmann, 1997).

5.68 *CONCLUSION Most of the work on neurotransmitter systems has used high power densities and has probably revealed thermoregulatory or other responses to temperature change. However, in view of the essential role of neurotransmitters in brain function and the involvement of specific transmitter systems in the regulation of emotion, memory, sleep, etc, this area deserves further investigation, including the assessment of these functions in human subjects.*

Electroencephalograms (EEGs) in animals

5.69 Electrophysiological experiments in animals have mainly involved the recording of "gross" potentials from the brain with electrodes placed on or within the brain. Since the work of Adey and his colleagues in the early 1970s, there has been interest in the possibility that exposure to low levels of pulsed RF alters the electrical activity of the brain in cats and rabbits (Table 5.3).

Table 5.3 Effects on EEG rhythms in animals

Model	Exposure conditions	Results	References
Cats	147 MHz; 1–25 Hz AM; up to 10 W/m^2. SAR estimated as 0.015 W/kg (WHO, 1993)	Changes in EEG conditioned rhythms	Bawin *et al*, 1973, 1974
Rats and rabbits	2.375 GHz; 7 h/day for 30 days; 0.1–5 mW/m^2	Changes in EEG	Shandala *et al*, 1979
Rats	425 MHz;100 W/m^2 AND 2.45 GHz; 50 W/m^2 ; from late gestation until 92 days old	No effects on spontaneous or evoked EEG	McRee *et al*, 1979
Rats	2.45 GHz CW; 2.7 W/kg; 7 h	No consistent changes in spectral power	Mitchell *et al*, 1989
Rats	2.45 GHz CW or AM at 16 Hz	Changes in spectral power with SARs in brain of 8.4 W/kg and above	Thuroczy *et al*, 1994

AM = amplitude modulated CW = continuous wave

5.70 Bawin *et al* (1973, 1974) exposed cats, which had been previously conditioned to produce selected EEG rhythms in response to a light flash, to low level RF fields. Changes were reported in the performance of the conditioned EEG response task and in various other behavioural parameters. It was argued that the fields acted directly on brain tissue causing a minute release of calcium (see paragraphs 5.53–5.59), resulting in changes in membrane excitability (see paragraphs 5.60–5.62), which could possibly affect EEG rhythms.

5.71 Takashima *et al* (1979) reported changes in the EEG of rabbits following exposure to a modulated RF field of 1–10 MHz, a frequency range outside the main interest of the present document. Following long-term exposure, changes in the frequency spectrum of the EEG were reported, with enhanced low frequency components and reduced higher frequency activity, similar to the changes that occur during drowsiness. Single, short-term exposures to modulated 1–30 MHz fields were without effect. The SAR could be estimated to be about 1 mW/kg and no rise in body temperature was detected during exposure. This study, however, employed few animals and there might have been artefacts due to the presence of implanted metal electrodes in the head during irradiation.

5.72 There have also been reports of subtle effects on the EEG in rats and rabbits exposed to RF fields within the frequency range of interest (Shandala *et al*, 1979; Thuroczy *et al*, 1994). However, the most consistent effects have been found at high intensities. Neither of these studies relates directly to mobile phone exposures, and neither the stimulation conditions needed or the nature of changes in the EEG are firmly established. McRee *et al* (1979) described experiments by Rosensteig of the US Environmental Protection Agency, who exposed rats to RF from late fetal life until adult. He saw no changes in either the spontaneous EEG or the electrical responses evoked by flashes of light (visual evoked responses). Mitchell *et al* (1989) reported the findings of a joint project on this subject carried out in the USA and the former Soviet Union. Both groups exposed rats to fairly intense continuous-wave RF fields for seven hours. Interestingly, both teams found small but statistically significant reductions of power in the EEG, but in different parts of the frequency spectrum.

5.73 *CONCLUSION Studies of the EEG in animals have generally not employed conditions that are directly relevant to mobile phone technology, and the results have been mixed. However, some experiments have produced evidence of non-thermal effects from RF fields on brain activity.*

Experiments on human volunteers, with exposure conditions similar to those experienced in using a mobile phone, have been more informative (see paragraphs 5.188–5.193).

Thermoregulatory behaviour

5.74 Mammals employ various physiological and behavioural mechanisms to regulate and stabilise their body temperature and thermoregulatory behaviour can be exhibited when RF exposure is sufficiently intense to generate heat. These studies have been well described by UNEP/WHO/IRPA (1993), who concluded that thermoregulatory responses from RF exposure were similar to those elicited by conventional radiant heat sources. However, the overall response depends on the distribution of RF absorption and thus on the RF frequency. At frequencies below 10 GHz, RF fields are more deeply penetrating than infrared radiation and thus less effective in stimulating superficial temperature-sensitive receptors in the skin, which are particularly involved in local and whole-body thermoregulatory responses (Adair, 1983; Adair *et al*, 1999). Thermoregulatory responses depend largely (but not exclusively) on the total heat load to the animal. Such responses can be elicited by whole-body SARs that are comparable to the basal metabolic rate (which in people is about 1 W/kg). It is therefore important to distinguish between whole-body and partial-body exposures. The latter can be of the order of 1 W/kg for mobile phones, even though the total heat added to the body is negligible.

Motor activity

5.75 Measurement of locomotion is often used to assess gross levels of brain activity and arousal. Some animals decrease their motor activity in order to lower their endogenous heat production, as part of their thermoregulatory behaviour. Acute and long-term RF exposures have been reported to reduce spontaneous motor activity in rodents. Typical studies are described by UNEP/WHO/IRPA (1993) and summarised in Table 5.4. A lifetime study reported that activity

Table 5.4 Effects on motor activity

Method	Exposure conditions	Results	References
Rats	2.45 GHz pulsed; 2.5 µs pulses at 120 pps; 6.3 W/kg; 30 min	Immediate decrease; no effect after 2 h	Hunt *et al*, 1975
Rats	2.45 GHz CW; 2.7 W/kg; 7 h	Decrease; plus less responsive to novel acoustic stimuli	Mitchell *et al*, 1988
Rats	918 MHz CW; 3.6–4.2 W/kg; 10 h/night for 3 weeks	Decrease in activity and changed time-distribution of activity	Moe *et al*, 1976
Rats	2.45 GHz CW; 0.14 W/kg and 0.7 W/kg; 7 h/day for 14 weeks	No effects at 0.14 W/kg. Decreased activity 30 days after exposure at 0.7 W/kg	D'Andrea *et al*, 1986a,b
Rats	2.45 GHz CW; 1.2 W/kg and 915 MHz CW; 2.5 W/kg; 8 h/day; 5 days/week for 16 weeks	Decrease using 2.45 GHz, but no change overnight. No effect at 915 MHz	D'Andrea *et al,* 1979, 1980
Rats	3 or 10.7 GHz CW; 185 h or 3 GHz pulsed; 1.3 µs pulses at 769 pps; 0.15–0.3 W/kg; 408 h	No effect. Other stereotypic activities also not affected	Roberti *et al,* 1975
Rats	2.45 GHz pulsed; 10 µs pulses at 800 pps; pulse-modulated at 8 Hz; 0.15–0.4 W/kg; 2–27 months	No effect except for a decrease in activity during first test session. Lifetime exposure of rats	Johnson *et al*, 1983 Guy *et al*, 1985

CW = continuous wave

levels were reduced after 6 weeks continuous exposure of young rats to 2.45 GHz pulsed RF fields at up to 0.4 W/kg. However, activity returned to control levels during subsequent exposure for up to 27 months.

Auditory responses

5.76 Animals may perceive auditory sensations when their heads are exposed to radar-like pulsed RF fields. Auditory perception of pulsed RF fields, the so-called "microwave hearing" effect, is an established phenomenon, which is produced by thermally generated sounds, due to minute thermoelastic expansion effects within the soft tissues of the head, which are conveyed to the inner ear by conduction through bone (UNEP/WHO/IRPA, 1993). The transient temperature increase and the duration of the increase (determined by the RF pulse width except for very short widths) determine whether the RF-induced acoustic vibrations can be perceived. There are good theoretical reasons to believe that microwave hearing does not occur with mobile phone signals. The acoustic sound pressure generated by a RF pulse is proportional to the rate of temperature increase. To produce audible acoustic vibrations requires a very high transient rate of heating of tissues in the head, around 1˚C per second, which is considerably higher than is produced by current pulsed mobile phone signals. Thus the effect has little relevance to health effects of mobile phone signals, but must be kept in mind when interpreting animal studies that employed radar-like pulses.

5.77 Responses to pulsed RF fields can be detected by recording from the auditory nerve or other parts of the auditory pathway in the brain (Taylor and Ashleman, 1974; Chou and Guy, 1979; Chou *et al*, 1982; Seaman and Lebowitz, 1989). These responses are in all respects identical to ordinary hearing phenomena and there is little doubt that they are perceived as sound by the animal. Chou *et al* (1985) estimated the threshold in the rat at about 1 mJ/kg for pulses briefer than 35 μs (1 mJ/kg in a pulse lasting 35 μs is equivalent to a peak SAR of 30 W/kg). It is well established that these auditory sensations produce behavioural effects in awake animals. Animals might be slightly stressed if they can hear pulsed RF fields, and this should be taken into account when considering any behavioural effects of RF exposure.

5.78 Exposure to very intense pulsed RF fields is reported to suppress the startle response and evoke body movements in conscious mice (NRPB, 1993; Sienkiewicz *et al,* 1993; UNEP/WHO/IRPA, 1993). The startle response was suppressed by 1 μs pulses with SAs of 200 mJ/kg (equivalent to a peak SAR of 200,000 W/kg), and body movement was elicited by 10 μs pulses with SAs of 2000 J/kg (peak SAR 20,000,000 W/kg) – far above the threshold for auditory perception. Although the mechanism for these effects is not well established, such intense pulses certainly elicit microwave hearing, and it is conceivable that some effects derive from the high electric fields associated with these intense pulses as well as from transient heating.

5.79 *CONCLUSION Auditory perception of intense pulsed RF fields may elicit behavioural responses. This phenomenon has not been explored using conditions that are directly relevant to mobile phone technology, but it is unlikely to occur at the peak intensities of pulsed fields associated with mobile phones.*

Learning and memory

5.80 To detect changes in learned behaviour, operant techniques are used that investigate behavioural responses, such as pressing a lever following a visual or auditory cue. Spatial memory can be tested in a radial-arm maze, in which a food pellet is placed at the end of each arm and the time is determined for the animal to collect all the food pellets. Animals must remember which arm they have entered so as to collect all the pellets in the shortest time. This technique provides a means of assessing the performance of specific learned tasks in a highly quantified and standardised

manner. However, threshold values for changes in behaviour will depend on many factors, such as the complexity of the task being performed. To quote a single threshold value for a range of tasks would be an oversimplification.

5.81 Results of earlier studies on rodents have shown that the threshold at which acute RF exposure disrupts learned operant behaviour lies between 2.5 and 8 W/kg whole-body SARs, with an associated rectal temperature rise of about 1˚C. Deficits in the performance of a previously learned behaviour occur following long-term exposure to 2.45 GHz fields at SARs as low as 2.3 W/kg whole-body exposure. The initial acquisition of operant learned tasks by rats appears to be more sensitive to disruption by RF fields, the thresholds for long-term exposure to pulsed 2.8 GHz fields being between 0.7 and 1.7 W/kg whole-body exposure (UNEP/WHO/IRPA, 1993). The pulsed fields used in many of these studies involved brief, rather intense pulses such as those produced by radar equipment, which may have elicited auditory sensations in the animals, a potential confounding factor in the interpretation of the studies.

5.82 There is a distinct difference in response between rodents and primates. Changes in operant performance responses in primates occur at higher threshold RF exposures. Such changes were detected from acute exposure of rhesus and squirrel monkeys to 1.3–5.8 GHz fields at whole-body SARs of 4–5 W/kg. Exposure of rhesus monkeys to the RF field at which they absorb the maximum amount of energy (resonant frequency, 225 MHz) resulted in reduced task performance at a whole-body SAR of 2.5 W/kg. As with rodents, these changes in performance were accompanied by a raised body temperature of about 1˚C (UNEP/WHO/IRPA, 1993). Since primates are much closer in size to people than are rodents, these data were used as the basis for standards limiting RF exposure.

5.83 Under some circumstances, ongoing learned behaviour can be stopped by exposure above a threshold corresponding to a whole-body SAR of about 4 W/kg. Many factors are known to modify this value, however, including the frequency of the applied field, the ambient temperature and relative humidity, and the animal size and species. Under the most adverse environmental conditions, changes in behaviour may be observed with whole-body SARs as low as 1 W/kg.

5.84 This interpretation is supported by the results of a study investigating working memory. Mickley *et al* (1994) found that acute exposure to 600 MHz fields at an SAR of up to 10 W/kg for 20 minutes produced significant deficits in memory in rats only when the exposure caused rises in rectal and brain temperatures of at least 1˚C. These changes were correlated with an increase in expression of the *c-fos* gene in the cortex (see paragraph 5.119). The authors concluded that the observed changes in memory and behaviour were dependent on a rise in body temperature.

5.85 However, some studies conducted on rats exposed to pulsed RF fields appear to challenge the conclusion that learning is disturbed only when radiation produces significant increases in body temperature. In experiments by Lai *et al* (1989a) on spatial learning, rats had to learn to forage for food pellets located at the ends of the arms of a radial-arm maze, using cues in the environment. Animals exposed for 20 minutes to low level, pulsed 2.45 GHz fields (average whole-body SAR of 0.6 W/kg), immediately before daily training sessions in the maze, were reported to show improved learning for the first two days, although final performance and overall accuracy were not affected. The RF fields did not cause a measurable rise in colonic temperature. However, the performance of the exposed animals was generally less stable than that of the controls, which raises questions about the significance of this result.

5.86 Later, Lai *et al* (1994) reported that rats acutely exposed to pulsed 2.45 GHz fields (whole-body SAR 0.6 W/kg), for 45 minutes each day, immediately before testing in the maze, consistently made *more* errors than did the control animals. Further work suggested that this apparent effect

on learning might be due to effects of RF exposure on nerve pathways in the brain that use endogenous opioid neurotransmitters.

5.87 In the same laboratory, Wang and Lai (2000) have also reported RF-induced changes in spatial memory as assessed in a circular water maze, in which rats had to learn to escape from the water by swimming to a submerged platform that they could not see. Male rats that had been acutely exposed for 60 minutes to pulsed 2.45 GHz fields (pulse width 2 μs, 500 pps), at a fairly high whole-body SAR of 1.2 W/kg, took longer to find the platform than did control animals during the training sessions. In a "probe trial" without the platform present, the exposed animals spent less time swimming in the quadrant of the maze that should have contained the platform than did control animals. They also spent more time trying to climb up the sidewalls of the maze, as if they had no recollection of the task. It was concluded that exposure had disrupted spatial reference memory functions and that the exposed animals had to use other, less efficient learning strategies to locate the platform. However, the energy per pulse in this study can be calculated to be 2.4 mJ/kg (peak SAR of 2400 W/kg), which would have caused rapid, transient heating. This level of pulse energy almost certainly exceeds the threshold for microwave hearing in rats (Chou *et al*, 1985; see paragraph 5.76). Since the slight stress caused by explicit noise can modify behaviour, it is possible that the results reported in some of the studies by Lai *et al* were due to microwave hearing. It is also conceivable that the electric fields associated with such a high peak SAR might have caused non-thermal effects on nerve cells. If the findings are due to the high peak-pulse energy, they are not relevant to human exposure to mobile phone radiation, which has different modulation characteristics.

5.88 For both the Lai *et al* (1994) and Wang and Lai (2000) studies, there are also statistical problems that suggest internal inconsistencies in the results. These studies involved investigation of spatial memory in either a radial-arm or water maze using a time (ie training session) x group (exposed *versus* non-exposed) design. In both studies large time effects were observed, such that the animals became more adept at the task as the training progressed. However, in neither study was there a group x time interaction, indicating that the exposure was affecting the rate at which the task was learned.

5.89 Sienkiewicz *et al* (2000), using an experimental design very similar to that of Lai *et al* (1994), exposed mice to 900 MHz RF radiation pulsed at 217 Hz at a whole-body SAR of 0.05 W/kg. The behaviour of the animals was tested each day for 10 days in an eight-arm radial maze, either immediately after exposure for 45 minutes, or after delays of 15 and 30 minutes. There were no significant differences in either the original performance of the exposed animals, the rate at which their learning increased or the final levels of performance. However, the animals tested immediately after exposure took longer to complete the task and exhibited a more erratic performance than the other animals. It is possible that these changes may have been induced by a mild stress associated with auditory perception of the field. This experiment was not an exact duplication of the Lai *et al* (1994) study, since it used mice, rather than rats, an eight-arm instead of a twelve-arm maze, and a much lower SAR.

5.90 Overall, these and other studies (see D'Andrea, 1999) provide weak evidence for a specific effect of RF fields on spatial memory, and some artefact associated with exposure may have affected the performance of the rats in those experiments in which effects were found. In particular, it is possible that the animals may have perceived the pulsed RF fields and this may have contributed to the observed behavioural changes. A summary of the results of earlier studies is given by UNEP/WHO/IRPA (1993) and more information about recent work is given in Table 5.5.

Table 5.5 Studies on learning

Model	Exposure conditions	Results	References
Rats, 12-arm maze	2.45 GHz pulsed; 2 µs pulses; 500 pps; 0.6 W/kg; 20 min/day for 10 days	Fewer errors for two days, no effect overall	Lai *et al*, 1989a
Rats, 12-arm maze	2.45 GHz pulsed; 2 µs pulses; 500 pps; 0.6 W/kg; 45 min/day for 10 days	More errors each day	Lai *et al*, 1989a, 1994
Rats, Water maze	2.45 GHz pulsed; 2 µs pulses; 500 pps; 1.2 W/kg; 60 min/session; 2 sessions/day for 3 days	Took longer to locate submerged platform	Wang and Lai, 2000
Mice, 8-arm maze	900 MHz pulsed; 576 µs pulses; 217 pps; 0.05 W/kg; 45 min/day for 10 days	No effect	Sienkiewicz *et al*, 2000
Operant tasks: rats	2.8 GHz pulsed; 2 µs pulses; 500 pps; for 30 min	Impaired acquisition threshold at 1.7 W/kg whole-body exposure	Schrot *et al*, 1980
Operant task: rats	360, 480, 500, 600 MHz CW; up to 25 min or 55 min	Threshold for reduced performance > 4–6 W/kg whole-body exposure	D'Andrea *et al*, 1976, 1977
Operant task: rats	2.45 GHz CW; 110 5 h sessions over 22 weeks	Impaired performance at 2.3 W/kg whole-body exposure	Mitchell *et al*, 1977
Operant task: rats	2.45 GHz CW; 60 min	Threshold for reduced performance > 2.5–8 W/kg whole-body exposure	Sanza and de Lorge (1977); de Lorge and Ezell (1980)
Operant task: rhesus monkey	1.2 GHz CW; SAR 0.8 or 1.6 W/kg; 120 min	No effect	Scholl and Allen, 1979
Operant task: rhesus monkey	225 MHz CW OR 1.3 GHz pulsed; 3 µs pulses; 370 pps OR 5.8 GHz pulsed; 0.5 or 2 µs pulses; 662 pps	Threshold for impaired performance 2.5 W/kg (at 225 MHz) or 4–5 W/kg (at 1.3 and 5.8 GHz) whole-body exposure	De Lorge, 1984
Working memory task: rats	600 MHz (CW); SAR 0.1–10 W/kg; 20 min	Impaired performance at 1 ˚C rise in body and brain temperature (>9 W/kg) whole-body exposure	Mickley *et al*, 1994

CW = continuous wave

5.91 The hippocampus (a forebrain structure, buried on the inside of the temporal lobe) has been implicated in spatial learning in many animals, and in the laying-down of "episodic", personal memories in people. Damage to the hippocampus interferes with these forms of learning and certain synaptic connections between neurons in the hippocampus are capable of rapid and long-lasting changes in transmission efficiency, which might constitute the cellular basis of the memory trace. The hippocampal slice preparation, in which the activity of neurons and the efficiency of synaptic transmission can be directly measured, *in vitro*, is widely used to investigate these cellular mechanisms (Kandel *et al*, 1991). Wood *et al* (2000) have recently used this technique to examine the effects of RF fields on electrical activity in the hippocampus. Short-term exposure to very low intensity 700 MHz radiation in the absence of any detectable increase in temperature resulted in transient changes in evoked and spontaneous activity. Curiously, the changes were very variable, but exposure at about 0.001 W/kg generally led to a decrease in activity. It is difficult to interpret these results, especially the variability of the effects,

but if they were to occur *in vivo*, they might influence learning and memory. It must be emphasised, however, that the human hippocampus lies deep in the human brain, where very little energy is absorbed from a mobile phone.

5.92 *CONCLUSION Increases in core temperature of 1˚C or more certainly lead to changes in the performance of well-learned tasks and other simple behaviours. However, there is no consistent experimental evidence that exposure to low level RF fields affects learning and memory in animals. The studies of Lai and co-workers challenge these conclusions and suggest that spatial learning can be disturbed at average SARs below 1 W/kg. However the peak-pulse energy was much higher than that associated with mobile phones, the effects reported were statistically weak and they have not been reproduced by Sienkiewicz et al (2000) using 900 MHz fields. D'Andrea (1999) has speculated that some cognitive tasks may show particular sensitivity to RF exposure, and effects on these behaviours may occur at SARs below those required to disrupt simple, well-learned tasks. Few studies have yet explored this possibility. The hippocampal slice preparation shows great potential for the study of RF field effects: more research is indicated. However, studies on human subjects are needed to assess whether fields associated with mobile phones have any effect on memory or learning.*

Blood–brain barrier

5.93 Early work suggested that the blood–brain barrier, which normally prevents large molecules from crossing into the cerebrospinal fluid from the blood, might be susceptible to low level pulsed RF fields. Effects on permeability of the barrier have been investigated by comparing the penetration into the brain in exposed and control animals after intravenous injection of various compounds. Interest began when Frey *et al* (1975) reported increased penetration of the blood–brain barrier of anaesthetised rats after acute low level exposure to pulsed or continuous-wave 1.2 GHz fields. Oscar and Hawkins (1977) then reported that the acute exposure of anaesthetised rats to pulsed 1.3 GHz fields at similar SARs increased the uptake of radiolabelled saccharides. However, later, more rigorous studies indicated that the early studies might have been confounded by various factors including alteration in cerebral blood flow, the effect of the anaesthetic, and changes in renal clearance (Blackwell and Saunders, 1986; UNEP/WHO/IRPA, 1993).

5.94 Two studies (Neubauer *et al*, 1990; Salford *et al*, 1994) have reported increased blood–brain barrier permeability to protein (albumin) following RF exposure at SARs as low as 0.016 W/kg. Later studies (Fritze *et al*, 1997a; Nagawa and Uneo, 1999) have, however, failed to confirm these results.

5.95 *CONCLUSION The available evidence for an effect of RF exposure on the blood–brain barrier is inconsistent and contradictory. Recent, well-conducted studies have not reported any effects.*

Studies of melatonin

5.96 Melatonin is a hormone secreted by the pineal gland, which controls our diurnal rhythm (day–night cycle). Peak levels are produced in people during the night (in the dark period). Melatonin affects the mammalian reproductive system, as well as other physiological and biochemical functions (Reiter, 1991). The function of the pineal gland is strongly influenced by visible radiation, because signals from the optic nerve affect the suprachiasmatic nucleus in the hypothalamus, which in turn regulates the secretion of melatonin from the pineal gland. The cyclical pattern of light and dark imposes seasonal as well as circadian rhythms in some mammals (Reiter, 1993). Melatonin is an efficient scavenger of free radicals, which can damage cells, and there is evidence that melatonin has a protective effect against cancer. Thus, changes in melatonin secretion could conceivably alter tumour initiation and promotion (NRC, 1997; NIEHS, 1998).

5.97 There are reports that extremely low frequency (ELF) electromagnetic fields may affect pineal function, although the data are inconsistent. This has led to the "melatonin hypothesis", suggesting a link between ELF fields and cancer (Stevens, 1987). This raises the question whether exposure to RF fields might also have an effect on the pineal gland. Radiofrequency photon energies are much higher than those at ELFs, lying between the ELF and visible parts of the electromagnetic spectrum. In contrast to visible radiation, neither ELF fields nor RF fields directly affect photopigments in photoreceptors in the eye, and they are therefore very unlikely to affect pineal function by the same anatomical pathway as does visible radiation. It is conceivable that RF fields might influence the synthesis or secretion of melatonin by the pineal gland through a direct influence on either the suprachiasmatic nucleus or the pineal gland itself, although there is no obvious theoretical reason to expect such influences.

5.98 Only a few studies testing effects of RF exposure on melatonin synthesis have been conducted. Stark *et al* (1997) studied dairy cattle herds located in the vicinity of a short-wave (3–30 MHz) radio antenna. Their data showed no chronic effect on salivary melatonin levels, although a short-term rise in melatonin was noted when the antenna was energised after being turned off for three days. In a laboratory study specifically designed to study pineal function of rats and hamsters exposed to very low level 900 MHz fields for up to six hours, no effects on nocturnal melatonin production were found (Vollrath *et al*, 1997).

5.99 *CONCLUSION Although few studies have been conducted, they do not suggest that exposure to RF fields affects pineal function or melatonin production. Relevance to the use of mobile phones could, in any case, be assessed only through laboratory studies of people because of species differences in the pattern of circadian rhythms. It must also be emphasised that the hypothalamus and pineal gland are much further from the surface of the head in people than in animals. Therefore, even if there were an effect on melatonin production in animals resulting from a direct interaction of fields within the brain, it would be much less likely to occur in people.*

Effects on the eye

5.100 The lens of the eye is potentially sensitive to RF exposure because it lacks a blood supply and therefore has reduced ability to dissipate heat. Further, the fibres that make up the bulk of the lens have only a limited capacity for repair and hence the effects of minor insults tend to accumulate, resulting in clouding of the lens (cataract).

5.101 Many studies conducted to determine the threshold for RF-induced cataracts have concluded that very high exposures are needed for at least 1 hour to produce lens cataracts (UNEP/WHO/IRPA, 1993). The single acute exposure threshold to produce a cataract at 2.45 GHz corresponds to an SAR in excess of 100 W/kg for more than 1 hour, with the temperatures in the eye exceeding 43 ˚C. Repeated subthreshold exposures at levels just below this threshold would finally produce a cataract, provided that the accumulated damage could not be repaired before the next exposure (Carpenter, 1979). Near-continuous, long-term RF exposures at moderate intensity (100 W/m^2; peak SAR in the head 17 W/kg) did not produce cataracts in rabbit eyes (Guy *et al*, 1980).

5.102 Degenerative changes have been reported in various eye tissues of primates after exposure to pulsed RF fields (Table 5.6). Studies in this area have been summarised by Kues and Monahan (1992a) and Lu *et al* (2000). Localised exposure of the eyes of anaesthetised monkeys to pulsed 2.45 GHz fields at an SAR in the eye of 2.6 W/kg for several hours resulted in lesions in the corneal endothelium (Kues *et al*, 1985) and increased the vascular leakage from the blood vessels of the iris. Lesions in the cornea were also induced by exposure to 2.45 GHz fields, with pulsed fields being more effective than continuous-wave radiation. Topical pretreatment with the ophthalmic drug timolol maleate (used for treatment of glaucoma) appeared to reduce the

threshold for these effects to 0.26 W/kg (Kues *et al*, 1992). Intermittent exposure over a 10-week period resulted in early degenerative changes in the retina, which were also exacerbated by application of timolol maleate. In contrast, Kamimura *et al* (1994) reported that they were unable to induce corneal, lenticular or retinal lesions in the eyes of non-anaesthetised macaque monkeys exposed to continuous-wave (but not pulsed) 2.45 GHz radiation at levels exceeding the threshold for continuous-wave-induced corneal damage described by Kues *et al* (1985).

Table 5.6 Effects of RF fields on the eye

Model	Exposure conditions	Results	References
Rabbits	2.45 GHz; 100 W/m^2 ; 17 W/kg; 23 h/day for 180 days	No cataracts produced	Carpenter, 1979
Anaesthetised monkeys	2.45 GHz pulsed; 10 µs pulses at 100 pps; 26 mJ/kg per pulse; average SAR 1.3–3.9 W/kg; 3 exposures of 4 h	Lesions in corneal endothelium, increased vascular leakage in iris	Kues *et al* 1985, 1992; Kues and Monahan, 1992a
Rabbits and macaque monkeys	60 GHz; 100 W/m^2; 8 h, plus 4 h/day for 5 days	No effects on the eye	Kues *et al*, 1999
Non-anaesthetised monkeys	2.45 GHz CW	No effects	Kamimura *et al*, 1994
Monkeys	1.25 GHz pulsed; 5.59 µs pulses; 0.59, 1.18 and 2.79 pps; peak SAR 1.3 MW/kg; average SAR in retina 4.3, 8.4 or 20.2 W/kg; 4 h/day, 3 days/week for 3 weeks	No effects on retinal structure. Electric responses of retinal cells slightly increased at higher SARs	Lu *et al*, 2000
Non-anaesthetised monkeys	1.25 GHz pulsed; 0.5 µs pulses; 16 pps; 0.4 W/kg OR 2.7 GHz; 1 µs pulses; 20 pps; 2.6 W/kg	Transient changes in electrical activity induced by repeated exposure	Kues and Monahan, 1992b
Monkeys	5.6 GHz pulsed; 2.3 µs pulses at 100 pps; 1 W/kg	No effect on visual function	D'Andrea *et al*, 1992

CW = continuous wave

5.103 Transient changes in the electrical activity of the retina of the eyes of monkeys, in response to light stimulation, have been reported following repeated exposures to pulsed 1.25 or 2.7 GHz fields (Kues and Monahan, 1992b). Changes in electrical responses were attributed to field-induced degeneration of the photoreceptors, particularly of the cones. However, D'Andrea *et al* (1992) reported a lack of effect on visual function of monkeys exposed to pulsed 5.6 GHz RF radiation. This disparity seems puzzling in view of the fact that the average SAR was quite similar in the two studies. However, it is possible that the difference between these two studies is explained by differences in the peak SAR per pulse in the pulsed radiation. Taking into account pulse duration and frequency, peak SARs in the study of Kues and Monahan (1992b) can be calculated to be 50,000 and 130,000 W/kg, while that in the work by D'Andrea *et al* (1992) was 4,000 W/kg. This highlights the crucial importance of specifying the energy per pulse in research using pulsed radiation. Very recently, Lu *et al* (2000) have described a slight enhancement of electrical responses from the retina in monkeys exposed to 1.25 GHz RF fields pulsed at low rates with intense pulses (peak retinal SAR 1,300,000 W/kg), but without any obvious change in retinal structure.

5.104 *CONCLUSION The intensities of pulsed RF fields employed in these studies were well above the SAR and specific absorption that could occur in the eye from the use of current mobile phones. However, the studies do raise important concerns about possible adverse health effects in the eye from high peak-power, pulsed RF fields.*

Overall conclusions of effects on the nervous system

5.105 *The potential of RF fields to affect the nervous system has been addressed using a variety of model systems. The most consistent evidence indicates that changes in neuronal excitability, neurotransmitter function, and innate and learned behaviours will occur when exposure induces significant heating, such that core body or local tissue temperatures increase by about 1˚C or more. The evidence for effects in the absence of heating is generally not consistent and convincing. However, some studies suggest that low level exposure at specific frequencies of amplitude modulation and energy levels may affect membrane proteins, the flux of calcium and other ions across the membranes of neurons, and EEG rhythms. The relevance of these results to mobile phone technology and to human health is unclear.*

5.106 *Despite much publicity, the evidence for an effect on spatial memory in rats in the absence of whole-body heating is weak. In addition, there are differences in the pattern of RF energy deposition between rodents and people. This makes direct extrapolation from these animal studies to changes in human cognitive performance uncertain. The tissue penetration of RF fields means that, while the intensity of exposure is fairly uniform within the small brain of a rodent, only regions close to the ear will be effectively exposed in the much larger human brain. Primate brains, however, not only have greater anatomical similarity to those of people, but also have similar proportions, resulting in a better model of the distribution of absorbed energy.*

Cancer-related studies: can RF exposure affect carcinogenic processes?

5.107 The DNA in our chromosomes, which controls the growth and function of our cells, is normally remarkably stable: indeed, there are a variety of mechanisms for protecting DNA and repairing damage. Certain substances and other agents (eg X-rays) that cause damage to DNA are called *genotoxic* or *mutagenic*. Genotoxic injury of a cell can reveal itself in various ways, particularly as abnormalities in the appearance of the chromosomes, shrinkage of the cell nucleus, and mutation.

5.108 Genotoxic injury occurs constantly in our bodies, partly because we are exposed to a variety of natural and artificial mutagens, and partly because it can occur spontaneously through random errors in the replication of DNA during cell division. Most damage is repaired. If it is substantial, the cell can die. However, certain sequences of modest genetic damage can result in mutations that push a cell in a number of steps towards the cancerous state (conventionally described as *initiation*, *promotion* and *progression*). The cells eventually proliferate through rapid cell division (Santini *et al*, 1988; Cohen and Ellwein, 1991; Wu *et al*, 1994).

5.109 It is now widely agreed that cancer is initiated by alterations in the genetic material (DNA) in the cell (genotoxic effects), although some non-genotoxic chemicals and processes (called epigenetic carcinogens) have been recognised. After initiation, the cell may progress to full malignancy without any further external stimulus but more often further events are required, which may be further genomic alterations or other cellular events such as a stimulus to divide or the absence of signals required for cell differentiation. An agent which will cause this further progression towards malignancy is often termed a promoting agent.

5.110 Studies of possible genotoxic effects of RF radiation, enhanced cell proliferation and inappropriate gene expression have been carried out at the cellular level. In addition, there have

been a number of long-term studies of cancer induction in animals, including tests of epigenetic interaction with known carcinogens.

Ornithine decarboxylase: does RF exposure, *in vitro* or *in vivo*, affect catalytic action?

5.111 Protein kinases, such as ornithine decarboxylase (ODC), are key enzymes that are normally activated as a result of the action of hormones, growth factors and lymphokines on receptors in cell membranes. ODC is the rate-limiting enzyme in the synthesis of substances called polyamines, which can trigger DNA synthesis, cell growth and cell differentiation. Inhibition of ODC activity retards the growth of both normal cells and tumour cells (Marton and Pegg, 1995). ODC activity is modulated by membrane-mediated signalling events, and its activation is associated with the activity of mitogens (substances that cause mutation) and tumour-promoting agents of various types, such as the phorbol ester TPA, during carcinogenesis. Activation of ODC has been related to the late, "promotional" phase of cancer production, which is usually (but not always) correlated with proliferation (an increase in the rate of cell division) in the affected tissue. Most chemical tumour promoters increase the level of ODC in cells (through stimulating expression of the gene that produces ODC). They also increase the activity or enzymatic effectiveness of ODC, leading to an accumulation of polyamines. It is important to note that although all carcinogenic factors stimulate ODC, not all stimuli that increase ODC activity promote cancer.

5.112 A report of an Expert Panel of the Royal Society of Canada (1999) has recently reviewed investigations of the effects of electromagnetic radiation on the level and activity of ODC (see Table 5.7). Various cell lines were exposed to RF radiation, including mobile phone radiation, amplitude modulated at frequencies in the ELF range. In general, the studies reported modest increases in ODC activity only at modulation frequencies of about 10–60 Hz. DNA synthesis, which would indicate a proliferative response to raised ODC activity, was not subsequently increased.

Table 5.7 Ornithine decarboxylase (ODC) activity *in vitro*

Model	Exposure conditions	Results	References
Reuber H35 hepatoma; Chinese hamster ovary; 294T human melanoma cells	450 MHz; AM at 5, 10, 16, 20, 60 and 100 Hz; 10 W/m^2; SAR estimated as 0.08 W/kg; 1 h	Increased ODC activity by up to 100% at 12–20 Hz modulation	Byus *et al*, 1988 Byus and Hawel, 1997
L929 mouse fibroblasts	915 MHz; AM at 50, 60 or 65 Hz; SAR estimated as 2.5 W/kg; up to 8 h	Increased ODC activity by up to 100%	Litovitz *et al*, 1993
L929 mouse fibroblasts	835 MHz; AM at 6, 16, 55, 60, 65, or 600 Hz; SAR of 2.5 W/kg	Increased ODC activity by up to 100% at 16–65 Hz modulation	Penafiel *et al*, 1997

AM = amplitude modulated

5.113 Many studies (Royal Society of Canada, 1999) have shown that conventional 50–60 Hz electromagnetic fields (without an RF carrier frequency) can produce a similar increase in ODC activity. Hence, the effect of amplitude-modulated RF fields might be due to the ELF currents within the tissue, although it is difficult to understand how such currents could be generated (see paragraph 5.16). However, the maximum increase in ODC activity produced by amplitude-modulated RF radiation (approximately a doubling) is much less than that elicited by known tumour-promoting substances, which can cause up to 500-fold changes in ODC activity in relevant tissues.

5.114 Although most cancer-producing conditions lead to large rises in the expression of ODC and proliferation of cells (which do not occur with even amplitude-modulated RF fields), promotional changes have been reported despite relatively small rises in ODC activity, and sometimes without cell proliferation (see, for example, Hibshoosh *et al*, 1991; Moshier *et al*, 1993, 1994; Kubota *et al*, 1997).

5.115 *CONCLUSION Pulse-modulated RF fields from mobile phones may cause a slight increase in ODC levels and activity, at non-thermal levels. However, it is very unlikely that these small changes could, on their own, have a tumour-promoting effect. It is also unlikely that such effects act synergistically with other environmental hazards and contribute to tumour promotion.*

Gene expression: does RF exposure initiate changes in the action of genes?

5.116 All the somatic cells (not the eggs or sperm) in the human body contain the same set of genes in their chromosomes. Each gene contains the information to make a particular protein (eg an enzyme or a structural protein). The differences in appearance and function of different types of cells (eg skin cells, liver cells and neurons) are caused by different sets of genes being active in each cell. The activation of genes is known as gene expression. The production of a protein involves two main steps: *transcription* (the synthesis of RNA, which contains the same information as that in the sequence of DNA in the gene) and *translation* (the building of the protein molecule from amino acids, under the control of the RNA). Changes in the characteristics of cells (differentiation), cell growth and programmed cell death can all occur as a result of the modulation of gene expression, which can be initiated by external events, acting through intracellular signalling pathways.

5.117 Certain genes are switched on specifically in response to stressful challenges to the cell. For instance, a set of genes producing *heat shock proteins,* which protect other proteins from damage, are activated by a sudden increase in temperature, and also by other forms of shock, such as toxic challenge. Further, as part of the response to stress, intense sensory stimulation, mitogenic chemicals, etc, other genes called *immediate early genes*, such as *c-fos* and *c-jun*, are turned on. Their proteins activate protective signalling pathways in the cell. The expression of these various genes is, then, a sensitive early marker of the cellular response to stress. The expression of genes called *proto-oncogenes* can be increased by tumour-promoting agents, ultraviolet radiation and X-rays. These genes, including *c-ras*, *c-myc* and *c-abl*, have normal cellular functions but can also contribute to the initiation of cancerous changes.

5.118 Recently, de Pomerai and colleagues have developed an invertebrate model to examine stress-induced gene expression. They used a transgenic soil nematode, *C elegans*, carrying a reporter gene under the control of the genetic mechanism for activation of heat shock genes. Hence the reporter (an enzyme or fluorescent protein that can easily be detected in the worm) is produced whenever the heat shock gene is turned on. Danniells *et al* (1998) and Power *et al* (1998) have shown that exposure of these worms to 750 MHz radiation (continuous or pulsed) for a few hours (0.5 W power), without detectable elevation of temperature, results in elevated expression of heat shock protein genes. Similar results were described after exposure for seven hours to the emission of a conventional digital mobile phone (de Pomerai *et al,* 1999).

5.119 By comparison, results of studies of gene expression in mammals have been variable and generally rather negative (Table 5.8). Studies of changes in the expression of the early response genes *c-fos* and *c-jun* (Mickley *et al*, 1994; Walters *et al*, 1995; Fritze *et al*, 1997b; Morrissey *et al*, 1999) in the brains of rats and mice exposed to RF radiation generally find no effects following exposure at thermally insignificant levels. In any case, *c-fos* expression is known to increase simply as a result of mild stress, such as that associated with immobilisation of an

Table 5.8 Recent studies on gene expression

Model	Exposure conditions	Results	References
In vitro studies			
PC12 rat pheochromocytoma cells	836.55 MHz; TDMA-modulated; 0.5–5 mW/kg; up to 60 min	Expression of *c-jun* elevated at highest exposure level only; no effect on *c-fos*	Ivaschuk *et al*, 1997
Mouse C3H 10 T ½ embryonic fibroblasts	835.62 MHz; FM and 847.74 MHz; CDMA-modulated; 0.6 W/kg; up to 4 days	Slight increase in *c-fos* expression; no effect on *c-jun* or *c-myc*	Goswami *et al*, 1999
In vivo studies			
Rat brain	600 MHz; whole-body SAR of 9.3 W/kg; 2 h	Increase in *c-fos* protein levels; brain surface temperature increased by 2 ˚C	Mickley *et al*, 1994
Rat brain	0.25–2.5 GHz; high peak power ultra-wide band radiation; 7–8 ns pulses; 60 pps for 2 min; peak *E*-field of 250 kV/m	No effect on *c-fos* protein levels; no heating effect	Walters *et al*, 1995
Mouse brain	1.6 GHz CW or pulsed with 9.2 ms pulses at 11 Hz	Increased *c-fos* expression in stress-responsive and thermoregulatory regions of brain at an SAR of 2.99 W/kg and above	Morrisey *et al*, 1999
Rat brain	900 MHz pulsed (GSM); brain SAR 0.3 or 1.5 W/kg OR 7.5 W/kg CW for 4 h	Increased *c-fos* only at highest SAR; no effect on *hsp70* or *c-jun*	Fritze *et al*, 1997b

CW = continuous wave FM = frequency modulated TDMA = time division multiple access CDMA = code division multiple access

animal (Cullinan *et al*, 1995), and this must be kept in mind when interpreting the results of experiments on awake animals. *In vitro* studies of mammalian cells have produced mixed results (Ivaschuk *et al*, 1997; Goswami *et al*, 1999).

5.120 *CONCLUSION While there is currently little evidence that exposure to mobile phone radiation causes a stress response in mammalian cells, judged by elevated gene expression, the results on nematode worms are indicative of a non-thermal influence on gene expression. This model and similar model systems, using cultured mammalian cells carrying reporter transgenes linked to important genes, could be valuable in defining genetic responses to RF radiation.*

Does RF radiation affect cell growth, survival or proliferation?

5.121 Changes in the kinetics of cell division and in the proliferation of cells play a crucial role in the generation of cancer. Any increase in cell proliferation resulting from RF radiation might indicate a carcinogenic influence. Several studies of the possible effects of exposure to RF radiation are described in Table 5.9.

5.122 Grundler *et al* (1992), pursuing Fröhlich's suggestion (see, for example, Fröhlich, 1986) that electromagnetic radiation in the microwave range could interact with some sort of resonant process in undefined molecules in living biosystems, studied the influence of very low power RF radiation at 41–42 GHz on the cell cycle and growth rate of yeast cells. Even at extremely low SARs, they found that such radiation could cause small, but reliable changes in growth rates (increases of less than 10% and decreases of almost 20%), with sharp resonant peaks, dependent on frequency. They interpreted these results in terms of some internal "oscillator" in the cells,

capable of coupling with these extremely weak fields to modulate growth rate. These results were not confirmed in several further studies (see paragraph 5.19).

Table 5.9 Recent studies of cell growth, survival and proliferation in the presence of RF fields

Cell line	Exposure conditions	Results	References
Yeast cells	41.650–41.798 GHz; 20 mW; forward power of 20 mW for 4 h	Absence of frequency-dependent effects on growth	Furia *et al*, 1986
Yeast cells	41–42 GHz	Small changes in growth rates, which were frequency dependent	Grundler *et al*, 1992
Yeast cells	41.682–41.710 GHz; 0.5 or 50 $\mu W/m^2$; up to 1 W/kg); 5.5 h	Absence of frequency-dependent effects on growth	Gos *et al*, 1997
LN71 glioma cells; human lymphocytes	27 MHz or 2.45 GHz; up to 75 W/kg; 2 h; isothermal conditions	Increased incorporation of radiolabelled nucleic acid at SARs of 50 W/kg or less	Cleary, 1995
C6 glioma and primary glial cells	836.55 MHz; TDMA-modulated; 0.6–60 mW/kg; 24 h	Increased uptake of radiolabelled nucleic acid precursors in glioma cells at 6 mW/kg	Stagg *et al*, 1997
Human epithelial amnion cells	960 MHz pulsed (GSM); 217 pps; 8.3 pps; 24 h	Decrease in cell growth	Kwee and Raskmark, 1998

TDMA = time division multiple access GSM = global system for mobile telecommunications

5.123 In general, other studies report modest increases in proliferation (see Cleary, 1990a,b; 1995), no effect except at only one low (5.9 mW/kg) SAR level (Stagg *et al*, 1997), or a decrease in cell proliferation (Kwee and Raskmark, 1998). Only the latter two studies investigated possible effects of exposure to mobile phone radiation.

5.124 *CONCLUSION Taken together, these and other experiments on DNA synthesis do not demonstrate convincing, consistent changes in cell proliferation under conditions that mimic emissions from mobile phones or base stations. However, in view of the work by Stagg et al (1997), the effects of RF fields on nucleic acid synthesis deserve further study.*

Genotoxicity: does RF exposure cause DNA damage, mutation or chromosomal aberrations?

5.125 Studies of the genotoxic actions of carcinogenic substances and ionising radiation on cells and experimental animals have been of value in supplementing epidemiological evidence about human disease. For instance, many experiments on the effects of tobacco tars on cells and animals have strengthened the hypothesis that smoking causes cancer.

5.126 Many studies of potential genotoxicity have been carried out, involving the exposure of molecules, cells, isolated explants of tissue and whole animals to RF radiation in and around the frequency band used for mobile telecommunications (recently reviewed by Verschaeve, 1995; Brusick *et al*, 1998; Verschaeve and Maes, 1998; Jokela *et al*, 1999; Moulder *et al*, 1999; Royal Society of Canada, 1999). Some studies, especially the early ones, are difficult to interpret either because the exposure was very intense or because its exact characteristics and physical effects (local electric field, intensity, SAR, etc) were not fully recorded or computed. In particular, the thermal effects of higher intensity stimulation complicate interpretation, since heating alone can

be genotoxic (Asanami and Shimono, 1999) and can enhance the action of known genotoxic agents (Jorritsma and Konings, 1984; Miura *et al*, 1986).

Mutagenesis

5.127 Experiments on bacteria have shown increased mutation rates, but in general the intensities used in these positive studies were high enough to cause significant heating (see, for example, Averbeck *et al*, 1976; Dutta *et al*, 1979; Blevins *et al*, 1980; Anderstam *et al*, 1983). In contrast, many researchers have reported that low power RF radiation produces no change in the rate of mutation of microbes or mouse lymphoma cells (see, for example, Blackman *et al*, 1976; Dardalhon *et al*, 1981; Hamnerius *et al*, 1985; Phillips *et al*, 1999: Royal Society of Canada, 1999).

5.128 A large number of studies on whole animals, from fruit flies to rats, have consistently failed to demonstrate mutation of either somatic cells or sperm after exposure to RF radiation, even with power densities up to 1000 W/m^2 and SARs up to 110 W/kg (Royal Society of Canada, 1999). However, Varma *et al* (1976), Varma and Traboulay (1976) and Goud *et al* (1982) reported increased dominant lethal mutations in the offspring of exposed male mice and abnormal sperm (see Table 5.10). The intensities used were very high and the effects were almost certainly due to elevated temperature. Studies carried out at lower intensities found no effect.

Table 5.10 Effects of RF fields on mutation, as indicated by dominant lethal mutations in male rodents

Model	Exposure conditions	Results	References
Swiss mice	2.45 GHz; 500 W/m^2; 30 min or 100 W/m^2; 80 min	Increased dominant lethal frequency	Varma *et al*, 1976
Swiss mice	1.7 GHz; 500 W/m^2; 30 min or 100 W/m^2; 80 min	Increased dominant lethal frequency	Varma and Traboulay, 1976
Sprague-Dawley rats	2.45 GHz; either prenatally and postnatally or as young adults; 50 W/m^2; about 105 days; 100 W/m^2; 5 days; 280 W/m^2; 4 weeks	No increase in dominant lethal mutations	Berman *et al*, 1980
Swiss mice	2.45 GHz; 1.7 kW/m^2; 70 s	Increased dominant lethal frequency	Goud *et al*, 1982
C3H mice	2.45 GHz; 43 W/kg to lower half of body;30 min	No increase in dominant lethal mutations	Saunders *et al*, 1983
C3H mice	2.45 GHz; 100 W/m^2; whole-body SAR 4 W/kg; 6 h/day over 8 weeks	No increase in dominant lethal mutations	Saunders *et al*, 1988

5.129 *CONCLUSION The balance of evidence suggests that at normal temperatures (consistent with exposures below guidelines), RF fields do not induce mutation of either somatic cells or germ cells.*

DNA damage: *in vitro* studies

5.130 Studies on bacteria, plant and animal cells exposed *in vitro*, where thermal effects can be directly observed and/or controlled, have failed to reveal direct evidence of DNA damage or repair, even at power densities up to 100 W/m^2 and SARs up to 20 W/kg (see, for example, Dutta *et al*, 1979; Meltz *et al*, 1987, 1990; Phillips *et al*, 1998; Verschaeve and Maes, 1998; Royal Society of Canada, 1999; Vasquez *et al*, 1999). The studies of Malyapa *et al* (1997a,b) on human cells are

perhaps the most relevant because they were modelled on experiments by Lai and Singh (1995, 1996) who suggested that exposure of rats *in vivo* to pulsed RF fields produces DNA strand breaks in cells of the brain. In one experiment, Malyapa *et al* (1997a) employed 2.45 GHz continuous-wave RF radiation. In the other (Malyapa *et al*, 1997b), they used 836 MHz frequency-modulated radiation or 848 MHz radiation with CDMA modulation, simulating the transmission characteristics of mobile phones in the USA. In neither case did they find DNA strand breaks.

DNA damage: *in vivo* studies

5.131 Studies of DNA damage *in vivo* are summarised in Table 5.11 (see also Verschaeve *et al*, 1998). In early work, Varma and Traboulay (1976, 1977) reported DNA damage in mice but the intensities used were high enough to cause thermal effects. More recently, three further studies in rodents have suggested that RF fields at lower intensities may affect DNA directly (Sarkar *et al*, 1994; Lai and Singh, 1995, 1996), although the data are, at best, preliminary. Evidence suggesting that the exposure of mice to 2.45 GHz radiation resulted in large-scale structural rearrangement of DNA in cells in the brain and testes was reported by Sarkar *et al* (1994). In this study, DNA was isolated from the brain and testes after exposure and cut into small fragments using a restriction enzyme. The fragments were separated according to size by electrophoresis, and probed for DNA sequences with a simple probe. The appearance or disappearance of bands could be indicative of some form of structural genomic rearrangement, but this technique is very susceptible to variable DNA digestion.

Table 5.11 Effects of RF fields on DNA damage and repair *in vivo*

Model	Exposure conditions	Results	References
Swiss albino mice	2.45 GHz; 10 W/m^2; whole-body SAR estimated as 0.2 W/kg; 2 h/day for 120, 150, 200 days	Evidence of increased DNA rearrangement in samples from testes and brain	Sarkar *et al*, 1994
Mice	34 GHz pulsed (Police radar); 200 mW/m^2; 17 h/day; 5 days/week for 2 weeks	DNA synthesis in cells from irradiated corneas reduced by 25%, but not statistically significantly	Rotkovska *et al*, 1993
Sprague-Dawley rats: comet assay for DNA damage in cerebral cortex	2.45 GHz CW; whole-body SAR 1.2 W/kg; 2 h	No effect on the frequency of DNA breaks immediately or 4 h after exposure	Malayapa *et al*, 1998
Sprague-Dawley rats: comet assay for DNA in rat brain	2.45 GHz CW or pulsed; 2 µs pulses; 500 pps; whole-body SAR 0.6 or 1.2 W/kg; SA per pulse = 1.2 or 2.4 mJ/kg; 2 h	Increased single- and double-strand DNA damage; maximum 4 h after exposure	Lai and Singh, 1995, 1996

CW = continuous wave

5.132 An increase in the number of single-strand and double-strand DNA breaks was reported in the brain cells of rats exposed for two hours to pulsed or continuous-wave 2.45 GHz radiation (Lai and Singh, 1995, 1996). Moreover, this effect was blocked by treatment, before or after exposure, with melatonin (see paragraph 5.96) or another free-radical scavenger (Lai and Singh, 1997). The DNA breaks were revealed using a single cell gel electrophoresis (or comet) assay in which the brain cells were isolated, placed on a microscope slide, lysed and eventually electrophoresed. DNA was stained using a fluorescent stain and DNA "fragmentation" assessed from the DNA migration path length. Curiously, in both studies the background levels of DNA breaks in brain cells from the unexposed animals were unusually high compared to values reported in other studies (see, for example, McKelvey-Martin *et al*, 1993), This raises the possibility that there was

insufficient control of DNA breakage during preparation of the rat brain cells, although this in itself could not explain significant differences between exposed and control groups.

5.133 More rigorous studies by Malyapa *et al* (1997a,b, 1998) were not able to reproduce these findings. In particular, no effect was seen in the brain (hippocampal cells) of rats exposed for two hours to 2.45 GHz radiation, nor in mouse C3H10T½ fibroblast cells and human glioblastoma U87MG cells exposed *in vitro* (Malyapa *et al*, 1997a,b) to 2.45 GHz radiation or modulated 835 MHz radiation. A number of other studies have also failed to reveal any evidence of DNA damage.

5.134 *CONCLUSION This area deserves further research, but the evidence of Sarkar et al (1994) and Lai and Singh (1995, 1996) for DNA damage in mice is contradicted by a number of other studies in vivo and is not supported by in vitro work.*

DNA damage: indirect indicators

5.135 Various agents (clastogens) induce distortions of chromosomes, visible under the microscope. These chromosomal aberrations are generally thought to be due to damage to DNA or unusual interactions between DNA and protein molecules. Their accumulation is evidence of genotoxicity and is usually associated with cancer, but can also result in developmental abnormalities or miscarriage, if present in the tissue that generates eggs or sperm, or in the developing embryo or fetus.

5.136 Genotoxic substances tend to cause sister chromatid exchanges (switching of DNA from one part of the chromosome to another), which can be quite sensitively detected.

5.137 The occurrence of cells with unusually small nuclei (micronuclei) is also taken as an indicator of DNA damage. The detection of cells with micronuclei is simple, but is relatively unreliable because of the high and variable incidence of cells judged to be micronucleate in healthy tissues (10%–20% false positive rate). Heating is also known to induce micronuclei.

Chromosomal aberrations

5.138 Many studies have not detected obvious chromosomal aberrations in isolated animal cells after exposure to low power RF radiation (see, for example, Alam *et al*, 1978; Lloyd *et al*, 1984, 1986; Wolff *et al*, 1985; Meltz *et al*, 1987, 1989, 1990; Kerbacher *et al*, 1990; Maes *et al*, *in press*). However, a similar number of studies have reported increased chromosomal aberration (Yao and Jiles, 1970; Chen *et al*, 1974; Yao, 1976, 1982; Garaj-Vrhovac *et al*, 1990a, 1991, 1992; Khalil *et al*, 1993; Maes *et al*, 1993, 1995). In those studies with positive results in which the stimulus intensity was properly documented, it was generally rather high and therefore thermal effects cannot be ruled out. The experiments of Garaj-Vrhovac *et al* (1990a, 1991) are a notable exception; they reported an increase in chromosomal aberration after exposure of Chinese hamster V79 cells to 7.7 GHz radiation. Khalil *et al* (1993), in a preliminary report, also described increased chomosomal aberration in human lymphocytes after exposure to 167 MHz RF fields at 55 W/m^2 for up to 72 h.

5.139 Most experiments on whole animals have shown no increase in chromosomal aberration after exposure to RF fields, even though many of them employed high intensities, which were likely to have caused a rise in body temperature (Table 5.12). There have also been a few reports of aberrations *in vivo*, again with intensities near to or above the thermal limit. Of particular interest is the work by Garaj-Vrhovac *et al* (1990b) on chromosomal aberration in the lymphocytes of people who had experienced occupational exposure to 30–300 GHz at 10–50 W/m^2, although Maes *et al* (1995) found no such chromosomal aberrations in antenna maintenance workers who had been exposed to various RF fields at least one hour each day for more than a year.

Table 5.12 *In vivo* studies of chromosomal aberrations

Model	Exposure conditions	Results	References
Human lymphocytes from antenna workers	2.45 GHz and 954 MHz	Increased CA frequency	Maes *et al*, 1993, 1995
Mammalian, C3H mice	2.45 GHz CW; 100 W/m^2; 4 W/kg; 6 hours/day; 120 hours over 8 weeks	No effect on CA frequency	Saunders *et al*, 1988
Male germ (sperm-forming) cells of C3H/HeH mice	2.45 GHz CW; 1, 100, 400 W/m^2; 0.05–20 W/kg; 30 min/day; 6 days per week, for 2 weeks	No effect on CA frequency	Beechey *et al*, 1986
Male germ (sperm-forming) cells of CBA/CEY mice	2.45 GHz CW; whole-body SAR 0.05–20 W/kg; 30 min per day; 6 days per week for 2 weeks	Increased CA frequency	Manikowska-Czerska *et al*, 1985
Human lymphocytes (radio-linemen)	0.4 MHz to 20 GHz; occupational exposure	No increase in CA frequency	Garson *et al*, 1991
Human lymphocytes (antenna workers)	Various frequencies (including 450 and 950 MHz); 1 h/day for at least one year; occupational exposure	No increase in CA frequency	Maes *et al*, 1995
Human lymphocytes (radar station workers)	30–300 GHz, 1000–5000 W/m^2; occupational exposure	Increased CA frequency	Garaj-Vrhovac *et al*, 1990b

CW = continuous-wave

Sister chromatid exchange

5.140 Most observations of sister chromatid exchange *in vitro* (Table 5.13) have failed to detect any effect of RF exposure, even at high intensities (see, for example, Lloyd *et al*, 1984 and 1986, Wolff *et al*, 1985, and Maes *et al*, 1993 and 2000, on human lymphocytes; Wolff *et al*, 1985, Meltz *et al*, 1990, and Ciaravino *et al*, 1991, on hamster ovary cells). Maes *et al* (1997) described a very small increase (statistically significant in two out of four samples) in sister chromatid exchange in human lymphocytes exposed to 935.2 MHz at an SAR of 0.3–0.4 W/kg. Khalil *et al* (1993) observed a clear increase in sister chromatid exchange in isolated human lymphocytes after exposure to 167 MHz at 55 W/m^2. However, this study was described as preliminary, and may have been compromised by the fact that the control cultures were kept in the stable environment of an incubator while the experimental samples were brought out for exposure.

Micronucleus formation

5.141 In contrast to these generally negative results from observations of chromosomal aberrations and sister chromatid exchange, there have been reports of an increase in the number of cells with micronuclei after exposure to RF radiation (Royal Society of Canada, 1999). Increased micronucleus formation has been seen in *in vitro* studies of various plant and animal cells (Garaj-Vrhovac *et al*, 1991, 1992; Maes *et al*, 1993; Haidler *et al*, 1994; Garaj-Vrhovac, 1999). However, the exposure conditions were generally rather poorly defined or sufficiently intense to have caused thermal effects (Table 5.14). Results of observations *in vivo* have been less clear. For instance, Vijayalaxmi *et al* (1997a,b) saw no effect on blood cells in mice exposed to 2.45 GHz radiation for 18 months at a whole-body SAR of 1 W/kg. However, Antipenko and Koveshinkova (1987) found an increase in the numbers of micronuclei in mouse hepatocytes after exposure to continuous-wave or pulsed RF radiation at an intensity of 5 W/m^2 for 45 days at 7 hours per day. Garaj-Vrhovac *et al* (1990b) reported no increase in micronuclei in the lymphocytes of human subjects who had had occupational exposure of unknown intensity.

However, Balode (1996) found significant effects in the blood of cattle on farms close to a major radar installation in Latvia.

Table 5.13 Effects of RF fields on sister chromatid exchange *in vitro*

Model	Exposure conditions	Results	References
Human lymphocytes in hypothermic or mildly hyperthermic conditions	2.45 GHz; up to 200 W/kg; 20 min	No effect on SCE frequency	Lloyd *et al*, 1984; 1986
Human lymphocytes and Chinese hamster ovary cells during MRI	100 MHz pulsed; 330 µs pulses; 100 pps; static magnetic field of 2.35 tesla	No effect on SCE frequency	Wolff *et al*, 1985
Chinese hamster ovary cells with or without chemical mutagen; mildly hyperthermic	2.45 GHz Pulsed; 10 µs pulses; 25 x 10^3 pps; 33.8 W/kg; 2 h	No effect of RF on SCE frequency; no effect on mutagen-induced SCE frequency	Ciaravino *et al*, 1991
Human lymphocytes	167 MHz; 55 W/m^2; up to 72 h	Increased frequency of SCE	Khalil *et al*, 1993
Human lymphocytes maintained at 36.1˚C	2.45 GHz; 75 W/kg; 30 or 120 min	No effect on SCE frequency	Maes *et al*, 1993
Human lymphocytes with or without chemical mutagen (Mitomycin C)	935.2 MHz CW; 0.3–0.4 W/kg; 2 h	Little or no effect of RF alone on SCE frequency; slight enhancement of effects of the mutagen	Maes *et al*, 1997
Human lymphocytes with or without chemical mutagen	900 MHz CW (pseudo-random signal and dummy burst signal); 0.4–10 W/kg; 2 h	No effect on SCE frequency	Maes *et al*, *in press*
Human lymphocytes maintained at 17˚C with or without chemical mutagen	954 MHz pulsed (GSM); 1.5 W/kg; 2 h	No effect of RF alone on SCE frequency; RF-enhanced effect of chemical mutagen	Maes *et al*, 1995

Table 5.14 *In vitro* studies on micronucleus formation

Model	Exposure conditions	Results	References
Peripheral blood of Latvian Brown cows (2000 erythrocytes)	Farm close to and in front of the Skrunda Radar	Significant differences in the frequency distribution of micronuclei	Balode, 1996
C3H/HeJ mice (prone to mammary tumours); peripheral blood	2.45 GHz; whole-body SAR 1 W/kg; 20 h/day; 7 days/week for 18 months	Small but significant increase in micronuclei in polychromatic erythrocytes in peripheral blood and bone marrow	Vijayalaxmi *et al*, 1997b (1998 erratum)
CF-1 male mice, peripheral blood and bone marrow	Ultra-wideband radiation; estimated whole-body SAR 37 mW/kg; 15 min;	No effect on micronuclei in polychromatic erythrocytes in peripheral blood and bone marrow	Vijayalaxmi *et al*, 1999
Mouse hepatocytes	CW or pulsed RF; 5 W/m^2; 7 h; 45 days	Increase in micronuclei	Antipenko and Koveshinkova, 1987
Lymphocytes from human subjects (radar station workers)	30–300 GHz; 1000–5000 W/m^2; occupational exposure	Variable increase in micronuclei In lymphocytes	Garaj-Vrhovac *et al*, 1990b

5.142 Micronucleus formation is thought to reflect DNA damage, and is a sensitive assay (since the aberrant cells tend to accumulate, especially among non-dividing and slowly-dividing cells). However, its implications for health are unclear. In any case, the incidence of micronuclei in normal tissues is rather high and variable, making the assessment of individual experimental results more difficult.

Other evidence

5.143 Several publications in Russian, two decades ago or more, described a number of changes in cellular appearance and function after exposure to RF radiation. For instance, Zalyubovskaya and Kiselev (1978, in Russian), cited by McRee (1980), reported various pathological effects of 6.5 mm (46 GHz) RF radiation on cultured embryonic human and pig kidney cells, and Hep-2 cells. The cells developed pyknotic or vacuolised nuclei and damaged membranes, and their survival rate was reduced by 30%–50%. Kiselev and Zalyubovskaya (1976, in Russian), cited by McRee (1980), found that exposure of adenovirus to 6.5 mm (46 GHz) RF radiation reduced the capacity of the virus to infect cultured human kidney cells. Unfortunately the exact conditions of stimulation in many of these early studies were either poorly defined or were so intense that they would have produced thermal effects.

5.144 *CONCLUSION Several different assays of genotoxicity have failed to produce clear evidence that RF radiation is genotoxic at non-thermal levels. The most consistent results come from observations of micronucleus formation, but these are not simple to interpret and have uncertain implications for health.*

Long-term studies of cancer induction in animals

5.145 A demonstration that long-term exposure to RF fields increases the incidence of tumours in animals would provide direct evidence that such radiation is carcinogenic. Early studies (Prausnitz and Susskind, 1962; Spalding *et al*, 1971; Skidmore and Baum, 1974; Baum *et al*, 1976) in which biological endpoints relevant to carcinogenesis were examined suffered from insufficient dosimetry, poor histopathology or inadequate follow-up.

Spontaneous tumour incidence

5.146 Later studies (Table 5.15) avoided many of these deficiencies. Chou *et al* (1992) exposed (from 2 to 27 months of age) 100 rats to low level pulsed 2.45 GHz fields. The total number of benign tumours of the adrenal medulla was higher in the exposed group compared to the control group,

Table 5.15 Effect on spontaneous tumour incidence

Model	Exposure conditions	Results	References
Sprague-Dawley rats	2.45 GHz pulsed; 10 µs pulses at 800 pps, pulse modulated at 8 pps; whole body SAR of 0.4–0.15 W/kg for up to 25 months	No increase in individual cancers; four-fold increase in primary malignancies	Chou *et al*, 1992
Spontaneous mammary tumours in C3H/HeA mice	2.45 GHz; up to 6–8 W/kg; to 12 months of age	Increased tumour development	Szmigielski *et al*, 1982
Mammary tumour prone C3H/HeA mice	435 MHz pulsed; 1 µs pulses at 1000 pps; whole-body SAR of 0.32 W/kg; 21 months	No effect	Toler *et al*, 1997
Mammary tumour prone C3H/HeA mice	2.45 GHz; whole-body SAR 0.3 or 1.0 W/kg; 18 months	No effect	Frei *et al*, 1998a,b

although not particularly higher than that reported elsewhere for this strain of rat. There was no significant difference between groups in the incidence of all benign neoplasms at death. In addition, no single type of malignant tumour was enhanced by exposure and, overall, the incidence of primary malignancies was similar to the spontaneous rate reported for this strain of rat. However, when the occurrence of primary malignant lesions was pooled without regard to site or mode of death, the exposed group had a significantly higher incidence compared to the control group.

5.147 Other authors have reported a lack of effect of RF exposure on cancer incidence in mice prone to mammary tumours. Toler *et al* (1997) found that the long-term exposure of mammary-tumour-prone C3H/HeJ mice to 435 MHz pulsed RF radiation had no effect on survival, nor on the latency, incidence or growth rate of mammary tumours compared to sham-exposed animals, nor were there differences in the numbers of malignant, metastatic or benign tumours. Frei *et al* (1998a,b) used the same animal model to investigate the effects of chronic (18-month) low level 2.45 GHz RF exposure and found no differences compared to sham-exposed animals.

Epigenetic effects: does RF radiation interact with known genotoxic agents to enhance their promotional effects?

5.148 Epigenetic factors, although not themselves genotoxic, act synergistically to enhance the carcinogenic effects of other agents (Williams and Whysner, 1996). There are several published studies that suggest that RF radiation can have an epigenetic effect *in vivo*, working to exaggerate the genotoxic influences of ionising radiation or cancer-inducing substances, or to potentiate other epigenetic factors. However, the evidence for an epigenetic effect of RF exposure is equivocal, with several failures to replicate positive results.

5.149 Balcer-Kubiczek and Harrison (1985, 1991) reported that 2.45 GHz radiation can induce "latent transformation" of a cultured cell line. This RF radiation potentiated the tumour-transforming effect of X-rays or the carcinogenic substance benzo[a]pyrene, but only in the presence of TPA, a known epigenetic agent. On the other hand, Cain *et al* (1997) found no cell transformation in similar experiments involving 836.55 MHz radiation.

5.150 Scarfi *et al* (1996) reported that intense RF fields amplified the genotoxic effects of the mitogenic substance mitomycin-C, as judged by the presence of micronuclei in cultured bovine lymphocytes. Maes *et al* (1997) described a small but statistically significant enhancement of the effects of mitomycin-C on human lymphocytes after exposure to 935.2 MHz radiation for two hours. However, Ciaravino *et al* (1987, 1991) found no epigenetic influence of RF radiation on the production of chromosomal aberrations by mitomycin-C or another mitogen, adriamycin. A number of other studies have failed to demonstrate enhancement of the mutagenic action of chemical carcinogens (Meltz *et al*, 1989, 1990; Kerbacher *et al*, 1990).

5.151 Heating alone can enhance the action of genotoxic agents (Jorritsma and Konings, 1984; Miura *et al*, 1986) and it is possible that some of the epigenetic influences reported for RF could be due to thermal effects. Indeed, this was one of the conclusions of Pakhomova *et al* (1997), who found that higher frequency RF radiation (61 GHz) did enhance DNA recombination (but not mutagenesis) in yeast cells exposed to ultraviolet radiation.

5.152 Chemical tumour-inducing agents have been used to examine possible epigenetic, promotional effects of RF radiation in animals (Table 5.16). Early studies were carried out by Szmigielski and colleagues (Szmigielski *et al*, 1982; Szudinski *et al*, 1982) who reported that the chronic RF exposure of mice resulted in a SAR-dependent increase in the development of spontaneous mammary tumours or skin tumours induced by the repeated application of benzo(a)pyrene. Body temperatures were not raised but the authors suggested a possibility of localised heating at the

highest level of exposure. A further experiment (Szmigielski *et al*, 1988) showed that exposure followed by the application of a "subcarcinogenic" dose of the same carcinogen to the skin, a procedure repeated daily, eventually resulted in a three-fold increase in the numbers of skin tumours appearing. The authors noted that these results needed confirmation but suggested that long-term exposure to RF fields might promote the development of neoplasms that would not normally reach a clinically identifiable stage.

Table 5.16 Promotional studies *in vivo*

Model	Exposure conditions	Results	Reference
Chemically induced skin tumours in BALB/c mice	2.45 GHz; whole-body SAR 2–3 or 6–8 W/kg; 1–3 months	Increased incidence of skin tumours	Szmigielski *et al*, 1982; Szudinski *et al*, 1982
Chemically induced hepatomas and sarcomas in mice	2.45 GHz; whole-body SAR up to 4–5 W/kg; several months	Increased incidence of tumours	Szmigielski *et al*, 1988
Chemically induced colon cancers in BALB/c mice	2.45 GHz; whole-body SAR 10–12 W/kg; 5 months	No effect	Wu *et al*, 1994
Lymphoma-prone Eμ-*Pim1* transgenic mice	900 MHz pulsed (GSM); 600 μs pulses; 217 pps; whole-body SAR; 0.008–4.2 W/kg; 1 h per day; 18 months	Two-fold increase in lymphoma incidence	Repacholi *et al*, 1997
Medium term rat liver tumour promotion model	1.439 GHz pulsed; 6.7 ms at 50 pps; whole-body SAR 0.4–0.7 W/kg; 6 weeks	No effect	Imaida *et al*, 1998a
Medium term rat liver tumour promotion model	929.2 MHz pulsed; 6.7 ms pulses; 50 pps; whole-body SAR 0.6–0.8 W/kg	No effect	Imaida *et al*, 1998b
Lymphoma incidence in CBA/S mice exposed to 4 Gy ionising radiation	902 MHz CW; 1.5 W/kg or 902 MHz pulsed (GSM); 0.35 W/kg; 1.5 h/day; 5 days/week	No effect on survival or incidence of lymphoma	Juutilainen *et al*, 1998; *in press*
Spontaneous and chemically induced CNS tumours in Fischer rats	836.55 MHz pulsed; 6.7 ms pulses; 50 pps; brain SAR 0.3–0.5 W/kg; perinatally and for 24 months	Fewer CNS glial tumours in exposed group	Adey *et al*, 1999
Chemically induced sarcomas in female Sprague-Dawley rats	900 MHz pulsed (GSM); whole-body SAR 75 or 270 MW/kg; 2 weeks; 20, 40 or 75 days after carcinogen	No effect on tumour appearance or survival	Chagnaud *et al*, 1999
Chemically induced brain tumours in Sprague-Dawley rats	860 MHz bipolar RF pulses (MIRS: 11.1 Hz frame rate unipolar wave form) or 860 MHz CW; head SAR 0.4–1.0 W/kg; 6 h/day; 5 days/week; 2–24 months of age	No effect on incidence, multiplicity, volume, malignancy or fatal outcome of neural tumours	Zook, 1998; 1999

CW = continuous wave MIRS = Motorola integrated radio service GSM = global system for mobile telecommunications

5.153 The most positive evidence, as yet unreplicated, of an effect of exposure to RF radiation similar to that used by digital mobile phones was reported by Repacholi *et al* (1997), using Eμ-*Pim1* transgenic mice. Experimental transgenic animals have genetic material added to their DNA to predispose them to the endpoint being investigated. Such animals that are prone to cancer can be

used to test the carcinogenic influence of chemical agents in a short time (generally six months). This not only provides a sensitive animal model to test for cancer, but tests can be conducted in a time much less than the animal's lifetime. Eμ-*Pim1* mice, which are moderately susceptible to the development of lymphoblastic lymphomas, were exposed or sham-exposed for one hour per day for eighteen months to pulse-modulated 900 MHz RF radiation. The authors reported an increase in the incidence of all lymphomas (lymphoblastic and non-lymphoblastic) in the exposed mice (43% compared to 22% in the controls), the most significant effect resulting from increased non-lymphoblatic lymphomas. However, lymphoma incidence was rapidly increasing in both exposed and sham-exposed animals when the study was terminated; in addition, only moribund animals were examined histopathologically. Replication of this study and extension with more complete follow-up and improved dosimetry would be useful and is currently under way in Australia. Another replication study is commencing in Europe in 2000. These replication studies will expose the mice to different levels of RF radiation to determine if there is a dose–response relationship. Even if replicated, there will need to be further assessment of the relevance of these findings for human health (Repacholi, 1998).

5.154 Other recent studies investigating a possible promoting effect on chemically induced cancers have generally found tumour incidence to be unaffected by RF exposure. Wu *et al* (1994) reported that the chronic exposure to 2.45 GHz RF radiation had no effect on the incidence or size of colon cancers induced in mice by dimethylhydrazine. Imaida *et al* (1998a) found no effect of exposure at the Japanese cellular phone frequency of 1.439 GHz for six weeks using the standard medium-term rat liver tumour promotion model, in which neoplastic foci are induced in the liver by diethylnitrosamine and partial hepatectomy. Similar results for 929.2 MHz radiation had been reported previously (Imaida *et al*, 1998b). In contrast to these reports, Adey *et al* (1999) found that exposure to 836.55 MHz radiation with North American Digital Cellular Modulation (at SAR levels reported to simulate the localised peak brain exposures experienced by a cell phone user) over a 24-month period had a slight inhibitory effect on both spontaneous and chemically induced brain tumour incidence in rats, the latter resulting from treatment *in utero* with the carcinogen ethylnitrosourea. There was, however, no evidence of any tumorigenic effect.

5.155 *CONCLUSION Although thermal effects may account for the positive reports that RF radiation enhances the actions of genotoxic agents, the evidence for epigenetic effects must be taken seriously. Further research is needed in this area to clarify the position.*

Tumour transplantation: does RF radiation enhance the progression of transplanted tumours?

5.156 The effect on tumour progression of exposure to continuous-wave or pulse-modulated RF radiation following the transplantation of tumour cells into mice and rats has been examined in several studies. Szmigielski *et al* (1982) reported a SAR-dependent increase in the number of neoplastic colonies on the lung surfaces of mice injected intravenously with suspensions of mouse sarcoma cells and exposed to 2.45 GHz radiation. In contrast, other studies (Santini *et al*, 1988; Salford *et al*, 1993; Higashikubo *et al*, 1999) have found no effect of RF exposure (see Table 5.17).

General conclusions from studies of cancer

5.157 *Some individual experimental studies have suggested that RF radiation can initiate tumour formation, enhance the effects of known carcinogens or promote the growth of transplanted tumours. However, in some of these the intensity was high enough to produce thermal effects. The balance of evidence, from both in vitro and in vivo experiments, indicates that neither acute*

Table 5.17 Tumour transplantation studies

Model	Exposure conditions	Results	References
BALB/c mice injected with L1 mouse sarcoma cells	2.45 GHz; whole-body SAR 2–3 or 6–8 W/kg; 3 months	Increased incidence of tumour colonies on lung surface	Szmigielski *et al*, 1982
Mice injected with mouse B16 melanoma cells after exposure	2.45 GHz CW or pulsed; whole-body SAR 1.2 W/kg	No effect on survival	Santini *et al*, 1988
RG2 glioma cells injected into rat brains	915 MHz CW or pulsed; whole-body SAR 0.01–2 W/kg; 2–3 weeks	No effect on tumour growth	Salford *et al*, 1993
9L gliosarcoma cells injected into rat brains	835.62 MHz; FM or 847.74 MHz CDMA; brain SARs estimated as around 0.75 W/kg; 21 weeks after treatment;	No effect on tumour growth	Higashikubo *et al*, 1999

CW = continuous wave FM = frequency modulated CDMA = code division multiple access

nor chronic exposure to RF fields increases mutation or chromosomal aberration frequencies when temperatures are maintained within physiological limits (UNEP/WHO/IRPA, 1993). This suggests that RF exposure is unlikely to act as a tumour initiator. Further, a variety of cancer studies using animals have sought evidence of an effect of RF exposure on spontaneous or natural cancer rates, the enhancement of the effects of known carcinogens or effects on the growth of implanted tumours. However, they have provided equivocal evidence for an effect on tumour incidence (ICNIRP, 1996; Repacholi, 1998; Moulder et al, 1999; Royal Society of Canada, 1999).

Haematopoetic system, immune system and longevity

5.158 Changes in the haematopoetic system (tissues related to the formation of blood cells) can have a direct effect on health. Information about any effects of RF exposure on this system is fundamentally important, then, for the assessment of possible risks to health and well-being.

5.159 No consistent effects of low level RF exposure have been reported on the blood-forming and circulating blood cells (eg changes in numbers of bone marrow cells, lymphocytes or erythrocytes, or in the amount of haematocrit) (UNEP/WHO/IRPA, 1993; Jauchem, 1998). Most of the earlier studies used continuous-wave fields, but a well-conducted lifetime study in rats exposed to low level, pulsed RF fields also found no effect on haematology or serum chemistry parameters (Chou *et al*, 1992).

5.160 The immune system defends against micro-organisms, viruses, and some cancer cells. Thus any effects of RF exposure on this system could have significant implications for health. Thermal levels of RF exposure elicit both stimulatory and inhibitory responses in components of the immune system (UNEP/WHO/IRPA, 1993). However, these effects (eg changes in lymphocyte activity and responsiveness) were generally found to be transitory, returning to normal levels following termination of RF exposure. Studies using low level RF exposure have given inconsistent results, making it difficult to attribute any effects to the exposure. Several acute and chronic *in vivo* studies, using low RF exposure levels, have indicated positive effects that were transient and similar to those resulting from thermal stress or physiological changes occurring during thermoregulation (Smialowicz *et al,* 1983; Yang *et al,* 1983). However, Fesenko *et al* (1999) have recently shown that exposure of mice to 10 GHz radiation, or radiation with a frequency swept between 8.15 and 18 GHz, at very low intensity (probable SAR 2–5 mW/kg), increases the production by macrophages of tumour necrosis factor, which is

involved in immune attack on viruses, foreign proteins and damaged tissues. Novoselova *et al* (1999) showed that this effect is enhanced by a diet rich in antioxidants. These authors suggest that "the activation of cellular immunity by RF irradiation and antioxidant treatment may provide therapeutic strategies for interfering with acute immunodeficiency processes".

5.161 In general, studies conducted to determine if RF exposure affects longevity have revealed no influence on lifespan in experimental animals. Indeed, the most convincing changes reported are very slight increases in lifespan, perhaps because of a subtle, thermally-mediated effect on food intake. Summaries of typical studies are given in Table 5.18.

Table 5.18 Longevity and general physiological condition

Model	Exposure conditions	Results	References
Mice	0.80 GHz; 43 W/m^2; 2 hours/day; 5 days/week; 35 weeks	No effect on lifespan	Spalding *et al*, 1971
CFW mice (*in utero* exposure to RF and implantation of tumour cells)	2.45 GHz; 35 W/kg (leading to an increase in body temperature of 2.24 ˚C); 20 min/day; 3 days; 11-14th day of gestation	Increased lifespan in tumour-bearing and tumour-free animals	Preskorn *et al*, 1978
Sprague-Dawley rats	2.45 GHz pulsed; 10 µs pulses; 800 pps; pulse modulated at 8 pps; whole-body SAR of 0.15–0.4 W/kg; up to 25 months	No effect on lifespan	Chou *et al*, 1992
CD1 mice	2.45 GHz; 30–100 W/m^2; whole-body SAR 2 or 6.8 W/kg; 1 h/day; 5 days/week throughout their life	Significantly shortened lifespan of mice exposed to 100 W/m^2 and slight but not significantly longer average lifespan of mice exposed to 30 W/m^2	Liddle *et al*, 1994
New Zealand rabbits	2.45 GHz; head SAR of 0.55 or 5.5 W/kg; 7 h per day; 13 weeks	No effect on haematology, cataract incidence or histopathology; slight drop in food consumption in high exposure group	Chou *et al*, 1983

Reproduction and development

5.162 Many of the processes associated with reproduction are especially sensitive to toxic influences. Meiosis (the division of cells to produce sperm and eggs), fertilisation and implantation of the embryo can all be disturbed by toxic insults. The high rates of cell division and differentiation in the developing fetus make it particularly vulnerable. It is well known that some drugs and environmental hazards have damaging (teratogenic) effects on the developing embryo or fetus at exposure levels that pose little or no risk to the adult animal. It is therefore important to assess the possible effects of RF fields on fertility and development.

5.163 Extensive research on a wide range of species (from beetles to guinea pigs) has failed to reveal any convincing effects of low level RF fields on developing animals (reviewed by UNEP/WHO/IRPA, 1993; Jauchem, 1998; O'Connor, 1999). For instance, exposure of pregnant Holtzman rats for 20 minutes at SARs of 14 or 28 W/kg, sufficient to raise the rectal temperature to an average of 42˚C, produced no observable effects on the offspring (O'Connor, 1980). Only if the field strength was so high that the rectal temperature of the dam reached near-lethal levels

(43˚C) was there an increase in fetal absorption, a decrease in fetal body mass and excessive occurrence of birth defects (Chernovetz *et al*, 1977; O'Connor, 1980). The survival of fetuses was inversely correlated with the temperature to which the dam was raised, and similar effects were produced by raising the dam's body temperature by infrared heating (Chernovetz *et al*, 1977).

5.164 Jensh (1997) described extensive studies on the effects of prenatal RF exposure at non-thermal levels on development in rats. Female rats were exposed for six hours each day throughout pregnancy to continuous-wave radiation at 915 MHz (100 W/m^2), 2.45 GHz (200 W/m^2) or 6 GHz (350 W/m^2) and many physiological and behavioural parameters were assessed in both mothers and offspring. Some of the offspring from RF-exposed pregnancies were later bred and the second generation offspring were also assessed. Exposure to 915 MHz radiation (in the range of mobile phone signals) had no detectable effect. For 2.45 GHz radiation, the only positive result was a very slight increase of activity in the offspring. Exposure to 6 GHz radiation at 350 W/m^2 did have some just statistically significant effects. The number of monocytes (a class of white blood cells) in the mothers' blood was slightly but significantly reduced. The weight of the offspring at term and during the first five postnatal weeks was slightly lower than that for control litters and there were small differences in the relative weights of kidney and liver. The exposed offspring opened their eyes slightly earlier than did control pups. The offspring from exposed litters tended to be more active in an "open field" test of spontaneous activity, and the females were slightly more active on an activity wheel, in a swimming test and in a water T-maze. Their actual performance on the water maze was slightly worse than that of control animals. In subsequent pregnancies from matings of animals derived from exposed mothers, the weight gain of the pregnant females and the size of their litters was slightly reduced. Jensh (1997) concluded that, since all these effects were so minor, and almost all were associated only with 6 GHz radiation, the fields associated with mobile phones do not "produce a significant increase in reproductive risk".

5.165 From other experiments on rats and mice, Berman *et al* (1978) and Lary *et al* (1982, 1983) concluded that the threshold for teratogenic effects corresponds to an elevation of temperature of the fetus to 40˚C for some considerable time. Prolonged exposure of pregnant rats at thermal levels can lead to behavioural deficits in the offspring, including poorer performance in a water T-maze and an open-field activity test (Jensh *et al*, 1983a,b; Jensh, 1984a,b), and altered thermoregulatory behaviour (O'Connor, 1988).

5.166 Exposure to relatively high level RF radiation (but not high enough to be teratogenic on its own) enhances the effects of chemical teratogens (see, for example, Marcickiewicz *et al*, 1986; Nelson *et al*, 1991), but this is almost certainly due to the rise in fetal temperature.

5.167 There were early reports of decreased fertility in male rats exposed to RF fields (Varma and Traboulay, 1976, 1977). However, it is likely that these effects were due to heating. Saunders and Kowalczuk (1981) found no abnormality of testis cells or sperm count even in adult male rats that had been exposed to quite strong RF fields (66 W/kg for 10 minutes or 7 W/kg for 260 minutes). Very prolonged exposure to 200 MHz fields, amplitude modulated at 16 Hz, at an SAR of about 2 W/kg, has been reported to decrease male fertility, judged by the number of offspring per litter and by histological changes in the seminiferous tubules (Khillare and Behari, 1998). These effects could well have been caused by modest warming integrated over the long exposure period.

5.168 Magras and Xenos (1997) have reported an apparently dramatic decrease in the fertility of mice exposed to extremely low level RF fields near the "antenna park of Thessaloniki" (almost 100 TV and FM-radio antennas on the Chortiatis mountain in Greece). The mice, which were

kept in, and moved between, various locations (including a school) in a nearby village, were exposed to a broad spectrum of RF radiation with an integrated intensity between about only 1.7 and 10 mW/cm^2. Within three to five matings, the number of pups per litter had fallen literally to zero. Unfortunately there was no matched control group and no account was taken of the possibility that the result was due to noise, smells or other stressful factors in the environment of the experimental mice.

5.169 *CONCLUSION There is no convincing evidence from studies of rodents that exposure to RF fields at levels associated with mobile telecommunications poses any risk for the fetus or for male fertility. While there are good reasons to doubt whether the decline in female fertility described by Magras and Xenos (1997) was actually due to the very low level exposure, it is important to repeat this study under better controlled conditions.*

Influences on the cardiovascular system

5.170 Radiofrequency radiation might affect the heart and circulation through a number of routes. There could be direct effects on the heart and blood vessels. There might be influences on the cardiovascular centres in the medulla of the brainstem, which regulate the heart and circulation via the outflow in the sympathetic and parasympathetic systems. Exposure to RF fields might conceivably affect the receptors in the carotid body, which normally detect blood pressure and blood gases and which initiate reflex influences on the heart and blood vessels. Finally, the cardiovascular system is known to be affected by a variety of circulating substances, especially catecholamine hormones, whose release might possibly be changed by exposure to RF fields.

5.171 Exposure of animals to high levels of RF radiation, sufficient to raise body temperature, certainly results in a variety of direct and indirect effects on the cardiovascular system. However, there is no evidence that they differ qualitatively or quantitatively from effects triggered by similar rises in body temperature produced by other means (Jauchem and Frei, 1992). High peak-power pulsed RF fields or broad-spectrum pulses of electromagnetic radiation (which would result in *total* absorbed energy in people well below guidelines) do not cause detectable changes in heart rate or blood pressure in animals (Erwin and Hurt, 1993; Jauchem and Frei, 1995; Jauchem, 1997).

5.172 *CONCLUSION Studies on animals do not justify any concern about the influence of RF radiation at levels associated with mobile phones on the heart or circulation. Effects at high intensities appear to be due to heating of the body.*

Summary and conclusions on animal and cellular experimental studies

5.173 *The thermal consequences of acute RF exposure in animals appear to account for many of the reported effects on the cardiovascular, endocrine and immune system and on behaviour (UNEP/WHO/IRPA, 1993). Rectal temperature increases of at least 1–2˚C are needed to produce these effects. Developmental effects, similar to those known to be induced by heat, have been reported in rodents following large (3–4˚C) temperature increases in the fetus during prenatal RF exposure (Lary and Conover, 1987).*

5.174 *Other effects remain somewhat controversial. The question of whether low level RF exposure might increase the risk of cancer is of particular concern, although, since the radiation lacks sufficient energy to disrupt molecular bonds directly, there appears to be no theoretical basis to suggest that it could adversely affect DNA.*

5.175 *It has been reported that exposure to low level pulsed (and sometimes continuous-wave) RF fields can damage ocular tissues in primates and produce non-specific stress-like changes in the*

rat brain. If such effects occur in people they could have implications for health. There is also a body of evidence describing biological responses to amplitude-modulated RF fields at SARs too low to involve any response to heating. This literature is, however, inconsistent, and the effects that are reported are typically small and close to the level of statistical noise. It is thus very difficult to interpret, in terms of either its biological significance or its implications for human health.

Laboratory Studies of the Effects of RF Radiation on People

Brain function

5.176 Among the concerns expressed over the use of mobile phones is the possibility that mobile phone signals have deleterious effects on cognitive functions such as memory, attention and concentration. Despite these concerns, relatively few laboratory studies have addressed this issue in people and, of those that have, all have investigated short-term effects of exposure. Further, with three exceptions, studies investigating exposure to low levels of RF radiation in the mobile phone frequency range have focussed not on indices of cognitive performance *per se*, but on physiological measures of brain function such as the electroencephalogram (EEG).

Studies of cognitive performance

5.177 Investigation of short-term effects of exposure to electromagnetic fields is, in principle, relatively easy. A wide range of tasks have been developed by experimental psychologists to assess specific aspects of cognitive function (eg short-term and long-term memory, selective attention, speed of decision-making), and the experimental designs required to determine whether electromagnetic field exposure affects performance on such tasks are unproblematic. To date, however, the effects of mobile phone signals on cognitive performance have been assessed in only three published studies (Preece *et al*, 1999; Koivisto *et al*, 2000, *in press*).

5.178 Preece *et al* (1999) investigated the performance of 36 volunteers on a wide range of tasks, including short-term and long-term memory, simple and choice reaction time (RT), and sustained attention, which, together, yielded a total of 15 dependent variables. These variables were grouped for combined analysis by multivariate analysis of variance into four sets: RT on attentional tasks (simple, choice and vigilance RT), speed on memory tasks, accuracy on memory tasks and accuracy on attentional tasks. Using a counterbalanced, crossover design, two exposure conditions – continuous and pulsed (217 Hz) 1 W, 915 MHz signals (simulating analogue and GSM mobile phone signals with mean power levels of 1 W and 0.125 W, respectively) – were compared to a no-exposure control condition. The signals were delivered through a simulated headset placed over the left ear with the antenna extending over the temporo-parietal scalp. Initial statistical analyses revealed a significant effect of exposure for RT on the attentional tasks. Follow-up analyses revealed a single statistically significant effect of exposure, which took the form of a shortening of RT (by 14 ms) during exposure to the analogue signal in the choice RT task, which required speeded discrimination between visual presentations of the words "yes" and "no". The effect was not accompanied by a reduction in the accuracy of responding, suggesting that it did not reflect a "speed–accuracy tradeoff". Exposure effects on RT in the two other attentional tasks showed trends in the same direction, but were of trivial magnitude (< 3 ms). Exposure effects for the three other groupings of dependent variables were far from statistical significance (minimum $p = 0.144$). However, non-significant trends towards shortened RT during exposure to the analogue signal were observed to varying degrees in all four of the memory tasks. The Expert Group reanalysed the most important parts of the data from Preece *et al* (1999). The reanalysis, in general, confirmed and indeed slightly strengthened the conclusions of Preece *et al*.

5.179 Preece *et al* (1999) conjectured that their findings were thermal in origin, and reflected the facilitatory effect of heating on synaptic transmission in cerebral cortex directly underlying the simulated headset. They argued that this explanation was consistent with their finding of an effect for the analogue but not the digital signal (the two signals differed in mean power by a factor of eight, and any heating effect would be proportional to power). They further argued that a local cortical effect was consistent with the task-specificity of their findings, as any effects on behaviour would be greatest for those tasks that depended most heavily on cognitive operations supported by the affected region of cortex.

5.180 Koivisto *et al* (2000) studied 48 volunteers, also with a wide range of cognitive tests (12 in total, providing 14 measures of RT and a similar number of accuracy measures). Their test battery included a two-choice RT task very similar to that employed by Preece *et al* (1999). A counterbalanced, crossover design was used to compare performance under conditions of no-exposure with performance during exposure to a 902 MHz GSM signal (modulated at 217 Hz) of 0.25 W mean power. The phone was positioned over the left ear with the antenna located over the posterior temporal scalp. Koivisto *et al* reported that three measures of RT – simple RT, the estimated time taken to perform a mental subtraction of two digits, and the average time taken to detect rarely occurring "targets" in a stream of visually presented letters (a "vigilance" task) – were shortened (by 9, 29 and 25 ms, respectively) to a statistically significant extent when the tasks were performed during exposure to the mobile phone signal. Accuracy scores did not indicate the presence of a speed–accuracy tradeoff; indeed, in the vigilance task errors of commission showed a small but statistically significant reduction during exposure. The effect of exposure on the choice RT task analogous to that employed by Preece *et al* (1999) was far from significant. Koivisto *et al* nonetheless considered their findings to be consistent with those of Preece *et al* (1999), noting that they too had failed to find an effect of GSM signals on a two-choice RT task. Koivisto *et al* argued that their own results suggested that exposure to the mobile phone signals had affected cortical regions, such as the inferior parietal cortex, that support sustained visual attention. Similarly to Preece *et al* (1999), they proposed that the mechanism underlying this effect may be thermal.

5.181 The Koivisto *et al* (2000) study can be criticised on the grounds that the data were subjected to an inappropriate form of statistical analysis. Fourteen measures of RT were considered, and the effect of exposure on each measure was assessed by a separate pairwise statistical test performed in the absence of any initial multivariate analysis that demonstrated a general exposure effect. Thus, across all the tests, the chance probability of obtaining a significant effect of exposure was considerably greater than the significance level ($p < 0.05$) that was employed to evaluate the outcome of each test. Indeed, according to the Bonferonni criterion (a standard method of correcting significance levels when multiple tests are performed), of the four "significant" findings reported, only RT in the vigilance task demonstrated a statistically significant exposure effect. That said, it is noteworthy that RT on 11 of the 14 measures was numerically, if not significantly, shorter during exposure. These trends offer some reassurance that a genuine exposure effect was operating, which would be strengthened if it was known that the 14 measures were not intercorrelated to any great extent; the relevant data were not, however, reported.

5.182 In a second study, Koivisto *et al* (*in press*) employed a very similar experimental design to investigate the effects of mobile phone signals (dosimetry as in their previous study) on the performance of 48 subjects in an "n-back" "working memory" task. In this task, subjects are presented with a sequence of characters (letters in the present case) and must respond discriminatively according to whether a letter is a "target" or "non-target". Targets comprise items repeated after a designated number of trials. In the easiest case, targets are designated as items that repeat on the immediately succeeding trial ("1-back" condition). The load on working memory is manipulated by increasing the number of trials that intervene between the first and

second presentations. In Koivisto *et al* (*in press*), 1-back, 2-back and 3-back conditions were employed. Accuracy and RT measures were obtained for both target and non-targets and analysed with analysis of variance. No exposure effects were found for accuracy. For target RT only, a reliable interaction effect was obtained between load (1-back, 2-back, or 3-back) and exposure condition. Pairwise comparisons revealed a significant ($p < 0.05$) RT reduction of 36 ms in the 3-back condition during exposure. Non-significant *increases* in RT were observed during exposure for the 1-back and 2-back conditions (11 ms and 21 ms, respectively).

5.183 These findings are consistent with those of Preece *et al* (1999) and Koivisto *et al* (2000) in suggesting that exposure to mobile phone signals facilitates RT during some cognitive tests. The findings further suggest that these facilitatory effects increase along with the cognitive demands of the test. Two aspects of the Koivisto *et al* (*in press*) study suggest, however, that the findings should be interpreted with a degree of caution. First, it is puzzling why exposure should have tended (albeit non-significantly) to increase RT in the 1-back and 2-back conditions. Second, the exposure effect in the 3-back condition was of only modest statistical significance.

5.184 Together, the findings of Preece *et al* (1999) and Koivisto *et al* (2000, *in press*) suggest that exposure to mobile phone signals at power levels within existing exposure guidelines has biological effects that are of sufficient magnitude to influence behaviour. Both groups conjectured that their findings reflected the effect of small temperature increases on synaptic transmission in the region of cerebral cortex directly under the headset antenna. An easily testable prediction of this account is that the tasks most sensitive to exposure to mobile phone signals should vary according to the position of the headset, and thus the cortical locus of the heating effect.

5.185 Considerably more work along the lines of the Preece *et al* (1999) and Koivisto *et al* (1999) studies is needed before it will be possible to evaluate fully the generality and significance of their findings. For example, it is currently unclear why Preece *et al* found effects only for analogue signals, whereas Koivisto *et al* were able to find effects with GSM signals. It is also unclear why the effects found in the studies of Preece *et al* and Koivisto *et al* (2000) involved different tasks. It is plausible that these disparities reflect differences in the signal powers and antenna positions employed in the two studies, and these variables should be among those investigated in future studies. As already noted, the reliability of some of the above findings is questionable because they were obtained from analysis of datasets containing numerous dependent variables. Future research conducted in light of these findings should, as much as is practical, employ a more hypothesis-driven approach to investigate exposure effects on relatively small numbers of variables.

5.186 Two further points should be made about the three studies described in this section. First, it should not be concluded that the findings of these studies indicate that the acute effects of exposure to mobile phone signals, when they are found, will invariably be beneficial to cognitive performance. This may be true for simple tasks of the kind on which Preece *et al* (1999) and Koivisto *et al* (2000, *in press*) were able to find exposure effects, although this result may not extend to more complex tasks which require co-ordination between several concurrently active cognitive operations (eg driving). In such cases, any change in the function of a single component operation may be detrimental for the functioning of the system as a whole.

5.187 Second, while further studies along the lines discussed above will be important in establishing whether the biological effects of mobile phone signals are indeed sufficient to cause short-term changes in cognitive performance, these studies will not directly address the question of whether long-term changes in cognitive function follow sustained exposure of the kind experienced by

mobile phone users. The relevance of such studies to the question of whether mobile phone use is detrimental to health is therefore limited.

Electroencephalogram (EEG)

5.188 The scalp-recorded EEG is a reflection of synchronous activity in relatively large populations of cortical neurons. The "spontaneous" EEG is conventionally divided into a number of frequency bands, the relative amounts of activity which depend upon the psychological state of the subject, and the nature of the cognitive function in which he or she is engaged. The functional significance of these different components of the normal, waking EEG is poorly understood. Thus, while a demonstration that mobile phone signals influenced these components would be indicative of a biological effect of such signals, interpretation of the effect would be uncertain. This is less so in the case of EEG patterns associated with sleep, which are well characterised and routinely used as indices of the different sleep stages that a typical healthy individual will move between during the night. There would also be little uncertainty in the interpretation of a change from a normal to a frankly pathological pattern of EEG activity, such as might be observed in epilepsy.

5.189 A measure of brain function closely related to the EEG is the "evoked" or "event-related" potential (ERP). ERPs are obtained by sampling the EEG time-locked to a reference event such as the presentation of a stimulus or the onset of a motor response, and averaging the samples together so as to obtain an electrical waveform that represents brain activity associated specifically with that class of event. ERPs are commonly used to study the timing and functional integrity of neural systems supporting sensory, cognitive and motor processing.

5.190 Laboratory studies investigating the effects of mobile phone signals on the spontaneous EEG in awake subjects have produced somewhat mixed results. For example, Reiser *et al* (1995) reported that exposure to GSM signals was associated with increases some 15 minutes later in the power of EEG frequencies of about 10 Hz and above (although this finding is of questionable statistical validity). Roschke and Mann (1997), however, were unable to detect any differences in EEG spectra related to exposure to GSM signals. A similar inconsistency appears to hold for the study of sleep EEG. Mann and Roschke (1996) reported that exposure to GSM-like signals reduced latency to sleep onset, and altered the abundance and spectral characteristics of REM sleep, although a subsequent study by the same group (Wagner *et al*, 1998) failed to replicate these findings. In a more recent study, however (Borbely *et al*, 1999), exposure to a "pseudo-GSM signal" (15 minute on/off cycles, 900 MHz, duty cycle of 87.5% rather than the 12.5% used in phone signals, and an estimated whole-body SAR of 1 W/kg) was associated with reduced waking after sleep onset and changes in EEG power spectra during the first of the night's episodes of non-REM sleep.

5.191 Krause *et al* (2000) studied the effects of GSM signals (dosimetry and general experimental design as in the studies of Koivisto *et al* (2000, *in press*) see paragraph 5.180) on "event-related" changes in the EEG recorded during the performance of a "memory scanning" task. Each trial involved the auditory presentation of four words (the "memory set"), followed after two seconds by the presentation of a "probe" word which matched an item from the memory set on 50% of trials; subjects were required to indicate whether or not the probe was a match. The principal finding was that the pattern of EEG changes (as indexed by changes in the power of four frequency bands between 4 Hz and 12 Hz) elicited by the probe word differed between exposure and no-exposure conditions. These differences were considered by Krause *et al* to be possible physiological correlates of the short-term effects of GSM signals on behaviour described by Koivisto *et al* (2000, *in press*). Unfortunately, Krause *et al* (2000) did not report measures of task performance. Thus it not possible to determine whether exposure facilitated RT in the memory

scanning task, as would be expected if the reported EEG changes are indeed a correlate of this effect.

5.192 In three studies, ERPs were investigated during exposure to GSM-like signals. In the first (Urban *et al*, 1998), visual sensory responses to checkerboard reversal were found to be unaffected during exposure. In two other studies (Eulitz *et al*, 1998; Freude *et al*, 1998) positive effects were reported. In the study of Eulitz *et al*, these took the form of the suppression of high frequency (18–30 Hz approximately) spectral power in ERP waveforms elicited by infrequent auditory "oddball" stimuli interspersed among a more frequent class of auditory stimulus. In Freude *et al*, the effect was a small reduction in the amplitude of response-related potentials in a visual monitoring task; no such effect was found in the potentials preceding spontaneous movements, nor were there any exposure effects on task performance.

5.193 Together, the findings from electrophysiological studies suggest that exposure to mobile phone signals influences brain function. The evidence is sufficiently substantial to warrant further investigation, notably with respect to the influence of GSM-like signals on sleep and on event-related EEG changes during the performance of cognitive tasks. It should be emphasised, however, that neither the biological nor the clinical significance of the findings described above is clear at present, and the relevance of the findings to the question of the safety of mobile phone technology is uncertain.

Conclusions from studies on brain function

5.194 *Together, the findings of Preece et al (1999) and Koivisto et al (2000, in press) from human laboratory studies of the acute effects of exposure to mobile phone signals suggest that exposure to mobile phone signals at exposure levels that fall within existing exposure guidelines have biological effects that are of sufficient magnitude to influence behaviour. The causal mechanism is unclear, but could include a small, localised heating effect.*

5.195 Human studies of cognitive performance and EEG have, however, yet to provide evidence directly relevant to the question of the safety of mobile phones in the long term. As already noted, the experimental designs employed thus far are not appropriate for this purpose, as they focus on the consequences of short-term exposure. To address the question of whether mobile phone use has long-lasting effects on measures of human brain activity or cognitive performance, it will be necessary to conduct laboratory-based studies on carefully matched groups of subjects who differ with respect to their history of exposure to mobile phone signals. A complementary approach would be to follow a group of new phone users over time so as to identify any changes in brain or cognitive function associated with cumulative exposure. Studies such as these should be a priority for future research.

Effects on the heart and blood pressure

5.196 As explained above (paragraphs 5.170–5.172), RF fields could, in principle, affect the cardiovascular system via a number of mechanisms. With normal use of a mobile phone, placed against the side of the head, direct influences on the human heart seem very unlikely. However, influences on the cardiovascular centres of the brainstem or the carotid body receptors are more conceivable.

5.197 Early reports from the former Soviet Union (see, for example, Drogichina *et al*, 1966; Sadčikova, 1974) implied that occupational exposure to RF radiation can directly or indirectly alter cardiovascular function. The most common observation was a reduction in blood pressure associated with either bradycardia or tachycardia (slowing or speeding of the heart). However, the general conclusion of a number of reviews of this literature is that most of the early studies

were poorly controlled and that the results may have reflected chance variation (Kaplan *et al*, 1971; Resnekov, 1981; Kristensen, 1989; Jauchem, 1997).

5.198 More recently, Braune *et al* (1998a) have reported acute effects on blood pressure in human volunteers exposed to a conventional GSM digital mobile phone positioned close to the right side of the head. After 35 minutes of exposure, heart rate, blood pressure and capillary perfusion were measured with the subject either supine or standing for 60 seconds. They found that the heart rate during these tests was slightly lower after exposure to RF radiation than following non-exposed control sessions, and both systolic and diastolic blood pressure were elevated by 5–10 mm of mercury. Since capillary perfusion (blood flow through capillaries of the hand) was decreased, the authors concluded that the effects on blood pressure were due to excessive vasoconstriction, caused, perhaps, by an increase in sympathetic activity originating in the brainstem.

5.199 This study has been criticised on the basis of both its design and the statistical analysis (Reid and Gettinby, 1998). In particular, the "placebo" (non-exposed) session preceded the test session for all subjects, and therefore the small cardiovascular changes might have resulted simply from the lengthy period of the experiment. Braune *et al* (1998b) argued that they chose not to randomise the sequences because of other evidence that effects of RF exposure could persist for some time. However, they should, then, have included a true control group in which testing was carried out during a *second* non-exposed session following the first placebo period.

Conclusions on heart and blood pressure studies

5.200 *There is, on the basis of published evidence, no basis for concern about effects of mobile phone use on the heart and circulation. However, this is a subject that merits more experimental work on human volunteers. In particular, we advise that a study similar to that of Braune et al (1998a) is carried out with larger numbers of subjects and appropriate control conditions.*

Mobile Phones and Driving

5.201 Mobile phones could have a detrimental effect on public health not only through the direct effects of exposure to electromagnetic radiation, but also indirectly by interfering with the phone user's ability to perform a concurrent task. Perhaps the most important, and certainly the most well-publicised, example of such interference involves the use of mobile phones while driving. These effects are considered by the Department of the Environment, Transport and the Regions to be sufficiently serious to warrant a major publicity campaign (DETR, 2000) aimed at dissuading drivers from using a mobile phone, especially one that is hand-held, when in control of a vehicle.

5.202 While it may seem obvious that using a hand-held mobile phone while driving will have negative consequences for road safety, it is perhaps less obvious that similar consequences may follow from the use of hands-free equipment. Experimental psychologists have, however, produced a wealth of evidence indicating that when mental (cognitive) tasks are performed concurrently, performance is often worse than when each task is performed alone. These "dual task" effects arise for a variety of reasons, chief among which are the need to switch or divide attention between the two tasks, and the interference that occurs when tasks compete for the same cognitive processes or mental representations.

5.203 In light of the psychological findings, it is likely that, in addition to the purely "peripheral" interference arising when a driver attempts both to operate a vehicle and to manipulate a

hand-held phone, there will also be sources of "central" interference that arise when the cognitive demands of a mobile phone conversation compete with those required for driving. In assessing the possible impact of mobile phones on road safety, it is important, therefore, to understand the relative magnitudes of these two sources of interference; there is little point in stressing the inadvisability of using a hand-held phone if the greater part of the associated risk is also present when hands-free equipment is used.

5.204 Evidence concerning the impact of mobile phones on driving ability is reviewed below in two sections. First, experimental studies are discussed in which the effects of phone use on driving have been assessed. Such studies allow the mechanisms by which phones interfere with driving to be elucidated, and the effects of different kinds of phone use to be compared. They provide, however, only indirect information about the actual impact of phone use on road safety. This issue is taken up in the second section, which discusses epidemiological studies that attempted to quantify the increased risk associated with the use of a mobile phone while driving. Finally, the implications of this evidence are discussed in relation to policy regarding the use of different kinds of phones in vehicles.

Experimental evidence for effects on driving

5.205 The impact of phone use on various aspects of driving performance has been investigated in a sizeable number of studies. Some studies investigated performance on laboratory tasks analogous to driving, such as a visuospatial tracking task (Strayer *et al*, 1999) or in a driving simulator (McKnight and McKnight,1993; Alm and Nilsson, 1994, 1995; Haigney, 2000); others employed real cars and road situations (Brown *et al*, 1969; Brookhuis *et al*, 1991; Lamble *et al*, 1999). Some studies limited their investigations to the effects of mobile phone conversations in a hands-free setting, sometimes employing phone tasks that differed with respect to "mental workload", eg "shadowing" (repeating back) words *versus* "generation" (producing a new word beginning with the last letter of the one just heard). Other studies directly contrasted the effects of using hand-held *versus* a hands-free sets (Brookhuis *et al*, 1991; Strayer *et al*, 1999; Haigney, 2000); or compared the use of hands-free phone to a task requiring some manual control of some other device, eg entering numbers on a keypad (Lamble *et al*, 1999) or a keypad task and tuning a radio (McKnight and McKnight, 1993).

5.206 The results of these experimental studies are consistent and easily summarised. Relative to either a "no-conversation" condition (or, in the case of Strayer *et al*, 1999, listening to a car radio), engaging in a mobile phone conversation had a detrimental effect on driving performance as measured by such indices as the time taken to react to an imperative stimulus (Alm and Nilsson, 1994; Strayer *et al*, 1999) or a change in the speed of a leading car (Brookhuis *et al*, 1991; Lamble *et al*, 1991; Alm and Nilsson, 1995); failure to react to a potentially dangerous road situation (McKnight and McKnight, 1993); speed adaptation (Haigney, 2000; interestingly, this measure was found to exhibit a "carry-over" effect, in that adaptation continued to be affected during a 2.5 minute period after the call was terminated); maintaining a safe distance from a leading car (Haigney, 2000); and the ability to control a car in "non-routine" situations (Brown *et al*, 1969). Although the detrimental effect of mobile phone use increased with the mental workload imposed by the conversation (McKnight and McKnight, 1993; Strayer *et al*, 1999), an effect was evident nonetheless with "casual" conversations (McKnight and McKnight, 1993), even when the control condition consisted of listening to a car radio (Strayer *et al*, 1999). Furthermore, in one simulator study (Alm and Nilsson, 1994) it was found that the detrimental effects of a phone conversation on reaction time were greater when driving conditions were relatively undemanding as opposed to when they were taxing. The findings of two studies (McKnight and McKnight, 1993; Alm and Nilsson, 1995) suggest that the negative effects of a phone conversation on driving increase with age. This finding is not unexpected given evidence

of a general age-related decline in ability to divide attention (see, for example, Gottlob and Madden, 1999).

5.207 Importantly, the foregoing studies suggest that the "central" effects of mobile phone use on driving are equivalent for hands-free and hand-held operation. Two studies that directly compared these modes of operation (Strayer *et al*, 1999; Haigney, 2000) reported no difference, albeit, in the case of Strayer *et al*, with a laboratory "tracking" task somewhat removed from driving (it is noteworthy that the Haigney study also failed to find any difference between hand-held and hands-free operation even when gear changes were made manually rather than automatically). In a third study (Brookhuis *et al*, 1991) the effects of the two modes of phone operation were compared during real driving (although with only six subjects in each group), and were found to have equally detrimental effects on speed of reaction to the slowing of a leading car. However, dialling with a hand-held set was associated with poor control over steering, especially when driving in city traffic. In the studies of McKnight and McKnight (1993) and Lamble *et al* (1999) the effects of conversation via a hand-held set were compared with those from tasks involving a combined cognitive and manual component (tuning a radio or keying in numbers); in both cases, merely engaging in a demanding conversation had effects equivalent to those of the combined task. According to Lamble *et al* (1999), the effect they observed on braking time was approximately three times that found for drivers with a blood alcohol level of 0.05% (the limit for many European countries; the UK limit is 0.08%).

Conclusions on experimental evidence for effects on driving

5.208 *There is strong experimental evidence that engaging in a mobile phone conversation impairs drivers' ability to react to potentially hazardous road situations. The impairment appears to be greater than that associated with merely listening to a radio or engaging in a relatively "automatic" task such as repeating back words heard over the phone; is evident during a "casual" conversation; increases along with the mental workload imposed by the conversation; is greater in elderly drivers; and is unaffected by mode of phone use (hand-held versus hands-free). There is less evidence as to whether aspects of driving other than speed or accuracy of reaction to changing road circumstances differ according to mode of phone operation. Consistent with what might be expected on the basis of common experience, one study found that placing a call on a hand-held set is associated with a transient impairment in the basic control of the vehicle. The extent to which this "peripheral" effect adds to the risk posed by the more sustained "central" effects that are shared by hand-held and hands-free operation appears to be unknown at present. It should be noted that none of the studies reviewed above compared the effects on driving performance of phone use to the effects caused by conversing with a passenger. Thus it remains to be established whether an in-car conversation that places a cognitive load on the driver equivalent to that imposed by a mobile phone call has similarly detrimental effects on performance. There are, however, good reasons to suppose that the effects of an in-car conversation will be less than those associated with the use of a phone. In contrast to the individual on the other end of a phone call, a passenger can monitor the road situation and "pace" the interaction according to circumstances (for example, suspending conversation during an overtaking manoeuvre). In addition, a passenger can act as a second "pair of eyes", alerting the driver to potential hazards.*

Epidemiological evidence for effects on driving

5.209 There are few systematic studies of the effects of mobile phone use on road traffic accident rates. Violanti and Marshall (1996) conducted a questionnaire survey of 100 randomly selected drivers who had been involved in a "reportable" road traffic accident during a 12 month period, and 100 geographically matched control drivers who had been accident-free for at least ten years.

Fourteen of the drivers who consented to participate were mobile phone users. After controlling for factors such as years of driving experience, Violanti and Marshall reported that there was a significant association between the likelihood of involvement in an accident and the use of a mobile phone for more than 50 minutes per month. These findings are, however, of questionable significance with respect to whether in-car phone use is detrimental to road safety; not only was the number of cases available for analysis very small, but the critical variable – monthly use of a mobile phone – provides, at most, a highly indirect index of in-car usage. The latter criticism also holds for the study of Dreyer *et al* (1999), who reported an association between the amount of mobile phone usage and road accident mortality.

5.210 Violanti (1997, 1998) examined accident records from the state of Oklahoma in the USA, where traffic police routinely record whether a mobile phone was present in a car involved in an accident, as well as whether the phone was reported to be in use when the accident occurred. In the first study (Violanti, 1997) rate-ratios were assessed for phone use and presence with respect to a number of accident characteristics. Use and presence were associated with a significantly elevated risk for accidents when these involved, among other things, "driver inattention", driving in cities, running off the road, overturned vehicles, and injuries and fatalities. Phone users most at risk from fatalities were young males, but otherwise there was a trend for phone-related risks to increase with age. No steps were taken in the analyses as presented to remove the effects of any potential confounding variables that might have acted to exaggerate differences between phone users and non-users. The second study (Violanti, 1998) focussed exclusively on fatal accidents, addressing the question of whether, among drivers who had experienced an accident, the probability of a fatal outcome was influenced by the use or presence in the car of a mobile phone. Logistic regression was used in an attempt to remove the confounding effects of such variables as age and involvement with alcohol or drugs, as well as, puzzlingly, accident characteristics such as "unsafe speed", and "driver inattention". It was estimated that the likelihood of a fatality, given involvement in an accident, increased by a factor of about nine if a phone was in use, and was doubled if a phone was merely present in the car.

5.211 These two studies suggest there is a strong statistical association between the presence and use of mobile phones in cars and the likelihood of a serious traffic accident. There are, however, possible reasons for this association other than a detrimental effect of phone use on driving. It might be, for example, that drivers who carry or use a mobile phone differ from those who do not in ways that make them more likely to be involved in an accident. The finding that the mere presence of a phone in a car was associated with elevated risk is consistent with this possibility, although the finding could also reflect an underestimation of the proportion of accidents in which the phone was actually in use.

5.212 Redelmeir and Tibshirani (1997) employed a "case–crossover" design, in which each participant served as his or her own control. They investigated a group of 699 drivers who had reported involvement in a minor traffic accident, addressing the question whether these individuals were more likely to have been using a phone in the period leading up to their accident than they were during a comparable "control" period in the recent past. A correction factor was employed to allow for the fact that participants may not have driven during a control period. Redelmeir and Tibshirani found that *relative risk* increased with the proximity of phone use to the time of the accident. For example, use of a phone within 10 minutes of the accident was associated with an approximate quadrupling of risk, whereas phone use more than 15 minutes before the accident did not carry a significant risk. These findings were robust with respect to the choice of control period (eg day before, same day a week previously, etc) and were similar for a subgroup of drivers who reported (when questioned some two years afterwards) that they had definitely driven during the control period, and therefore for whom no correction for "driving intermittency" was required. Although numbers were small (N = 129 for hand-held;

41 for hands-free), there was no evidence that risk differed according to mode of phone operation; indeed, there was a non-significant trend for hands-free operation to carry the greater risk.

5.213 The findings of Redelmeir and Tibshirani (1997) converge with those of Violanti (1997, 1998) to suggest that the use of a mobile phone while driving is associated with an increased risk of an accident. While the study does not suffer from the problems inherent to comparisons involving different groups of individuals, it nonetheless has a number of limitations, as indeed discussed by the investigators. Chief among these is that the findings do not permit the conclusion that the association between phone use and traffic accidents is causal. For example, it might be that the critical variable is the level of stress or time-pressure experienced by the driver, which is associated with an increased probability of making a phone call while on the move and an increased probability of an accident. Among other limitations of the study are its use of a self-selected group of drivers (of 1000 or so candidates, about 300 refused to participate), its focus on minor accidents, and the need, in the overall analysis, to rely upon a correction factor to allow for "driving intermittency" during control periods. These limitations notwithstanding, the study provides persuasive evidence that the use of a mobile phone while driving has a detrimental effect on road safety.

Conclusions on epidemiological evidence for effects on driving

5.214 *Experimental studies provide compelling evidence that engaging in a mobile phone conversation impairs driving performance. Consistent with this evidence, epidemiological research points to an association between mobile phone use while driving and an increased risk of involvement in an accident. Together, these two sources of evidence indicate that current concerns about the impact of mobile phones on road safety are well founded. As already noted, however, current experimental evidence suggests there is little or no justification for the assumption that the detrimental effects of phone use on driving are ameliorated by hands-free operation, a conclusion supported by the limited epidemiological evidence relevant to this question (Redelmeir and Tibshirani, 1997). There is therefore no strong empirical justification at present for the enactment of a policy or legislation that differentiates between the use of hand-held and hands-free phone sets in motor vehicles. While an argument might be made for focussing legislation on the more detectable of these two modes of use – it is of course much easier to detect the use of a hand-held set than a hands-free set – such an approach runs the risk of seeming to condone, or at least to tolerate, the use of hands-free phones.*

Epidemiological Studies on General Health Effects

5.215 Epidemiology is the branch of science that is concerned with the distribution and determinants of disease in human populations. As such, it provides the most direct evidence on whether and to what extent suspected environmental hazards cause disease. This evidence comes mainly from investigations that compare risks of disease in different groups of people according to their exposure to a suspected hazard. Usually, the results are expressed in terms of an estimate of relative risk, ie the multiplier by which risk is higher in people exposed to the hazard than in others who are unexposed or exposed at a lower level. For example, a relative risk of two for brain cancer in exposed compared with unexposed people would imply that the rate of brain cancer was twice as high in the former as in the latter group. It should be noted that the impact of a raised relative risk will depend on the underlying incidence of the disease to which it applies. A doubling in the frequency of a very rare disease represents a much smaller absolute increase in risk than a doubling of a common disorder.

5.216 The practical and ethical constraints on research in human populations mean that all epidemiological studies have limitations that must be taken into account in the interpretation of their findings. There may be deficiencies in their design or execution, often unavoidable, which tend spuriously to inflate or diminish estimates of risk – an effect known as "bias". Practical restrictions on the numbers of people who can be studied mean that epidemiological studies are subject to statistical uncertainty, and may produce misleading results simply by chance (statistical techniques can be used to quantify the extent of this uncertainty). Even where risk is genuinely elevated, this does not necessarily indicate a causal relationship between the hazard and the disease. There may be one or more "confounding factors" that are associated with exposure to the hazard and that independently influence the chance of occurrence of a disease. For example, an increased risk of lung cancer in people living near to a radio mast might reflect the fact that on average they smoke more than people living elsewhere, rather than an effect of radiation emitted by the mast. In general, the closer the relative risk is to unity, the more difficult it is to rule out bias and confounding as explanations for an association between exposure and disease.

5.217 The scope for bias, chance effects and confounding is such that generally little weight can be given to a single epidemiological study in isolation and, when evaluating epidemiological evidence, it is important to base conclusions on the totality of all relevant studies. Sometimes, several studies that address the same problem can produce quite different estimates of risk. As well as differences in the impact of bias, chance and confounding, such discrepancies may reflect differences in the extent or pattern of exposure that was examined or the presence of other factors that modify the body's response to a hazard.

5.218 To date, few epidemiological studies have directly examined the relationship of mobile phones to morbidity or mortality, and none has explored the effects of exposure to RF radiation from base stations. However, rather more information is available regarding exposure to other types of RF radiation – for example, in radar mechanics and radio operators, and from residence near broadcasting towers and masts. These exposures differ in frequency, dose and other characteristics from those produced by mobile phones and base stations, so they give only an indirect indication of the possible risks from mobile phone technology. Nevertheless, they are relevant and worth consideration.

5.219 Four main types of study have been used in this area of research – cohort studies, case–control studies, cross-sectional surveys of morbidity, and cross-sectional, "ecological" comparisons of mortality or cancer incidence between populations. In a cohort study, individuals who have been exposed to a known or suspected hazard are identified and their subsequent disease incidence or mortality is assessed over a period of follow-up. This is then compared with the corresponding rate of disease or death in a control group who have been unexposed or only exposed at a lower level. A case–control study starts with patients who have developed a disease (cases) and compares their past exposure to known or suspected causes with that of suitably chosen controls who do not have the disease. Cross-sectional surveys of morbidity focus on a representative sample of a population (eg residents of a specified area), and collect information from individuals in the sample about their disease experience and about current and past exposures to known or suspected causes of disease. The statistical association between exposure and disease is then examined. Ecological analyses of mortality and cancer incidence assess the frequency of death or of new cases of cancer in different populations (eg in occupational groups or residents of defined geographical areas) over a period (say a few years), and relate this to differences in the levels of exposure of the whole population to known and suspected hazards. For example, death rates might be compared in people living close to and further away from a television mast. It should be noted that the first three methods assess disease and exposure in individuals, whereas the ecological method relates to populations. Even where disease rates are consistently high in heavily exposed populations, it does not necessarily follow that the individuals within those

populations who suffer the disease have themselves been exposed. In general, the ecological method, because of the lack of individual data, is the least robust of the four study designs, and often, although by no means always, the cohort method is the most rigorous.

Studies of people using mobile phones

5.220 Widespread use of mobile phones is a recent phenomenon, and as yet few epidemiological studies have looked directly at whether there are associated risks of illness or death.

Mortality and cancer incidence

5.221 One investigation has examined mortality among customers of a large mobile phone operator in the USA (Rothman *et al*, 1996). It covered some 250,000 phone users, who were followed for one year. During this time, the overall death rate was similar in people using hand-held phones and in users of other mobile phones that did not have an antenna in the handset, and therefore gave lower exposures to RF radiation. For those customers who had been listed as continuous users for at least three years, overall mortality was slightly lower in the hand-held phone users than the other mobile phone users, but the difference was not statistically significant (relative risk = 0.86). Numbers of brain tumour and leukaemia deaths were small and showed no substantial indication of increasing risk with number of minutes of hand-held phone use per day, or with years of hand-held phone use (Dreyer *et al*, 1999). No data were reported on whether phones used analogue or digital signals (at this time, in the USA most mobile phone networks used analogue signals). If mobile phones do affect mortality, the impact is likely to be on only certain specific causes of death. Also, any increase in diseases such as cancer may not be manifest until many years after people are first exposed to a hazard. *Therefore, although no significant differences in mortality were demonstrated between the two exposure groups, the conclusions that can be drawn from this report are limited, and it does not rule out important effects.*

5.222 In a case–control study in Sweden, patients with brain tumours were asked about various aspects of their life including their use of mobile phones, and the findings were compared with those in controls selected from the general population (Hardell *et al*, 1999). Overall, the risk of brain tumours did not appear to be elevated in people who used mobile phones, either analogue or digital, even if their use was relatively heavy. In a series of subsidiary analyses, an association was observed between tumours in the temporal and occipital lobes of the brain and reported use of analogue phones on the same side of the head (regardless of whether that was to the left or right). However, this was not statistically significant, and could easily have occurred by chance. Interpretation of this study is complicated because it failed to identify a substantial number of brain tumour patients who were eligible for inclusion according to the reported entry criteria (Ahlbom and Feychting, 1999), and in the absence of an explanation for this under-ascertainment, it is unclear whether important bias could have resulted. Also, as in the study by Rothman *et al*, an effect of exposure that was delayed for ten or more years would not have been apparent.

Other health effects

5.223 In an attempt to identify other, more immediate, adverse health effects that might be associated with the use of mobile phones, Hocking (1998) placed a notice in a medical journal in Australia. From this and subsequent publicity, he recruited 40 individuals with symptoms which they related to using mobile phones. These symptoms were mainly in the head, and included pain, unpleasant warmth or heating, blurring of vision and deafness or vertigo. Most started within five minutes of beginning a call, but some built up over the course of the day. A perceived temporal association between use of a phone and the development of symptoms does not necessarily imply that RF radiation is responsible. Rather, the findings of Hocking's study should be regarded as an

indication of the types of symptom that users claim, and which might merit more rigorous investigation to determine whether there is any causal relationship to mobile phone use. It is notable, however, that there were no reports of epileptic seizures triggered by phone use, although this has been alleged to occur in relation to exposures from base stations.

5.224 Larger-scale data on self-reported, subjective symptoms are available from a cross-sectional survey of some 11,000 mobile phone users in Sweden and Norway (Hansson Mild *et al*, 1998). A postal questionnaire was used to collect information about various symptoms including fatigue, headache and warmth behind and on the ear. Of the participants, 13% in Sweden and 30% in Norway reported the occurrence of at least one symptom, which they themselves related to mobile phone use. For both analogue and digital phones, the prevalence of reported symptoms increased with minutes per day of phone use. The proportion of GSM phone users reporting a symptom was rather lower than in other groups. The marked difference in the prevalence of complaints between Sweden and Norway has a number of possible explanations. It is well known that somatic complaints can be influenced by psychosocial circumstances. For example, back pain is more likely to be reported by people who are depressed or dissatisfied in their work. Furthermore, at the time of the survey there had been much publicity in Scandinavia about possible adverse health effects of electromagnetic fields. In these circumstances, it would not be surprising if people who used mobile phones extensively were more aware of and troubled by minor symptoms, and more likely to report them when questioned. Thus as in the Australian study, the various symptoms reported by the users of mobile phones cannot necessarily be attributed to RF radiation. To address this question, further research is needed with a different study design (see paragraphs 5.258–5.260).

Exposure to RF radiation through work and hobbies

5.225 A number of epidemiological investigations have examined the risk of illness or death in people potentially exposed to RF radiation through their work or hobbies, and these have been the subject of several previous reviews (EC, 1996; Elwood, 1999; Moulder *et al*, 1999; Royal Society of Canada, 1999). The diseases most often studied have been lymphatic and haematopoietic cancers (including lymphoma and leukaemia) and brain cancer.

Cancer

5.226 Table 5.19 summarises the investigations that provide information about lymphatic and haematopoietic cancers. The study by Szmigielski (1996) stands out in suggesting a more than six-fold elevation in the risk of these diseases among Polish military personnel with occupational exposure to RF radiation. However, Szmigelski's report is unsatisfactory, and can be given little, if any, weight. In particular, it appears that the exposures of cancer cases were ascertained from a different source (medical records) from those of the study population as a whole (provided by safety staff), and this could seriously have biased risk estimates. Also, the statistical methods are not adequately described, and certain important data are missing from the report.

5.227 If this investigation is discounted, only one study shows a statistically significant increase in risk – of leukaemia in Norwegian electrical workers (Tynes *et al*, 1992). In some of the other studies, risks were also elevated, although not to the point of statistical significance. These elevations of risk were either for leukaemia or for lymphatic and haematopoietic cancers as a group. (This grouping is often used in epidemiological studies, partly because misclassification of cases may occur when more specific diagnostic categories are employed. However, no carcinogen has yet been identified that causes all of the cancers in the group.) In most of the studies, the index of exposure to radiation was relatively crude and non-specific, and the workers may have experienced other confounding exposures. Thus, the observed increases in risk could have been due to chance or to factors other than RF radiation. At the same time, however, because of their low statistical power and the heterogeneity of the exposures examined, the absence of a clear and

Table 5.19 Epidemiological studies of lymphatic and haematopoietic cancer in people potentially exposed to RF radiation through work or hobbies

Type of study	Study population	Exposure condition	Disease outcome	Number of exposed cases	Estimated relative risk (with 95% CI)*	References
Cohort	Radar technicians in US Navy	Occupations with higher exposure to RF radiation (radar)	Death from lymphatic or haematopoietic cancer	26	1.18	Robinette *et al*, 1980
Cross-sectional analysis of proportional mortality	Men age 20+ years in Washington State, USA	Radio and telegraph operators Radio and television repairmen	Death from lymphatic or haematopoietic cancer	15 12	1.37 1.27	Milham, 1985
Cohort	Amateur radio operators in California and Washington State, USA	Amateur radio operators	Death from lymphatic or haematopoietic cancer	89	1.23 (0.99–1.52)	Milham, 1988
Cohort	White male enlisted men in US Navy	Radiomen Aviation electronics technician	Non-Hodgkin's lymphoma	2 1	0.6 (0.1–2.0) 0.4 (0.0–2.2)	Garland *et al*, 1988
Case–control	Men aged 20+ years in New Zealand	Radio and television repairmen	Leukaemia	2	7.9 (2.2–28.0)	Pearce and Fraser, 1989
Cohort	White male enlisted men in US Navy	Radiomen Electronics technician	Leukaemia	4 5	1.1 (0.3–2.8) 1.1 (0.4–2.6)	Garland *et al*, 1990
Cohort	Norwegian electrical workers	Occupations with potential exposure to RF radiation	Leukaemia	9	2.85 (1.30–5.41)	Tynes *et al*, 1992
Cohort	Norwegian female radio and telegraph operators	Radio and telegraph operators	Leukaemia Lymphoma	2 5	1.1 (0.1–4.1) 1.3 (0.4–2.9)	Tynes *et al*, 1996
Cohort	Polish military personnel aged 20–59 years	Occupational exposure to RF radiation	Lymphatic and haematopoietic cancer	Not given	6.31 (3.12–14.32)	Szmigielski, 1996
Cohort	Female employees in an Italian plastics factory	Exposure to RF radiation through work in a dielectric heat sealing department	Death from leukaemia	1	5.0	Lagorio *et al*, 1997
Cohort	Men and women employed in the design, manufacture and testing of wireless devices	Work in occupations with moderate or high peak exposures to RF radiation	Death from lymphatic or haematopoetic cancer	20	0.54 (0.33–0.83)	Morgan *et al*, 2000

*Confidence intervals, where shown, are as calculated by the authors

Table 5.20 Epidemiological studies of brain cancer in people partially exposed to RF radiation through work or hobbies

Type of study	Study population	Exposure condition	Disease outcome	Number of exposed cases	Estimated relative risk (with 95% CI)*	References
Cross-sectional analysis of proportional mortality	Man aged 20+ years in Washington State, USA	Radio and telegraph operators Radio and television repairmen	Death from brain cancer	1 2	0.38 0.59	Milham, 1985
Case–control	White men aged 30+ years from three areas of USA	Ever worked in a job with likely exposure to RF radiation	Death from brain cancer	69	1.6 (1.0–2.4)	Thomas *et al*, 1987
Cohort	Amateur radio operators in California and Washington State, USA	Amateur radio operators	Death from brain cancer	29	1.39 (0.93–2.00)	Milham, 1988
Cohort	Norwegian electrical workers	Occupations with potential exposure to RF radiation	Brain tumours	3	0.61 (0.13–1.78)	Tynes *et al*, 1992
Cohort	Norwegian female radio and telegraph operators	Radio and telegraph operators	Brain tumours	5	1.0 (0.3–2.3)	Tynes *et al*, 1996
Cohort	Polish military personnel aged 20–59 years	Occupational exposure to RF radiation	Tumours of the nervous system and brain	Not given	1.91 (1.08–3.47)	Szmigielski, 1996
Case–control	Male personnel in US Air Force	Potential exposure to RF radiation	Brain tumours	94	1.39 (1.01–1.90)	Grayson, 1996
Cohort	Female employees in an Italian plastics factory	Exposure to RF radiation through work in a dielectric heat sealing department	Death from brain cancer	1	10.0	Lagorio *et al*, 1997
Cohort	Men and women employed in the design, manufacture and testing of wireless devices	Work in occupations with moderate or high peak exposure to RF radiation	Deaths from cancers of the nervous system and brain	7	0.53 (0.21–1.09)	Morgan *et al*, 2000

*Confidence intervals, where shown, are as calculated by the authors

consistent elevation of risk in these studies cannot be taken as strong evidence against an association with specific types of RF radiation.

5.228 Where several epidemiological studies have addressed the same question, each individually with low statistical power, it is sometimes possible to draw firmer conclusions by combining their findings in a formal statistical "meta-analysis". However, this approach is not appropriate for the studies listed in Table 5.19 because they are too disparate.

5.229 Table 5.20 lists the studies that give information on the risk of brain tumours. Again, for reasons already stated, little weight can be given to the report by Szmigielski (1996). Apart from this, two investigations have found a statistically significant elevation of risk, both of them case–control studies.

5.230 The first (Thomas *et al*, 1987) recruited cases from the general population of three areas of the USA, and classified exposure on the basis of job titles as reported by the subjects' next of kin. The association with exposure to RF radiation was confined to jobs involving the design, manufacture, installation and maintenance of electrical and electronic equipment, leading the authors to suggest that some aspect of work other than RF radiation might be responsible. The aetiology of brain tumours, however, is largely unknown, so it is uncertain whether these occupations have relevant exposures in common.

5.231 The second study (Grayson, 1996) focussed on male personnel in the US Air Force, with exposures assessed from occupational histories. Although risk was significantly higher in men exposed to RF radiation than in those classed as unexposed, the elevation was modest even in those thought to have the heaviest exposures.

5.232 Other studies of brain cancer have given inconsistent results. As for haematopoietic and lymphatic cancer, the results overall do not indicate an increased risk of brain tumours from RF radiation, but because of various limitations neither do they provide strong reassurance that there is no hazard. These limitations include poor, often highly indirect, assessment of RF exposure and low statistical power.

5.233 Data on other types of cancer are more sparse and although some have suggested increased risks from RF exposure, their limitations are such that these findings should not be a cause for concern.

5.234 In a cohort study of Norwegian female radio and telegraph operators, Tynes *et al* (1996) found a relative risk of 1.5 for breast cancer (95% confidence interval 1.1–2.0). However, no relationship to RF radiation was apparent in a large case–control study of female breast cancer in the USA that used occupational information obtained from death certificates (Cantor *et al*, 1995), or in a cohort study of workers employed in the design, manufacture and testing of wireless devices in the USA (Morgan *et al*, 2000). However, this study does not give information directly on mobile phone use or exposures occurring from such use (Owen, 2000).

5.235 A cluster of six men with testicular cancer has been reported in a population of US police officers who used hand-held radar guns (Davis and Mostofi, 1993). However, in the only epidemiological study to examine the relationship between testicular cancer and work with radar equipment (Hayes *et al*, 1990), results were inconclusive: the risk of testicular cancer was significantly elevated in men who reported occupational exposure to RF radiation, but not when the radiation exposures were inferred from job titles by an occupational hygienist. This inconsistency between the results from the two methods of assessment raises the possibility that cases recalled their

exposures more completely than controls, or overestimated their exposures, leading to a biased risk estimate.

5.236 In a study in the USA, a raised risk of uveal melanoma was found in men with self-reported exposure to RF or radar (Holly *et al*, 1996), but RF radiation has not been examined in other published studies of this tumour.

5.237 *In summary, the overall balance of evidence from epidemiological occupational studies does not indicate that RF radiation affects the risk of cancer in people. However, the types of exposure investigated have varied between studies and are not identical to those associated with mobile phone technology. Also, many of the studies have had low statistical power and some have suffered from methodological deficiencies. Therefore, the absence of consistently positive findings does not establish firmly that RF radiation from mobile phones carries no important risk of cancer.*

Health outcomes other than cancer

5.238 Although cancer has been the main health outcome studied in relation to work with RF radiation, several cohort studies of occupational groups exposed to RF radiation have also examined non-cancer mortality and in some instances morbidity (see, for example, Robinette *et al*, 1980; Muhm, 1992). These do not provide any overall evidence of hazard.

5.239 In addition, several case–control studies have explored the risk of adverse outcomes of pregnancy in physiotherapists using microwaves in the RF range to treat their patients. One such study found a significantly raised risk of spontaneous abortion (miscarriage) in physiotherapists who reported exposure during the six months before and three months after becoming pregnant (relative risk 1.28, 95% confidence interval 1.02–1.59), and a higher risk in those with more frequent exposure (Ouellet-Hellstrom and Stewart, 1993), although with a relatively low response rate to the questionnaire that was used to collect information. No corresponding association was found with use of short-wave diathermy. Overall, however, studies of pregnancy in physiotherapists have not supported a relation of microwave exposure with miscarriage or other adverse outcomes (Kallén *et al*, 1982; Taskinen *et al*, 1990; Royal Society of Canada, 1999).

5.240 Despite this lack of evidence for health risks resulting from the exposure of workers to RF radiation, it would be sensible to set in place a long-term follow-up of workers who are occupationally exposed to RF radiation at relatively high levels. **We recommend that a register of occupationally exposed workers be established and that cancer risks and mortality be examined to determine whether there are any harmful effects. If any adverse effects of exposure to RF radiation are identified then the Health and Safety Executive should establish a system of health surveillance.**

Residence near radio and television transmitters

5.241 The incidence of cancer in people living near to radio or television transmitters has been examined in studies from the USA, Britain and Australia.

5.242 Selvin *et al* (1992) looked for clustering of childhood leukaemia, lymphoma and brain cancer within 3.5 km of a microwave tower in San Francisco. The main purpose of their investigation was to compare methods of statistical analysis, and they did not adjust closely for potential confounding factors. However, they found no evidence of any excess incidence in the study area.

5.243 In contrast, a case–control study in Hawaii suggested an approximate doubling in the occurrence of childhood leukaemia within 4.2 km of a group of radio masts (Maskarinec *et al*, 1994). The

number of exposed cases was small, however, and the excess was far from statistically significant. Furthermore, the investigation had been prompted by a perceived local excess of the disease, and in this circumstance it is more difficult to rule out the play of chance. In the same way that some communities can have a disproportionate excess of lottery winners purely by chance, a cancer can occur more frequently in a small geographical area (and will then be noticed to be common there) without there being a local cause for the phenomenon.

5.244 Concerns about an apparent excess of leukaemia and lymphoma were also the trigger for a geographical analysis of cancer incidence in the neighbourhood of a television and radio transmitter in Sutton Coldfield, England (Dolk *et al*, 1997a). The investigation confirmed that there had been an increased incidence of leukaemia within 2 km of the transmitter, the relative risk being approximately 1.8 in comparison with the regional population. None of the other cancers analysed apart from bladder cancer (relative risk 1.5), showed a statistically significant excess near to the transmitter. The authors recognised that despite their statistical significance, these findings could easily have occurred by chance, and therefore carried out a similar analysis for people living close to other high power radio and television transmitters in Britain (Dolk *et al*, 1997b). This showed no excess of leukaemia within 2 km of the transmitters. Rates of bladder cancer were marginally elevated within a 10 km radius (relative risk 1.09), but did not decline progressively with distance from the transmitters as might be expected if RF radiation were responsible.

5.245 In Australia, Hocking *et al* (1996) compared rates of leukaemia and brain tumours in three municipalities of Sydney surrounding television masts and six others at a further distance from the masts. The findings for brain tumours were unremarkable, but there was an approximate 60% excess of leukaemia among children from the three areas close to the towers. Subsequently, McKenzie *et al* (1998) explored this pattern of leukaemia incidence further with an expanded control area. The excess incidence was found to be limited to only one of the three municipalities surrounding the masts, suggesting that chance or some local factor other than RF radiation was responsible.

5.246 In addition to these investigations published in scientific journals, some reviews refer to studies of people living near a military microwave generator–detector system in Latvia, and of staff and their dependants in American embassies in Eastern Europe who may have been exposed to microwave radiation beamed into the embassies (Goldsmith, 1995; Repacholi, 1998). However, these have not been published in the peer-reviewed literature, and we have not been able to obtain sufficiently detailed descriptions of these investigations to evaluate them.

5.247 The studies to date that have looked at cancer incidence in relation to residence near broadcasting facilities have major limitations, which weaken the conclusions that can be drawn from them. The analyses have not been based on measured levels of radiation. Distance from a broadcasting tower has been taken as a proxy for exposure, but no account has been taken of ground reflections and signal reductions by buildings, vegetation and undulations, which may alter actual exposure considerably. The studies have been based on cancer and exposure data for populations not individuals, with the associated weaknesses of "ecological" studies (see paragraph 5.219). Personal exposures will vary according to how much time people spend at home, whether they are indoors or outdoors, the other sources of RF radiation in and near to their homes, and their levels of exposure at work, when travelling, and from mobile phone use. None of these has been taken into account. Furthermore, the studies have analysed risks in relation to place of residence at the time of cancer incidence or death, but if RF radiation does cause cancer, the relevant exposure may well be years or even decades before the disease becomes manifest. Thus while the balance of evidence from such studies does not indicate a hazard – and where increased rates of

disease have been found they could have occurred by chance or as a consequence of unrecognised confounding factors – the findings do not provide strong evidence against a hazard.

Conclusions from epidemiological studies

5.248 *Apart from the risks associated with the use of mobile phones while driving, which are discussed in paragraphs 5.201–5.214, there is no persuasive epidemiological evidence that exposure to RF radiation in general – or to the limited extent that it has been investigated, mobile-phone-related exposures in particular – causes disease in people. Although the epidemiological research that has been carried out to date does not give cause for concern, it has too many limitations to give reassurance that there is no hazard. A substantial number of people report symptoms such as fatigue, headache and feelings of warmth behind the ear that occur during or shortly after the use of mobile phones. However, it is unclear to what extent, if any, these symptoms are caused by RF radiation.*

Proposals for further research

5.249 In view of the widespread use of mobile phone technology, any adverse effects on health could affect large numbers of people. This is clearly a source of anxiety among some members of the public. We therefore identify the following epidemiological research to try to resolve the current uncertainty. Details of the methodological considerations needed to conduct these studies with high quality are given by EC (1996), Swerdlow (1997), and Repacholi and Cardis (1997).

Case–control studies of cancer risk in relation to the use of mobile phones

5.250 There is a pressing need for case–control studies to examine whether leukaemia and cancers of the brain, acoustic nerve and salivary gland are caused by mobile phone use.

5.251 A large case–control study of the risk of brain tumours in relation to the use of mobile phones is close to publication in the USA (Inskip *et al*, 1999), but its results are not yet available to us. An international case–control study of brain cancer, acoustic neuroma, salivary gland tumours and leukaemia co-ordinated by the International Agency for Research on Cancer (IARC), and including components in Britain, has received partial funding from the European Commission. At the time of our review, however, funding is incomplete and it is unclear how much of the study will be undertaken. The IARC study also includes important methodological work to assess the validity of subjects' recall of mobile phone use in comparison with information from billing records.

5.252 In view of the possibility that mobile phones might cause malignancies after a long induction period, such case–control studies may need to be repeated in the future when the technology has been in place for a longer time, and also in order to cover the possibility that changes in technology might be material to risk.

5.253 *We propose that large case–control studies of brain cancer, acoustic neuroma, salivary gland cancer, and leukaemia should be funded.*

Cohort study of users of mobile phones

5.254 The case–control method has several limitations. These include difficulties in the selection of appropriate controls and the possibility that cases recall and report exposures more completely than controls. Also, case–control studies usually focus on only one or two diseases, whereas in a single cohort study many different health outcomes can be examined. In the case of RF radiation from mobile phones, concerns have been expressed about a possible risk of leukaemia or tumours of the head and neck, but there is also uncertainty about the risks of other diseases.

5.255 Therefore, despite its cost, we believe that a large cohort study following up individuals according to their use of mobile phones would be desirable, and would complement the case–control studies discussed in paragraphs 5.250–5.253. Such a study would start to produce results within a few years, but because of the need to monitor possible long-term effects, it would need to continue for many years in the future. It could provide information on risks of all cancers and causes of death, rather than the few specific diseases currently being investigated by the case–control method, and would eventually allow examination of effects that might occur with longer induction periods. It would also offer scope for studies of morbidity in subsets of individuals (eg of neuropsychological disorders) if these were required.

5.256 We are aware of three existing cohort studies worldwide: one of 250,000 mobile phone users in the USA that we understand is currently in abeyance (Rothman *et al*, 1996; Dreyer *et al*, 1999), one of 550,000 users in Denmark (Johansen and Olsen, 1999), and one of 50,000 users in the UK (Beral, 2000). The US and Danish studies were based on individuals identified from operators' records, and were planned as mobile phone cohorts, whereas the British subjects, who are from a national cohort of women attending breast screening clinics, were not chosen on the basis of mobile phone use and include few long-term users. Although these studies should provide useful information, we think that there would be benefits from a further cohort study in the UK focussing specifically on long-term use of mobile phones.

5.257 In order to have statistical power to examine risks of specific cancers and non-cancer outcomes, a cohort study would need to be very large, including tens or even hundreds of thousands of individuals. For this reason, it would be expensive. In order to maximise the information that could usefully be gained within the next few years, it would be essential that it included large numbers of long-term users of mobile phones. There is usually an interval of some years between first exposure to a cause of cancer and the manifestation of an increased risk of the disease. Also, any risks from the use of early models of mobile phone are of particular importance since these early models had greater power outputs than those used more recently. As mobile phone use in the UK started particularly early by international standards, but covered only a small proportion of the population (120,000 in 1986; 500,000 in 1988), there is the potential to conduct a study of international importance, provided that it focusses on this early-user population. The study will therefore need to concentrate on populations where intensive use began earliest; we understand that this was in the London area. It is also desirable that the study should collect information not just on phone use, but also on potentially confounding variables, as far as practical. Early users of mobile phones may have very different characteristics from the general population with regard to, for instance, reproductive history, socioeconomic factors and other factors related to risk of disease.

5.258 *We propose that in addition to already ongoing cohort studies, a large cohort study of long-term mobile phone users be undertaken in the UK, which focusses particularly on people who started use in the 1980s and that, given the considerable design difficulties and potential costs entailed, a pilot study should be undertaken before a full-scale investigation.*

Symptoms in mobile phone users

5.259 The Expert Group heard several reports of mobile phone users who claimed symptoms relating to phone use, and the evidence from studies enquiring about this has indicated a substantial prevalence of symptoms thought by users to be related to phone use. To determine whether such symptoms are a consequence of RF radiation from phone use, a "double-blind" trial is needed, ie an experimental study of the occurrence of symptoms in circumstances where neither the user nor the observer knows whether a phone device is switched on or not. The Group also heard reports

of "highly sensitive" individuals; these individuals particularly need testing as to whether or not they truly have symptoms that relate to phone use under double-blind conditions.

5.260 Although the main focus of research on this question needs to be on trials in volunteers rather than on epidemiology, there would nevertheless be value in conducting a survey in the UK to discover the nature and prevalence of reported symptoms in the British context.

5.261 *We propose that double-blind trials be undertaken to assess the relation of mobile phone use to symptoms such as headache that have been reported by users, and that a cross-sectional survey of symptoms be conducted in relation to mobile phone use in the UK.*

Mobile phone use and motor vehicle accidents

5.262 As reviewed above, the available data suggest that mobile phone use can be a factor causing road traffic accidents, but they do not show greater risk in relation to hand-held as compared with hands-free phones nor whether mobile phones increase risk more than other causes of inattention such as the use of radios or conversations with passengers. The relationship of mobile phone use to the occurrence of accidents has major implications for public health policy, and it is therefore important to gain evidence on which to base this policy, especially on the comparative risks from hand-held and hands-free devices.

5.263 *We propose that further epidemiological studies should be undertaken to clarify the relation of mobile phone use to the risk of motor vehicle accidents, and in particular whether the risk differs between hand-held and hands-free phones, and whether the risk of hands-free use exceeds that of other forms of driver distraction, notably conversation with passengers.*

Effect of mobile phone base stations on well-being

5.264 Whilst we have focussed on the information available in the published literature, the Group was struck by the concerns expressed by many who attended the public meetings and who wrote to us about adverse effects on their well-being which they attributed to the presence of mobile phone base stations near to residences, schools, etc (see paragraphs 3.5 and 3.6). The social impact of mobile phone technologies needs to be fully considered. In addition to the improvements in planning which we consider to be essential (paragraphs 6.55–6.62) *there is a need for a significant research programme to be initiated so that the impact of mobile phone technologies on well-being in its broadest sense is properly addressed and understood through epidemiological or other approaches.* This should be brought to the attention of funding agencies such as the Economic and Social Research Council, the Medical Research Council, the European Commission and other bodies.

Conclusions Based on Overall Scientific Evidence

5.265 We conclude that there is one substantial established risk to health from mobile phone technology, namely through the increased incidence of motor vehicle accidents when drivers use mobile phones. Since the chance of an accident appears to be equally elevated for hands-free and hand-held use, this effect is almost certainly due to the distracting effect of the conversation, rather than to interference with steering the vehicle or to a direct influence of RF radiation on the brain.

5.266 There is also good evidence that exposure to mobile phone signals at intensities within existing ICNIRP guidelines has direct, short-term effects on the electrical activity of the human brain and

on cognitive function. These could have their origin in a variety of biological phenomena, for which there is some evidence from experiments on isolated cells and animals. There is an urgent need to establish whether these direct effects on the brain have consequences for health, because, if so, and if a threshold can be defined, exposure guidelines will have to be reconsidered. It is also important to determine whether these effects are caused by local elevation of temperature or, as seems possible, by some other, "non-thermal", mechanism.

5.267 The epidemiological evidence currently available does not suggest that RF exposure causes cancer. This conclusion is compatible with the balance of biological evidence, which suggests that RF fields below guidelines do not cause mutation, or initiate or promote tumour formation. However, mobile phones have not been in use for long enough to allow comprehensive epidemiological assessment of their impact on health, and we cannot, at this stage, exclude the possibility of some association between mobile phone technology and cancer. In view of widespread concern about this issue, continued research is essential.

5.268 Experimental studies on cells and animals do not suggest that mobile phone emissions below guidelines have damaging effects on the heart, on blood, on the immune system or on reproduction and development. Moreover, even prolonged exposure does not appear to affect longevity. The limited epidemiological evidence currently available also gives no cause for concern about these questions.

5.269 The balance of evidence indicates that there is no general risk to the health of people living near to base stations where the exposures are only small fractions of guidelines.

Overall Recommendations for Future Research

5.270 On the basis of the current state of knowledge **we recommend that priority be given to a number of areas of research related particularly to signals from handsets.** These should include the following:

- effects on brain function,
- consequences of exposures to pulsed signals,
- improvements in dosimetry,
- the possible impact on health of subcellular and cellular changes induced by RF radiation,
- psychological and sociological studies related to the use of mobile phones,
- epidemiological and human volunteer studies (paragraphs 5.249–5.264), including the study of children, and individuals who might be more susceptible to RF radiation (paragraphs 4.37, 6.29 and 6.30).

5.271 **We recommend that a substantial research programme should operate under the aegis of a demonstrably independent panel.** The aim should be to develop a programme of research related to health aspects of mobile phones and associated technologies. This should complement work sponsored by the EU and in other countries (paragraphs 5.274–5.285). In developing a research agenda the peer-reviewed scientific literature, non-peer-reviewed papers and anecdotal evidence should be taken into account.

5.272 **We further recommend that this programme be financed by the mobile phone companies and the public sector (industry departments, health departments and the research councils), possibly on a 50 : 50 basis.** The contribution from industry could be made on a voluntary basis or by a continuing levy reviewable every five years.

5.273 It will be essential for further research in this area to be kept under review. **We recommend that the issue of possible health effects of mobile phone technology should be the subject of a further review in three years time, or earlier if circumstances demand it.** We note the World Health Organization (WHO) has an established formal process of risk assessment relating to RF fields within this time frame.

ANNEX

Current Research Funding

5.274 Funding for research on health effects arising from exposure to RF radiation progressively increased through the 1990s, although it has not been straightforward to raise sufficient funds for a comprehensive programme. This has resulted from a number of reasons. In particular, funding from Government has been limited, as has support from industry and in the latter case there continues to be a problem of potential conflicts of interest, which needs to be addressed in any future funding arrangements.

5.275 In the early 1990s, the emphasis of research on possible health effects associated with exposure to electromagnetic fields and radiations (EMFs) was principally driven by concerns about exposure to extremely low frequency (ELF) electromagnetic fields. Substantial funding was made available in the USA for both epidemiological and experimental studies through the RAPID programme, sponsored jointly by the Department of Energy (DOE) and the National Institute of Environmental Health Sciences (NIEHS) with support from industry. This was also the main emphasis for funding in Europe and around the world.

5.276 In the mid-1990s, however, in the early days of mass marketing of mobile telecommunications, issues about possible health effects began to arise. A turning point in the debate was a discussion of the issue on the CNN programme*, Larry King Live*, in the early 1990s, which focussed on a brain tumour in a man who had been occupationally exposed to RF radiation from mobile phones. The suggestion on the programme was that this cancer could have been caused as a result of his exposure. This TV programme was probably instrumental in establishing a programme of research in the USA, funded principally by industry, and finally called the Wireless Technology Research programme. It had a budget in the range from about $20M to $30M and supported a programme of experimental and epidemiological studies. It came to a close at the end of the 1990s and much of the work that was carried out under the programme is presently being prepared for publication. There are no indications of a further substantial research effort being mounted in the USA at present.

5.277 In Europe, concerns about possible health effects of exposure to RF radiation from mobile phones followed those in North America but with a delay of about two years. In 1996, the European Commission contracted an EC Expert Group to make recommendations for a programme of scientific research on personal telecommunications and human health. The EC Expert Group reported in September 1996 but it has taken until the beginning of 2000 for the programme to get under way. The EC Expert Group recommended a number of areas for research including:

- *in vitro* studies;
- experimental studies in laboratory animals covering
    - genotoxicity,
    - cancer studies,
    - effects on the immune system,
    - nervous system related studies;
- human laboratory research on possible neurophysiological effects;

- provocation studies involving the acute exposure of people claiming neurological systems, changes in sleep pattern and effects on the immune system;
- epidemiological studies related to the possible risk of brain cancer;
- cancers of other exposed tissues.

5.278 Although specific details of the programme to be funded by the EC have not been published, many of the proposals have been supported including experimental studies in laboratory animals and epidemiological investigations. Human volunteer studies do not appear to have been supported at present. The telecommunications industry is supporting the EC programme with input on the design and development of exposure facilities and exposure assessment.

5.279 Within the EC, a COST Action Plan has also been developed on possible health effects related to the use of mobile phones. The plan contains an update of the report and recommendations of the 1996 EC Expert Group report and the published proceedings of a forum on future European research on mobile telecommunications and health, held at the University of Bordeaux, 19–20 April 1999. The research recommendations are generally in line with those in the 1996 Expert Group report, updated where appropriate.

5.280 Within the UK, NRPB has a programme of research related to possible health effects of RF radiation. Its total budget is about £300k per year and covers the development of the application of anatomically realistic phantoms, based on medical imaging data, to assess exposure; experimental studies on cells in culture; and studies on the possible behavioural effects of RF exposure using experimental animals.

5.281 NRPB is providing substantial support on exposure assessment protocols and measuring equipment calibration for a national study to investigate occupational exposure to RF electromagnetic fields and radiation from various sources, including broadcast transmitters and telecommunications. The study started on 1 November 1998 and is being carried out in collaboration with the Institute of Occupational Health, University of Birmingham, with support from industry. It aims to determine the feasibility of undertaking an industry-wide epidemiological study and seeks to develop an appropriate exposure metric.

5.282 Over the last three years the Department of Health and the Health and Safety Executive have funded two studies in the UK covering human volunteer investigations and experimental studies on the effects of RF radiation on brain tissue *in vitro*. The total budget has been £117k. In addition, £20k per year is contributed to the WHO EMF programme. Presently Government is in discussion with industry about funding a collaborative UK-based research programme to which public funds could be allocated. This is in response to the Third Report of the Science and Technology Committee (1999).

5.283 Internationally, a number of other programmes are under way. The WHO Reseach Agenda is of particular importance in this context. The Agenda was set out by WHO following an evaluation of research priorities based on the recommendations of the EC Expert Group and conclusions from relevant workshops and reviews.

5.284 The items on the WHO Research Agenda relevant to mobile phone technology and human health follow.

- Large-scale standard two-year animal bioassays such as those typically conducted by the US National Toxicology Program. These studies should be carried out using normal animals and animals initiated with chemical carcinogens. The exposures should use RF radiation in the

mobile phone frequency range and one of the common mobile phone pulsing patterns for two to six hours daily. Each study should use a range of different intensities (normally 4 SARs).

- At least two large follow-up studies on transgenic mice using study designs similar to the Repacholi study on Eμ-*PIM1* mice. Follow-up research is also needed that provides information on the health implications of effects found in transgenic animals.
- Studies to test the reproducibility of reported changes on hormone levels, effects on the eye, inner ear and cochlea, memory loss, neurodegenerative diseases and neurophysiological effects. Studies to be performed on people where possible and on animals as appropriate.
- At least two large-scale epidemiological studies with well-characterised higher level RF exposures to investigate cancers, particularly in the head and neck, and any disorders associated with the eye or inner ear. These studies should preferably be on mobile phone users or on workers in industries giving high RF exposures provided valid exposure assessments can be developed.
- Well-controlled studies to test people reporting specific symptoms such as headache, sleep disorders or auditory effects, and who attribute these symptoms to RF exposure. Several more studies to investigate neurological, neuroendocrine and immunological effects.
- *In vitro* studies relevant to possible *in vivo* effects and addressing the issues of RF exposure, thresholds and reproducibility for reported positive effects on cell cycle kinetics, proliferation, gene expression, signal transduction pathways and membrane changes.

5.285 WHO also provides advice and information on experimental design criteria, experimental systems and dosimetry, data collection and quality assurance, data analysis, reporting results, independent research review and administration and coordination of research.

6

A Precautionary Approach

6.1 Many of those who submitted evidence to the Expert Group, either orally or in writing, urged the application of a precautionary approach to the new technology of mobile phones, and especially to the siting of base stations. Before considering the case for this and the ways in which it might operate, it is helpful to review the general principles of risk assessment and risk management.

Risk Assessment

6.2 Risk assessment is the process whereby the potential adverse consequences (hazards) associated with a technology or development are identified, and the probability (risk) of their occurrence is estimated. The hazards may be to human health or to the environment, or may be economic, but below we focus on hazards to human health. The identification of health hazards and the estimation of associated risks may be based on various sources of information.

6.3 It is often possible to predict the hazards from a new technology on theoretical grounds, especially where it has evolved from other similar technologies already in use. If these hazards are well understood then risk assessment may only require an estimate of the levels of exposure that will occur. For example, the main hazard associated with a new industrial plant might be one of noise-induced deafness in the people who will work on it. The quantitative relation between noise exposure and deafness is well characterised, and an assessment of risk would therefore be possible once the likely levels of workers' exposure to noise had been established.

6.4 Laboratory experiments are another source of information. Experiments may be carried out *in vitro* (eg tests of a chemical's capacity to cause mutations in the genetic material of bacteria), using living animals (eg tests for long-term toxicity when a chemical is regularly inhaled or ingested), or more rarely, using human subjects. Such investigations form the basis for the risk assessment of many new chemicals such as drugs and pesticides.

6.5 Epidemiological studies of people are also important. These involve comparing rates of disease in different groups of people according to their exposure to known or suspected hazards.

6.6 Each source of information has advantages and disadvantages to its use. Background scientific knowledge can be applied relatively cheaply and quickly. Experience indicates that it is usually reliable, although not always. For example, it would have been difficult to predict the hazard of cancer from asbestos on the basis of scientific knowledge at the time the mineral first came into use. Similarly, before the emergence of new-variant CJD, it seemed unlikely that BSE would pose a significant health risk to people.

6.7 Laboratory experiments may take up to several years to complete, but can usually be carried out before any extensive human exposure to a new technology has occurred. There are, however, uncertainties in the extrapolation of findings from animals to people. For example, arsenic is

known to cause skin and lung cancer in people, but attempts to demonstrate the hazard in animals have failed.

6.8 Epidemiological studies provide direct information about risks in people, but by definition elevations of risk can only be demonstrated once disease has started to occur. Ideally, hazards would be prevented or eliminated before any ill-effects in people had resulted. Furthermore, the accuracy with which risks can be estimated from epidemiological studies is limited by the practical and ethical constraints of working with human subjects.

6.9 At each stage in the development of a new technology, risk assessment entails a synthesis of all the relevant information that is available from the sources described. Depending on how much information is available, risk estimates will be more or less certain. Thus, our knowledge about the adverse effects of ionising radiation is such that we can predict risks with relative precision. On the other hand, the risks associated with many industrial chemicals have been much less studied, and while current evidence may not suggest any important risk, we cannot always exclude this possibility with the same confidence.

Risk Management

6.10 Risk management is the process by which the risks and benefits associated with a technology or development are weighed against each other and decisions are made on whether and how to proceed with its implementation. The benefits may be real or potential, and direct (eg an improvement in health from a new drug) or indirect (eg making an industry more competitive and thereby promoting employment). The balancing of risks and benefits should take account of the uncertainties in risk estimates and also the severity of the adverse effects that might result. A small risk of a minor health effect such as transient headache might be acceptable, whereas the same risk of a more serious outcome such as brain cancer would not be acceptable.

6.11 A common approach in risk management is to identify a critical adverse health effect, (usually that which occurs at the lowest level of exposure). The lowest exposure at which this effect has been shown to occur is then multiplied by an “assessment” factor, also known as a “safety” or “uncertainty” factor, to derive an exposure limit or guideline. The aim is that, below this limit, exposures will not cause the adverse effect in any individual. Moreover, because the starting point for the calculation is the adverse health effect that occurs most readily, others, which only occur at higher exposures, should also be prevented. The assessment factor is designed to allow for differences in sensitivity between individuals, and also, if the assessment is based on data from animals, between species. It may be increased if the critical health outcome is particularly serious, eg cancer or congenital malformations. The exact size of assessment factors used, however, is to some extent arbitrary. It should be noted that this derivation of exposure limits or guidelines is based only on observed adverse effects. There may be evidence for other biological effects at lower exposures, but if these are not considered to be adverse, they do not enter the calculation.

6.12 Risk management is not a simple accounting process since, in general, risks cannot be quantified in the same units as benefits. For example, it may be necessary to weigh a risk to health against an economic gain. This is not an impossible task. It is something that all of us do regularly in our day-to-day lives. When we buy a new car, we make a decision – conscious or unconscious – whether to pay extra for additional safety features. When we decide to save money by undertaking a “do-it-yourself” (DIY) task in the home, we accept the risks of accidental injury that may be entailed. Such decisions involve value judgements, and individuals will differ in where they draw the balance between perceived risks and benefits.

6.13 A further complication arises because the people who benefit most from a new development are not necessarily those who will incur the highest risks. A new municipal incinerator may be to the advantage of most people in a community, but may pose an increased risk of road traffic accidents to those who live nearby. In this circumstance, balancing the risks and benefits poses moral and ethical questions and, in a democratic society, is overseen by the elected representatives of the people.

The Precautionary Principle/Approach

6.14 We live in an era in which science and technology are advancing at an ever-increasing rate. This has led to many improvements in health and in the quality of life. Thus in the UK, as in many other developed countries, life expectancy is currently increasing. At the same time, however, many people have anxieties about the pace of change and the potential for major adverse consequences if new developments are not appropriately controlled – if science has greater power to do good, it also has greater power to do harm. They therefore advocate a precautionary approach to new technology where there are uncertainties about the associated risks. In this context it is important to note the recent publication of the European Commission on the precautionary principle (EC, 2000), where guidance is given on approaches that should be used. Most importantly, this document indicates that actions taken under the precautionary principle should be commensurate with anticipated risks of health detriment. This has also been outlined in a World Health Organization (WHO) background publication entitled "Electromagnetic Fields and Public Health: Cautionary Policies" (WHO, 2000).

6.15 Some people propose that new developments should only be permitted when they have been shown to be completely safe, but this is unrealistic. Science can never provide a guarantee of zero risk. It may, however, offer strong reassurance that any risks from a technology are small in comparison with many other risks that we accept in our lives.

6.16 The precautionary approach is not all or none in nature. Rather, it is a matter of degree. In essence, it requires that before accepting a new development we should have positive evidence that any risks from it are acceptably low, and not simply an absence of convincing evidence that risks are unacceptably high. However, individuals will differ in the strength of evidence that they need before concluding that risks are sufficiently small. The implementation of a precautionary approach carries costs, which may be direct, eg for better engineering, or from a delay in the benefits that the new technology will bring. Important indirect costs may also arise if resources are directed away from a more serious risk to deal with another risk that is in fact very minor. The aim, therefore, must be to follow a policy that is acceptable to most people, and which minimises the chance of adverse outcomes without unnecessarily stifling progress.

6.17 The policy by which a precautionary approach is applied to risk management in situations of scientific uncertainty has been termed the precautionary principle. This principle was formally adopted by countries of the European Union in the Treaty of Maastricht (1992), and is evident in a ruling of the European Court of Justice when it upheld the decision of the European Commission to ban beef from the UK with a view to limiting the risk of transmission of BSE. The Court concluded as follows:

> *"In view of the seriousness of the risk and the urgency of the situation, and having regard to the objective of the decision, the Commission did not act in a manifestly inappropriate manner by adopting the decision, on a temporary basis and pending the production of more detailed scientific information.*

> *"Where there is uncertainty as to the existence or extent of risks to human health, the Commission may take protective measures without having to wait until the reality or seriousness of those risks becomes apparent."*

The application of this principle was defined further in an EC Commentary in February 2000 (EC, 2000).

6.18 Against this general background, we now consider how the potential health risks from mobile phone technology should best be managed.

Application of the Precautionary Approach to Mobile Phone Technology

Exposure guidelines for RF radiation

6.19 As described in paragraph 6.11, one approach that is often adopted in risk management is to define exposure limits or guidelines, below which the recognised adverse effects of a hazard would not be expected to occur. In the UK, national guidelines on exposure to RF radiation were drawn up by NRPB (NRPB, 1993a,b), and have been accepted and implemented by Government Departments and Agencies.

6.20 Having reviewed all relevant epidemiological studies, NRPB concluded that the results were inconclusive and did not provide an adequate starting point from which to derive exposure guidelines (NRPB, 1993b,c). Instead, therefore, the guidelines were based on the potential of RF radiation to cause illness or injury through heating of body tissues. While some research had suggested that adverse health effects might occur from exposures lower than those needed to produce significant heating, the evidence for this was not considered sufficiently robust to form a basis for the derivation of exposure guidelines.

6.21 The NRPB exposure guidelines incorporate *basic restrictions* on the specific energy absorption rate (SAR, see paragraph 4.37). For mobile phones, the relevant restrictions are for frequencies between 10 MHz and 10 GHz, and these are given in Table 6.1. To verify that the exposure of an individual is within NRPB guidelines, it is necessary to demonstrate that none of the four basic restrictions is exceeded. The SAR is averaged over an exposure time and a specified mass of tissue, depending on the tissue region. Averaging times are specified because of the time taken for the temperature of tissues to equilibriate when they are exposed to the radiation.

Table 6.1 NRPB basic restrictions on exposure in the frequency range 10 MHz to 10 GHz (NRPB, 1993b)

		Averaging parameters	
Tissue region	SAR limit (W/kg)	Mass (g)	Time (minutes)
Whole body	0.4	–	15
Head, fetus	10	10	6
Neck, trunk	10	100	6
Limbs	20	100	6

6.22 These restrictions apply equally to workers and to members of the general public. NRPB has taken the view that they provide adequate protection against harmful thermal effects for all exposed individuals under all conditions (NRPB, 1999a,b).

6.23 Since SARs cannot easily be measured in living people, the NRPB guidelines also specify *investigation levels* for external electromagnetic field strengths, at or below which the basic restriction on whole-body SAR will not be exceeded. If an investigation level is exceeded, more detailed investigation of the resultant SAR is indicated. For children, additional reductions in investigation levels for the whole body are applied because, over certain RF frequencies, small children absorb more energy from external electromagnetic fields than adults. Table 6.2 shows these investigation levels for the frequency range covered by mobile phones.

Table 6.2 NRPB investigation levels for exposure at mobile telecommunications frequencies (NRPB, 1993b)

Frequency (MHz)	Electric field strength (V/m)	Magnetic field strength (A/m)	Power density (W/m^2)
400 – 800	100	0.26	26
800 – 1550	125*f*	0.33*f*	$41f^2$
1550 – 3000	194	0.52	100

f is the frequency in GHz.

6.24 The three investigation level quantities shown in Table 6.2 are related to each other (the fields are assumed to be in the far-field region – see paragraph 4.24), and it is only necessary to consider one of them. To investigate compliance, measurements are usually made of either electric field strength or magnetic field strength.

6.25 For the current generation of mobile phones and their base stations the investigation levels in the frequency range 800–900 MHz are from 26 to 33 W/m^2 and for the range 1800–1900 MHz the level is 100 W/m^2.

6.26 Since publication of its guidelines, NRPB has continued to monitor and review the published scientific literature relevant to exposure to electromagnetic fields and human health. In this, it is supported by its independent Advisory Group on Non-ionising Radiation chaired by Sir Richard Doll. Its current position is that compliance with its guidelines for exposures to electromagnetic fields will prevent any known adverse effects on human health (NRPB, 1999a,b).

6.27 Guidelines on exposure to RF radiation have also been published by the International Commission on Non-Ionizing Radiation Protection (ICNIRP, 1998a,b). Similarly to the NRPB guidelines, these are designed to prevent illness or injury through heating effects. Their starting point is the behavioural changes that have been found when experimental animals were exposed to RF radiation at levels that produced a rise in whole-body temperature in excess of 1°C. An SAR of 1–4 W/kg or higher is needed to cause these changes (1 W/kg when animals are exposed in conditions of adverse temperature, humidity and air movement, and 4 W/kg under normal environmental conditions). ICNIRP considered that there was no firm evidence for any effects that would impair health at lower levels of exposure to RF radiation.

6.28 In contrast to the NRPB guidelines, the ICNIRP guidelines feature a two-tier system with lower limits for exposure of the general public than for occupational exposure. For occupational exposure, the basic restrictions on SAR are the same as those recommended by NRPB (see Table 6.3), except that an averaging time of six minutes and an averaging mass of 10 g apply to the three localised SAR restrictions.

6.29 However, for exposure of the general public, the guidelines are five times lower than for occupational exposure. This difference was intended to allow for the following circumstances.

Table 6.3 ICNIRP basic restrictions on occupational exposure and general public exposure (in brackets) in the frequency range 10 MHz to 10 GHz (ICNIRP, 1998a)

		Averaging parameters	
Tissue region	SAR limit (W/kg)	Mass (g)	Time (minutes)
Whole body	0.4 (0.08)	–	6
Head, trunk	10 (2)	10	6
Limbs	20 (4)	10	6

- *Exposure under extreme environmental conditions – high temperatures, high humidity, low air movement and high activity increase the thermal burden from RF exposure.*
- *Potentially higher thermal sensitivity in certain population groups such as those who are frail or elderly, infants, young children, and people with diseases or taking medications that compromise thermal tolerance.*

6.30 Further arguments in support of the additional reduction factor for public exposure were also given in an earlier publication (INIRC/IRPA, 1988) as follows.

- *Workers are normally healthy adults exposed under controlled conditions, who are trained to be aware of potential risks and to take appropriate precautions to avoid unnecessary exposure. The general public cannot reasonably be expected to take the same precautions.*
- *Workers are exposed only during the working day (usually 8 hours per day). On the other hand, the general public can be exposed for 24 hours per day (this total weekly exposure duration is approximately five times that of workers; hence the derivation of the extra safety factor of five for the general public).*
- *In general, children and babies are normally considered to be more sensitive to exposures to physical, chemical or biological agents. At higher frequencies, children absorb more energy from external electromagnetic fields than adults.*

6.31 The ICNIRP guidelines are presented with *reference levels* analogous to the NRPB *investigation levels*, and these also reflect the factor of five difference between the public and occupational basic restrictions. In general, over the frequency range used by mobile phones, the ICNIRP reference level for the public is lower (in terms of power density) than the NRPB investigation level by a factor of between 6.5 and 11. The ICNIRP public reference levels for the frequencies used by mobile phones are shown in Table 6.4. Reference levels for mobile telecommunications in the frequency range 800–1000 MHz are from 4 to 5 W/m^2 and for 1800–1900 MHz from 9 to 9.5 W/m^2 (ICNIRP, 1998a).

Table 6.4 ICNIRP reference levels for public exposure at mobile telecommunications frequencies (ICNIRP, 1998a)

Frequency (MHz)	Electric field strength (V/m)	Magnetic field strength (A/m)	Power density (W/m^2)
400 – 2000	$1.375f^{½}$	$0.0037f^{½}$	f/200
2000 – 3000	61	0.16	10

f is the frequency in MHz.

6.32 Following a detailed comparison of the NRPB and ICNIRP guidelines and the implications for practical hazard assessment (NRPB, 1999a), the National Radiological Protection Board issued the following Statement:

> *"The Board has concluded that for occupational exposure the basic restrictions in the new ICNIRP guidelines do not differ in any significant way from those previously recommended by NRPB and have no implications for the UK guidelines. For members of the public, ICNIRP has generally included a reduction factor of up to five in setting basic restrictions across the frequency range to 300 GHz. There is, however, a lack of scientific evidence to support the introduction of these additional reduction factors. The Board believes that the existing UK advice by NRPB on limiting exposures for the general public already provides sufficient protection from direct and indirect effects and that any health benefits to be obtained from further reductions in exposure have not been demonstrated. It sees no scientific justification, therefore, for altering the advice previously given by NRPB on exposure guidelines for members of the public. It does, however, accept that other factors may need to be taken into account by government in establishing generally accepted exposure guidelines for the public.*
>
> *"In relation to furthering knowledge on possible health effects of exposure to EMFs, the Board supports the need for further epidemiological and experimental studies."*

The Board also said that the Statement reflected "understanding and evaluation of the current scientific evidence. If and when further relevant information becomes available, the Board will review its advice".

6.33 The ICNIRP guidelines for the public have been adopted in a European Council Recommendation (1999), which has been agreed in principle by all countries in the European Union, including the UK. In Germany the ICNIRP guidelines have been incorporated into statute.

6.34 *From a review of the scientific evidence, we conclude that the prevention of heating effects currently remains the best basis for exposure guidelines. We further conclude that the approach adopted by ICNIRP is preferable to that of NRPB. Within the general public there may be people with illnesses that render them unusually susceptible to the heating effects of RF radiation, and this justifies the use of a higher assessment factor than for occupational exposures.*

6.35 **We recommend that, as a precautionary approach, the ICNIRP guidelines for public exposure be adopted for use in the UK rather than the NRPB guidelines.** This would bring the UK into line with other countries in the European Union and accord with the recommendations of the House of Commons Select Committee on Science and Technology Report on Mobile Phones and Health (1999).

6.36 **We are not convinced of the need to incorporate ICNIRP guidelines into statutes.** We believe that they are liable to change as more scientific information on possible health effects becomes available.

6.37 **The balance of evidence to date suggests that exposures to RF radiation below NRPB and ICNIRP guidelines do not cause adverse health effects to the general population (paragraphs 5.267–5.269).**

6.38 **There is now scientific evidence, however, which suggests that there may be biological effects occurring at exposures below these guidelines.** This does not necessarily mean that these effects lead to disease (paragraph 5.266).

6.39 There are additional factors that need to be taken into account in assessing any possible health effects. Populations as a whole are not genetically homogeneous and people can vary in their susceptibility to environmental hazards. There are well-established examples in the literature of the genetic predisposition of some groups, which could influence sensitivity to disease. There could also be a dependence on age. **We conclude therefore that it is not possible at present to say that exposure to RF radiation, even at levels below national guidelines, is totally without potential adverse health effects, and that the gaps in knowledge are sufficient to justify a precautionary approach.**

6.40 In the light of the above considerations **we recommend that a precautionary approach to the use of mobile phone technologies be adopted until much more detailed and scientifically robust information on any health effects becomes available. We further recommend that national and local government, industry and the consumer should all become actively involved in addressing concerns about possible health effects of mobile phones.**

6.41 On its own, adoption of the ICNIRP exposure guidelines will not allow fully for the current gaps in scientific knowledge, and particularly the possibility of, as yet, unrecognised thermal or non-thermal adverse effects at lower levels of exposure. One way in which this uncertainty could be taken into account would be to apply a higher assessment factor in the derivation of the exposure guidelines. This would have the merit of simplicity. However, as yet, there is no satisfactory scientific basis on which to set the size of any increase.

6.42 An alternative would be to adopt the exposure guidelines recommended by ICNIRP, and in addition have a policy that requires best engineering practice for equipment and installations that ensures that fields are kept to the lowest levels commensurate with the telecommunications system operating effectively. We believe that this approach is preferable. We next consider how it might be applied in relation to the design and siting of base stations, and the design, marketing and use of mobile phones.

Base Stations

6.43 The location of base stations and the processes by which they are authorised appear to be the aspects of mobile phone technology that generate most public concern (see Chapter 3). Public telecommunications operators have been granted a number of rights similar to those enjoyed by gas, water and electricity companies. These include permitted development rights, which allow them to carry out certain developments, including the erection of masts less than 15 m high, without the need to make a full planning application. (A more detailed description of the current planning situation with respect to telecommunications is given in paragraphs 6.96–6.117.) In assessing the potential impact of a planned base station on health, the current approach in the UK is to determine whether it might cause exposures in excess of NRPB guidelines (NRPB, 1993a,b; 1999). If this can be ruled out satisfactorily, risks to health are not considered further.

6.44 We believe this approach is not optimal since it does not allow adequately for the uncertainties in scientific knowledge. Although it seems highly unlikely that the low levels of RF radiation from base stations would have significant, direct adverse effects on health, the possibility of harm from exposures insufficient to cause important heating of tissues cannot yet be ruled out with confidence. Furthermore, the anxieties that some people feel when this uncertainty is ignored can in themselves affect their well-being.

6.45 Other aspects of the planning process for base stations are also unsatisfactory. Some citizens feel that the siting of base stations, and particularly of masts, can result in a loss of amenity and possibly a reduction in the value of property, and it is clear that, in the face of this threat, many feel excluded and disempowered by the planning system now in operation. The resultant frustration also has negative effects on people's health and well-being.

6.46 *We conclude therefore, that changes to the regulation of base stations are necessary.*

National register of base stations

6.47 A first requirement is for reliable and openly available information about the location and operating characteristics of all base stations. Easy access to such information would help to reduce mistrust among the public. Furthermore, the data would be useful when applications for new base stations were being considered, and might also be of value in epidemiological investigations.

6.48 **We recommend that a national database be set up by Government giving details of all base stations and their emissions.** For each this should list: the name of the operating company; the grid reference; the height of the antenna above ground level; the date that transmission started; the frequency range and signal characteristics of transmission; the transmitter power; and the maximum power output under the Wireless Telegraphy Act. Moreover, this information should be readily accessible by the public, and held in such a form that it would be easy to identify, for example, all base stations within a defined geographical area, and all belonging to a specified operator.

Exclusion zones

6.49 Although exposures to RF radiation from base stations will generally be well below exposure guidelines, the need remains to prevent access by workers or the public to places where the relevant guidelines might be exceeded. Therefore, we endorse the practice of defining clear exclusion zones around base stations.

6.50 **We recommend the establishment of clearly defined physical exclusion zones around base station antennas, which delineate areas within which exposure guidelines may be exceeded. The incorporation of exclusion zones should be part of the template of planning protocols that we advocate (paragraphs 6.54, 6.58 and 6.59).**

6.51 Each exclusion zone should be defined by a physical barrier and a readily identifiable nationally agreed sign with a logo. This should inform the public and workers that inside the exclusion zone there might be RF emissions that exceed national guidelines. **We recommend that the design of the logo should be taken forward by the British Standards Institute and implemented within 12 months.**

6.52 **We recommend that warning signs should be incorporated into microcell and picocell transmitters to indicate that they should not be opened when in use.**

Audit of base stations

6.53 There is a need to ensure that base stations are operating within the parameters specified when they were approved.

6.54 **We recommend that an independent, random, ongoing audit of all base stations be carried out to ensure that exposure guidelines are not exceeded outside the marked exclusion zone and that the base stations comply with their agreed specifications. If base station emissions**

are found to exceed guideline levels, or there is significant departure from the stated characteristics, then the base station should be decommissioned until compliance is demonstrated. We recommend that particular attention should be paid initially to the auditing of base stations near to schools and other sensitive sites. The audit should include appropriate checks to ensure that base stations conform to the operational parameters specified when they were approved, and that exclusion zones are properly demarcated and signed.

Planning process

6.55 As described in the annex to this chapter, the erection of base stations for mobile phone networks is not subject to such stringent planning procedures as some other types of construction project. In particular, masts less than 15 m high can be built without the planning permission that would normally be required. The lack of public consultation is a major cause of grievance in people who suffer a loss of amenity when base stations are erected and we consider the current situation to be unacceptable.

6.56 One operator has told us that it now seeks full planning permission for all new masts, even if they will be less than 15 m high, but there appears to be significant variation in the extent to which operators consult the public about the siting of base stations.

6.57 We recommend that for all base stations, including those with masts under 15 m, permitted development rights should be revoked, and that the siting of all new base stations should be subject to the normal planning process. This planning process should also apply when a change to an existing base station will increase its power output.

6.58 We recommend that, at national Government level, a template of protocols be developed, in concert with industry and consumers, which can be used to inform the planning process and which must be assiduously and openly followed before permission is given for the siting of a base station.

6.59 We consider that the protocol should cover the following points.

- All telecommunications network operators must notify the local authority of the proposed installation of base stations. This should cover installations for macrocells, microcells and picocells.
- The local authority should maintain an up-to-date list of all such notifications, which should be readily available for public consultation.
- The operator should provide to the local authority a statement for each site indicating its grid reference, the height of the antenna above ground level, the frequency and signal characteristics, and details of maximum power output.
- Any change to an existing base station that increases its size, or the overall power radiated, should be subject to the normal planning process as if it were a new development.

6.60 We recommend that a robust planning template be set in place within 12 months of the publication of this report. It should incorporate a requirement for public involvement, an input by health authorities/health boards and a clear and open system of documentation which can be readily inspected by the general public.

6.61 We recommend that in making decisions about the siting of base stations, planning authorities should have power to ensure that the RF fields to which the public will be exposed will be kept to the lowest practical levels that will be commensurate with the telecommunications system operating effectively.

6.62 Where recommendations (paragraphs 1.30–1.46) impact on the devolved responsibilities of the Scottish Parliament, the Welsh National Assembly and the Northern Ireland Assembly then they should be considered by their appropriate authorities or bodies. We have noted with interest the recent report on planning procedures for telecommunications developments produced by the Transport and the Environment Committee of the Scottish Parliament in 2000 (paragraphs 6.112–6.114).

Base stations near schools

6.63 A common concern among members of the public who attended our open meetings was the siting of macrocell base stations on or near school premises. The placement of a base station on a school building may indirectly benefit its pupils through the income generated in rent. The balance of evidence indicates that there is no general risk to the health of people living near to base stations where the exposures are only small fractions of guidelines. However, it was suggested to us that children might be especially vulnerable to any adverse effects of RF radiation. There is evidence that at the frequencies used in mobile phone technology, children will absorb more energy per kilogram of body weight from an external electromagnetic field than adults (see paragraph 4.37). A one year old could absorb around double, and a five year old around 60%, more than an adult. Additionally, since children are being exposed to RF radiation from base stations (and from mobile phones) from a younger age than adults, they will have a longer time in which to accumulate exposure over the course of their lives, and a longer time for any delayed effects of exposure to develop.

6.64 In recognition of this, some countries have prohibited the placement of macrocell base stations on sensitive sites such as schools. Such policies have the merit of being easy to administer, but they may not always produce the desired effect. For example, because of the way in which emissions are beamed, a macrocell base station located near to a school may cause higher exposure to pupils than if it were placed on the roof of the school building.

6.65 We suggest therefore that a better approach would be to require that the beam of greatest RF intensity (see paragraph 4.32) from a macrocell base station sited within the grounds of a school should not be permitted to fall on any part of the school grounds or buildings without agreement from the school and parents. Furthermore, when consent is sought from a school and parents about this question, they should be provided with adequate information to make an informed decision, including an explanation of the way in which the intensity of radiation falls off with distance from the antenna. This may be particularly relevant for schools with large grounds. If, for an existing base station, agreement could not be obtained, its antennas might need to be readjusted.

6.66 We further suggest that similar considerations should apply in relation to a macrocell base station outside the grounds of a school but at a distance from the edge of the grounds comparable to that of a macrocell base station were it to be placed within the school grounds. In this case, if requested by the school or parents, the network operator should be required to inform the school whether the beam of greatest intensity (see paragraph 4.32) falls on the school grounds or buildings. If it does, the operator should tell them where it falls and the nearest distance from the antenna to these points. It should also provide them with adequate information to make an informed consideration of the level of the intensity of RF radiation. This information should include an explanation of the way in which the intensity of radiation falls off with distance from the antenna. If there is major concern about the situation from the school and parents, it may be necessary for the network operator to make adjustments to the antennas.

6.67 We suggest that the responsibility for monitoring the requirements of paragraphs 6.65 and 6.66 should be given to local authorities with advice from the agency responsible for maintaining the database. Disputes could be referred to the Ombudsman (see paragraph 3.51).

6.68 **We recommend, in relation to macrocell base stations sited within school grounds, that the beam of greatest RF intensity should not fall on any part of the school grounds or buildings without agreement from the school and parents. Similar considerations should apply to macrocell base stations sited near to school grounds.**

Developments in rural areas

6.69 In urban environments and adjacent to major roads and railways, the need for new base stations will arise principally from growth in the number of phone calls that must be handled at any one time. In rural areas, however, the main drive to expansion of networks at present is the need for wider geographical coverage. In this circumstance, there may be scope to limit the number of masts that are required through agreements between operators on mast sharing and roaming.

6.70 **We recommend that operators should actively pursue a policy of mast sharing and roaming where practical,** and that they should be considered by planning authorities as an alternative option when new masts are proposed.

Mobile Phones

6.71 Use of a mobile phone can expose tissues adjacent to the antenna to levels of RF radiation more than a thousand times higher than people would normally encounter from base stations. We understand from the Mobile Manufacturers Forum that all mobile phones presently marketed in the UK comply with both NRPB and ICNIRP guidelines for RF radiation and on current evidence, it seems unlikely that the exposures experienced by users would have important adverse effects on health. However, direct empirical support for this assessment is limited, and several observations suggest a need for caution.

6.72 As described in Chapter 5, recent experiments in people have suggested that subtle effects on brain function might occur from the use of mobile phones held to the head (Preece *et al*, 1999; Koivisto *et al*, 2000, *in press*; Krause *et al*, 2000), although even if confirmed by further research, these effects on function would not necessarily result in illness. Also of concern is the observation in one study that exposure to pulsed RF radiation may accelerate the development of tumours (Repacholi *et al*, 1997). These findings require independent confirmation. However, the uncertainties that such research raises are a reminder that the current evidence base is not yet so secure that the possibility of harmful effects from the use of mobile phones can be totally discounted.

6.73 These uncertainties are less problematic in so far as people can choose whether or not to use a mobile phone. However, it is important they should be adequately informed when making their choice, and that they be advised of the best way in which to reduce their exposure if that is what they wish to do.

Information for consumers

6.74 To this end, purchasers of mobile phones should have information to allow them to make informed choices about personal exposures resulting from their use. Based on current evidence, the main points to convey would be as follows.

- At present scientific evidence suggests that the RF radiation produced by mobile phones is highly unlikely to be a cause of direct adverse health effects on the general population of the UK.
- There is, however, still some uncertainty about this, and individuals may therefore wish to minimise their exposure to such radiation.
- This can be achieved in several ways including, for example, by making fewer and shorter calls.
- Specific absorption rate (SAR) values are a relevant measure of exposure in this situation and should allow people to make an informed choice.
- Another way of reducing exposure would be by use of an approved, hands-free set (see paragraphs 6.86–6.88).

6.75 We understand that an internationally agreed standard testing protocol for the assessment of SAR values from mobile phones will soon be available. We welcome this development.

6.76 **We recommend that an international standard for the assessment of SAR values from mobile phones should be adopted for use in the UK once it has been demonstrated to be scientifically sound.**

6.77 **We recommend that information on SAR values for mobile phones must be readily accessible to consumers:**

- **at the point of sale with information on the box,**
- **on leaflets available in stores giving comparative information on different phones and with explanatory information,**
- **as a menu option on the screen of the phone, and as a label on the phone,**
- **on a national web site, which lists the SAR values of different phone types**.

6.78 In order that consumers can interpret SAR values it will also be necessary to provide them with an explanation of the measure and its application.

6.79 Such information could be given by mobile phone manufacturers or retailers in addition to that already provided (see paragraphs 3.28 and 3.29). However, we believe that it would carry more weight if it came from Government and were clearly seen to be independent.

Shields

6.80 Shields seek to reduce the RF radiation to which users of mobile phones are exposed, and various types of device have been produced for this purpose. For most of them, eg ceramic absorbing devices, there is no apparent physical basis for their alleged effect, and there are no convincing test results to verify that they reduce exposure.

6.81 One particular type does have a physical basis. This type consists of a case that fits over the handset and has a metallic or metallic-mesh screen within the case and a "guard" for the antenna. Together these partially screen the radiation emitted by the phone.

6.82 Tests by various laboratories, including some that formed the basis of a *Which?* report published in April 2000, have measured the effect of this type of shield on the radiation from a mobile phone when it was set to produce constant power. The shield substantially reduced the radiation by a factor that could be adjusted by the user.

6.83 In most normal use, however, the shield would not reduce the exposure of the user to this extent, since the reduction in radiation produced by the shield would automatically be compensated for by adaptive power control. (This increases or reduces emissions to give an optimal signal at the base station, see paragraph 4.14.) An exception would occur where the mobile phone was already operating at or close to its maximum power, eg because it was a long way from the base station or in a building, but in this situation, the signal at the base station would be weakened by the shield and communication might not be possible.

6.84 Some of the test results have shown that the radiation is reduced more in the direction of the head than in the direction away from it. If so, users could reduce their exposure somewhat by turning the appropriate side of their head towards the base station. However, this could only be done if they could see the base station, which is not the usual situation. For other orientations of the head the tests imply that the reduction in exposure would be very small.

6.85 We conclude that, in practice, there would be very little reduction in the exposure received by most users through use of a shield of this type, and that their reception could be impaired when they were a long way from a base station or in buildings, cars, etc. If the use of shields became widespread there could also be adverse effects on the environment, since more base stations would likely be needed to maintain the quality of communication.

Hands-free kits

6.86 Exposure to RF radiation from a mobile phone can be reduced by increasing the distance of the phone from the body. This could be achieved by using an appropriately designed hands-free kit. Little or no advantage will be gained, however, if the phone is merely moved from the head to, say, the waist since in that case other organs may receive comparable exposure.

6.87 Even if the mobile phone is some distance from the body, reduction in exposure may not be achieved if the wires from the handset to the earpiece can carry radio signals to the ear or themselves radiate significantly. Since the original purpose of hands-free kits was to permit the use of both hands while phoning, they may not all have been designed with exposure in mind. The *Which?* report published in April 2000 showed that the hands-free kits tested could increase the exposure to the user. On the other hand, we are aware of other tests which claim a very substantial reduction. In both cases there is insufficient published information about the measurement methods to form a clear view. We believe, however, that it should be possible to design hands-free kits which would significantly reduce exposure to the user if used correctly (ie with the phone some distance from the body).

6.88 The regulatory position on the use of hands-free kits and shields is unclear and the only information available to the public appears to be that supplied by their manufacturers. **We recommend that the Government sets in place a national system which enables independent testing of shielding devices and hands-free kits to be carried out, and which enables clear information to be given about the effectiveness of such devices. A kite mark or equivalent should be introduced to demonstrate conformity with the testing standard.**

Use by children

6.89 We have already discussed the arguments for minimising the exposure of children in school to RF radiation from base stations (see paragraphs 6.63–6.68). These apply even more to the higher exposures that occur from use of mobile phones. There may be circumstances where the use of a mobile phone by a child can promote safety (eg to ask a parent for a lift rather than walk home alone).

6.90 **If there are currently unrecognised adverse health effects from the use of mobile phones, children may be more vulnerable because of their developing nervous system, the greater absorption of energy in the tissues of the head (paragraph 4.37), and a longer lifetime of exposure. In line with our precautionary approach, we believe that the widespread use of mobile phones by children for non-essential calls should be discouraged. We also recommend that the mobile phone industry should refrain from promoting the use of mobile phones by children.**

Use near hospitals

6.91 As described in paragraphs 4.5 and 4.6, there is a potential hazard from the indiscriminate use of mobile phones in hospitals and other sites where RF radiation could interfere with sensitive electronic equipment. We support the steps that are already being taken both by mobile phone manufacturers and hospitals to warn people about the dangers of using phones in such sites.

6.92 **We understand that health authorities/boards issue guidance on the use of mobile phones. We recommend that they should ensure that all hospitals comply. This guidance should include the placing of visible warning signs at entrances to buildings to indicate that mobile phones should be switched off.**

Use while driving

6.93 As described in paragraphs 5.201–5.214, there is strong evidence that use of a mobile phone whilst driving significantly increases the risk of accidents. It has been suggested to us that the use of hand-held phones while driving should be banned, and the Department of the Environment, Transport and the Regions (DETR, 2000) considered this issue sufficiently important to warrant a publicity campaign aimed at dissuading drivers from using a mobile phone, especially one which is hand-held, when in control of a vehicle.

6.94 We welcome this initiative, but note that, perhaps surprisingly, current evidence indicates that the negative effects of phone use while driving are broadly similar whether the phone is hand-held or hands-free.

6.95 **We conclude that the detrimental effects of hands-free operation are sufficiently large that drivers should be dissuaded from using either hand-held or hands-free phones whilst on the move.**

ANNEX (based on material provided to the Expert Group by DETR)

Current Planning Procedures for Telecommunications Development

6.96 All development requires planning permission. In most cases, this will entail a full application to the local planning authority (LPA) for express permission. LPAs are required to determine applications in accordance with the development plan, unless material considerations indicate otherwise. These considerations can include views expressed by local people. LPAs have to publicise all planning applications.

6.97 Relatively minor development does not require express permission. It is granted planning permission under the Town and Country Planning (General Permitted Development) Order 1995 – the "GPDO". These "permitted development rights" are enjoyed by a range of bodies, including householders and statutory undertakers.

Legal position

6.98 Larger developments, such as masts over 15 m high, require a full planning application, which will be considered by the LPA.

6.99 Public telecommunications operators hold licences under Section 7 of the Telecommunications Act 1984 to run telecommunications systems. In order to help them do this, they have been granted Telecommunications Code Powers (contained in Schedule 2 of the Telecommunications Act 1984). These essentially confer on telecommunications operators a number of rights similar to those enjoyed by the gas, water and electricity companies. These Powers enable them to install their systems in the maintainable highway and, with the appropriate consents, on private land. The Code also places a number of obligations on operators.

6.100 Part 24 of the GPDO grants a range of permitted development rights for telecommunications code system operators. These allow operators to carry out specified development, subject to certain conditions and limitations, without the need to make a full planning application to the LPA. This development includes masts of 15 m and below. However, there are special provisions regarding the installation of any mast in designated areas such as National Parks, Areas of Outstanding Natural Beauty, Conservation Areas and Sites of Special Scientific Interest. In these areas the installation of masts of 15 m and below do not enjoy permitted development rights under the GPDO. The installation of all masts in such areas is subject to a full planning application.

6.101 In general, permitted development under the GPDO is subject to various conditions and limitations. For telecommunications permitted development in England and Wales the GPDO includes an important additional control mechanism – the prior approval procedure. Following changes made in 1999 LPAs now have 42 days (rather than the previous 28) in which to determine and notify whether they wish to approve the siting and appearance of ground-based masts. Operators are also now required to post a site notice to publicise the proposed development. These changes were designed to give the public a better opportunity to comment to the LPA on its siting and appearance. There is also a statutory requirement for LPAs to consult the relevant parish council, at the council's request. Where the LPA considers that the proposed development would have a detrimental effect upon local amenity, it is able to refuse approval. However, LPAs are advised to explore the scope for modifying its siting and/or appearance before doing so.

6.102 For a number of other forms of telecommunications development under Part 24 of the GPDO the 28-day prior approval procedure continues to apply. This includes masts installed on a building or structure. Although there is not a statutory requirement for operators (or LPAs) to publicise such proposals, LPAs are encouraged to give proposals publicity so that local people can make their views known.

6.103 It is a condition of the permission granted by the GPDO that apparatus that is no longer required for telecommunications purposes should be removed as soon as reasonably practicable from the land or building on which it is located, and the land restored to its previous condition.

Policy guidance

6.104 In England, Government policy on planning for telecommunications development is set out in "Planning Policy Guidance Note 8" (PPG8), issued in December 1992. In Wales, similar guidance is to be found in "Planning Guidance (Wales): Planning Policy, First Revision" (April 1999), together with Technical Advice Note (Wales) 19, "Telecommunications", August 1998. This guidance should be taken into account by LPAs as they prepare their development plans, and may be material to decisions in individual planning applications and appeals. It is supplemented by DETR Circular 4/99 and Welsh Office Circular 29/99 ("Planning for Telecommunications"), issued in June 1999 in parallel with the GPDO changes outlined in paragraph 6.101 above.

6.105 It is Government policy that the number of telecommunications masts should be kept to a minimum and to encourage mast sharing where appropriate. The licences issued to the four existing network operators require them to take all reasonable steps to investigate using, or replacing, an existing mast or other structure before erecting a new mast. Where a new mast is required, operators are required to investigate co-operating with another operator in erecting a mast for joint use.

6.106 Planning Circulars 4/99 and 29/99 underline the Government's expectation that developers should provide the LPA with clear evidence that they have fully considered the use of existing masts, buildings and other structures before seeking to erect any new mast. If the evidence regarding the consideration of such alternative sites is not considered satisfactory, the LPA may be justified in refusing approval to the installation of the mast.

6.107 However, mast sharing is not always possible. Although an existing mast might be in close proximity to a proposed site, the precise location and height of the existing mast may not be compatible with the operator's network. The size of the mast will also affect the ability to mast share; smaller masts may not be suitable for additional operators either because the structure is inadequate for the additional weight, or because there would be insufficient vertical separation between different sets of antennas to avoid interference. In addition, LPAs may consider the merits of mast sharing on a case-by-case basis. In some locations it may appear that a single large mast would have less impact, whilst in others it may be considered that several smaller masts, even in close proximity, are less visually intrusive.

6.108 The Government is keen to encourage early discussions between the operator and the LPA about proposed telecommunications development. Under a Code of Best Practice, issued by DETR in 1998, for telecommunications prior approval procedures in England and Wales, operators are encouraged to provide information to the LPA on significant installation plans in the LPA area and to undertake informal discussions on these plans. Close consultation between the operator and LPA before an application for consent for telecommunications development is made will allow the two sides to examine locally not only alternative mast locations, including

opportunities for site sharing, but also different design solutions. We understand that consideration is being given to extending this to include health concerns.

6.109 Draft guidance to LPAs in drawing up development plan policies or deciding planning applications for development giving rise to electromagnetic fields, such as telecommunications base stations, is contained in the joint DETR/DH draft circular, "Land-use Planning and Electromagnetic Fields (EMFs)", issued for consultation in December 1998. A similar consultation exercise was carried out in Wales by the then Welsh Office. The circulars will be finalised as soon as practicable. In July 1999 the then Minister for Public Health and the then Minister for Planning jointly wrote to all Members of Parliament for constituencies in England, and to all Council Leaders in England, setting out the Government position about the possibility of adverse health effects associated with telecommunications base stations. In October 1999, a similar letter was issued in Wales by the two Assembly Secretaries with responsibility for health and planning to all Members of Parliament for constituencies in Wales, all Assembly Members and to all Council Leaders in Wales.

Scotland

6.110 In Scotland broadly similar permitted development rights apply to telecommunications development but with a number of significant differences. For example, the restrictions on permitted development rights for masts in certain designated areas currently relate only to masts for "microwave antennas" (as defined in Scottish planning legislation) in National Scenic Areas and conservation areas. There are no prior approval procedures in Scotland for telecommunications permitted development, the system in Scotland relying more upon the obligations contained in the telecommunications code system operator's licence. This is also true in relation to the general condition requiring the removal of redundant telecommunications equipment that has benefited from permitted development rights.

6.111 The Scottish Executive intends to increase the controls on permitted development rights in Scotland to a level approximating that in England and Wales, including the introduction of a prior approval regime. Work on the relevant legislative amendments is in hand and will be considered by the Scottish Parliament in due course. New guidance will also be issued to update that currently contained in the Scottish Executive's Circulars 25/1985 and 5/1992. Similarly, a Code of Best Practice for telecommunications prior approval procedures, in the form of a Planning Advice Note, is also intended. These documents will reflect the policies mentioned at paragraphs 6.104–6.109 above. The Scottish Executive produced its own version of the draft circular on "Land-use Planning and Electro-Magnetic Fields", which will also be finalised as soon as practicable.

6.112 The Scottish Parliament's Transport and the Environment Committee recently produced a report entitled "Planning Procedures for Telecommunications Development" (Scottish Parliament, 2000). This was prompted by public concern about mobile phone masts and the Scottish Executive's proposals to increase controls on permitted development rights (see paragraph 6.111). The Committee concluded that the existing system was inadequate and that change was required. It expressed significant concern about the prior approvals process and recommended the introduction of full planning controls. In addition, the Committee identified three key factors that should inform policy in this area: amenity, health, and a precautionary approach.

6.113 In relation to amenity, the Transport and Environment Committee concluded that environmental impact could be minimised through early discussion of strategic network requirements, site sharing, mast sharing, design and disguise, and the introduction of national roaming. Although guidance was required to minimise the impact of development on environmentally sensitive

areas, this should allow for local flexibility. In relation to health the Committee decided that whilst there was no conclusive scientific evidence for non-thermal effects, there was reasonable doubt about health risks, and it therefore recommended that health should be a material planning consideration. It also recommended adopting a precautionary approach, and urged planning authorities to consider a hierarchy of preferred locations that would favour development in sparsely populated areas, such as industrial sites. However, the Committee concluded that there was insufficient evidence to justify a *cordon sanitaire*.

6.114 The Committee recommended the development of a national policy framework in consultation with interested bodies, including telecommunications developers and operators. Within this framework the Committee identified a need for clear guidance based on a precautionary approach.

Northern Ireland

6.115 In Northern Ireland planning decisions are the responsibility of the Planning Service, a Next Steps Agency of the Department of the Environment. This Agency has six divisional offices and two subdivisional offices, each covering a number of district council areas.

6.116 As in England and Wales, larger developments, such as masts over 15 m high, require full planning permission. However, telecommunications code system operators enjoy permitted development rights for a range of developments under the Planning (General Development) Order (Northern Ireland) 1993, as amended by the Planning (General Development) (Amendment) Order (Northern Ireland) 1998. These rights are subject to limitations and conditions to protect amenity and the environment, and essentially mirror those in England and Wales.

6.117 Some permitted developments, such as erection of masts up to 15 m high, are conditional upon prior approval by the Planning Service. Under this procedure, the Planning Service has 28 days in which to whether to approve the site and appearance of the installation. Where it considers that the development would pose a threat to amenity, the Planning Service may refuse approval. In England and Wales the prior approvals process was modified by the Town and Country Planning (General Permitted Development) (Amendment) Order 1999, and similar changes are currently being introduced in Northern Ireland. It is expected that these changes will be implemented in 2000, and will extend to 42 days the period allowed for notification that prior approval is required, and the decision on whether to approve the siting and appearance of the installation.

7

References

Adair E R (1983). Sensation, subtleties and standards: Synopsis of a panel discussion. IN *Microwaves and Thermoregulation* (E R Adair, Ed). New York, Academic Press, p 231.

Adair E R, Cobb B L, Mylacraine K S and Kelleher S A (1999). Human exposure at two radiofrequencies (450 and 2450 MHz): similarities and differences in physiological response. *Bioelectromagnetics*, **20,** 12.

Adair R K (1994). Effects of weak high-frequency electromagnetic fields on biological systems. IN *Radiofrequency Radiation Standards* (B J Klauenberg, M Grandolfo and D N Erwin, Eds). New York, Plenum Press, p 207. (The intensity of 10 mW/m^2 quoted in the penultimate paragraph on p 218 should be 10 mW/cm^2 – Adair, private communication.)

Adey W R (1981). Tissue interactions with nonionizing electromagnetic fields. *Physiol Rev*, **61**, 435.

Adey W R (1989). The extracellular space and energetic hierarchies in electrochemical signalling between cells. IN *Charge and Field Effects in Biosystems 2* (M J Allen, S F Cleary and F M Hawkridge, Eds). New York, Plenum Press, p 264.

Adey W R (1993). Biological effects of electromagnetic fields. *J Cell Biochem*, **5**, 410.

Adey W R, Bawin S M and Lawrence A F (1982). Effects of weak amplitude-modulated microwave fields on calcium efflux from awake cat cerebral cortex. *Bioelectromagnetics*, **3**, 295.

Adey W R, Byus C V, Cain C D, Higgins R J, Jones R A, Kean C J, Kuster N, MacMurray A, Stagg R B, Zimmerman G, Phillips J L and Haggren W (1999). Spontaneous and nitrosourea-induced primary tumours in Fischer 344 rats chronically exposed to 836 MHz modulated microwaves. *Radiat Res*, **152**, 293.

Ahlbom A and Feychting M (1999). Re: Use of cellular phones and the risk of brain tumours: a case–control study. *Int J Oncol,* **15,** 1045.

Alam M T, Barthakur N, Lambert N G and Kasatiya S S (1978). Cytological effects of microwave radiation in Chinese hamster cells *in vitro*. *Can J Genet Cytol*, **20**, 23.

Albert E N (1977). Light and electron microscopic observations on the blood–brain barrier after microwave irradiation. IN *Symposium on Biological Effects and Measurement of Radio Frequency/Microwaves* (D G Hazzard, Ed). Rockville, Maryland, US Department of Health, Education and Welfare, HEW Publication (FDA) 8026, p 294.

Albert E N (1979). Reversibility of microwave-induced blood–brain barrier permeability. *Radio Sci*, **14(S),** 323.

Albert E N and Kerns J M (1981). Reversible microwave effects on the blood–brain barrier. *Brain Res*, **230**, 153.

Albert E N, Slaby F, Roche J and Loftus J (1987). Effect of amplitude modulated 147 MHz radiofrequency radiation on calcium ion efflux from avian brain tissue. *Radiat Res*, **109**, 19.

Alm H and Nilsson L (1994). Changes in driver behaviour as a function of handsfree mobile phones – a simulator study. *Accid Anal Prev*, **26**, 441.

Alm H and Nilsson L (1995). The effects of a mobile telephone task on driver behaviour in a car following situation. *Accid Anal Prev*, **27**, 707.

Allis J W and Sinha-Robinson B L (1987). Temperature-specific inhibition of human cell Na+/K+ ATPase by 2450 MHz microwave radiation. *Bioelectromagnetics*, **8**, 203.

Anderstam B, Hamnerium Y, Hussain S and Ehrenberg L (1983). Studies of possible genetic effects in bacteria of high frequency electromagnetic fields. *Hereditas*, **98**, 11.

Antipenko E N and Koveshnikova I V (1987). Cytogenetic effects of microwaves of non-thermal intensity in mammals. *Dokl Akad Nauk SSSR*, **296**, 724.

Antonopoulos A, Eisenbrandt H and Obe G (1997). Effects of high-frequency electromagnetic fields on human lymphocytes *in vitro*. *Mutat Res*, **395**, 209.

Asanami S and Shimono K (1999). High body temperature induces micronuclei in mouse bone marrow. *Mutat Res*, **390**, 79.

Arber S L and Lin J C (1984). Microwave enhancement of membrane conductance: effects of EDTA, caffeine and tetracaine. *Physiol Chem Phys Med NMR*, **16**, 469.

Arber S L and Lin J C (1985). Microwave induced changes in nerve cells: effects of modulation and temperature. *Bioelectromagnetics*, **6**, 257.

Arkin H, Xu L X and Holmes K R (1994). Recent developments in modeling heat transfer in blood perfused tissues. *IEEE Trans Biomed Eng,* **41,** 97**.**

Athey T W and Krop B A (1980). Millimetre wave induction of lamda prophage – dependent on growth medium? Bioelectromagnetics Society, 2nd Annual Meeting, September 1980, San Antonio. *Bioelectromagnetics*, **1**, 241.

Averbeck D, Dardalhon M and Berteaud A J (1976). Microwaves action in procaryotic and eucaryotic cells and a possible interaction with x-rays. *J Microwave Power Electromag Energy*, **11**, 143.

Balcer-Kubiczek E K and Harrison G H (1985). Evidence for microwave carcinogenesis *in vitro*. *Carcinogenesis*, **6**, 859.

Balcer-Kubiczek E K and Harrison G H (1989). Induction of neoplastic transformation in C3H/10$T_{1/2}$ cells by 2.45-GHz microwaves and phorbol ester. *Radiat Res*, **117**, 531.

Balcer-Kubiczek E K and Harrison G H (1991). Neoplastic transformation of C3H/10$T_{1/2}$ cells following exposure to 120-Hz modulated 2.45 GHz microwaves and phorbol ester tumor promoter. *Radiat Res*, **126**, 65.

Balode Z (1996). Assessment of radio-frequency electromagnetic radiation by the micronucleus test in Bovine peripheral erythrocytes. *Sci Total Environ*, **180**, 81.

Baranski S, Arber S L and Lin J C (1972). Histological and histochemical effects of microwave irradiation on the central nervous system of rabbits and guinea pigs. *Am J Physiol Med*, **51**, 182.

Baum S J, Ekstrom M E, Skidmore W D, Wyant D E and Atkinson J L (1976). Biological measurements in rodents exposed continuously throughout their adult life to pulsed electromagnetic radiation. *Health Phys*, **30**, 161.

Bawin S M, Gavalas-Medici R J and Adey W R (1973). Effects of modulated very high frequency fields on specific brain rhythms in cats. *Brain Res*, **58**, 365.

Bawin S M, Gavalas-Medici R J and Adey W R (1974). Reinforcement of transient brain rhythms by amplitude-modulated VHF fields. IN *Biological and Clinical Effects of Low Frequency Magnetic and Electric Fields* (J G Llaurado, A Sances and J H Battocletti, Eds). Springfield, Charles C Thomas, p 172.

Bawin S M, Kaczmarek L K and Adey W R (1975). Effects of modulated VHF fields on the central nervous system. *Ann NY Acad Sci*, **247**, 74.

Beechey C V, Brooker D, Kowalczuk C I, Saunders R D and Searle A G (1986). Cytogenetic effects of microwave irradiation on male germ cells of the mouse. *Int J Radiat Biol*, **50**, 909.

Belyaev I Ya (1992). Some biophysical aspects of the genetic effect of low-intensity millimeter waves. *Bioelectrochem Bioenerg*, **27**, 11.

Beral V (2000). *Personal communication*.

Berman E, Kinn J B and Carter H B (1978). Observations of mouse fetuses after irradiation with 2.45 GHz microwaves. *Health Phys*, **35**, 791.

Berman E, Carter H B and House D (1980). Tests for mutagenesis and reproduction in male rats exposed to 2450 MHz (CW) microwaves. *Bioelectromagnetics*, **1**, 65.

Blackman C F, Surles M C and Benane S G (1976). The effect of microwave exposure on bacteria: mutation induction. IN *Biological Effects of Electromagnetic Waves*. Selected papers of the UNSC/URSI Annual Meeting, Boulder, Colorado, October 1975 (C C Johnson and M L Shore, Eds). Rockville, Maryland, US Department of Health, Education and Welfare, Volume 1, p 406.

Blackman C F, Elder J A, Weil C M, Benane S G, Eichinger D C and House D E (1979). Induction of calcium-ion efflux from brain tissue by radiofrequency radiation: effects of modulation frequency and field strength. *Radio Sci*, **14(S),** 93.

References

Blackman C F, Benane S G, Elder J A, House D E, Lampe J A and Faulk J M (1980a). Induction of calcium-ion efflux from brain tissue by radiofrequency radiation: effect of sample number and modulation frequency on the power-density window. *Bioelectromagnetics*, **1**, 35.

Blackman C F, Benane S G, Joines W T, Hollis M A and House D E (1980b). Calcium-ion efflux from brain tissue: power-density versus internal field-intensity dependencies at 50-MHz RF radiation. *Bioelectromagnetics*, **1**, 277.

Blackman C F, Kinney L S, House D E and Joines W T (1989). Multiple power density windows and their possible origin. *Bioelectromagnetics*, **10(2)**, 115.

Blackwell R P and Saunders R D (1986). The effects of low-level radiofrequency and microwave radiation on brain tissue and animal behaviour. *Int J Radiat Biol,* **50**, 761.

Blevins R D, Crenshaw R C, Houghland A E and Clark C E (1980). The effects of microwave radiation and heat on specific mutants of Salmonella typhimurium LT2. *Radiat Res*, **82**, 511.

Bohr H and Bohr J (2000). Microwave enhanced kinetics observed in ORD studies of a protein. *Bioelectromagnetics*, **21**, 68.

Borbely A A, Huber R, Graf T, Fuchs B, Gallmann E and Achermann P (1999). Pulsed high-frequency electromagnetic field affects human sleep and sleep electroencephalogram. *Neurosci Lett*, **275**, 207.

Braune S, Wrocklage C Raczek J, Gailus T and Lucking C H (1998a). Resting blood pressure increase during exposure to a radiofrequency electromagnetic field. *Lancet*, **351**, 1857.

Braune S, Wrocklage C Raczek J, Gailus T and Lucking C H (1998b). Radiofrequency electromagnetic field from mobile phones. *Lancet*, **352**, 576.

Brookhuis K A, De Vries G and de Waard D (1991). The effects of mobile telephoning on driving performance. *Accid Anal Prev*, **23**, 309.

Brown D, Tickner A H and Simmonds D C V (1969). Interference between concurrent tasks of driving and telephoning. *J Appl Psychol*, **53**, 419.

Brusick D, Albertini R, McRee D, Peterson D, Williams G, Hanawalt P and Preston J (1998). Genotoxicity of radiofrequency radiations. *Environ Mol Mutagen*, **32**, 1.

Brück K and Hinckel P (1990). Thermoafferent networks and their adaptative modifications. *Encycl Pharmacol Ther*, **6**, 129.

Byus C V and Hawel L (1997). Additional considerations about bioeffects. IN *Mobile Communications Safety* (Q Balzano and J C Lin, Eds). London, Chapman and Hall, p 133.

Byus C V, Kartun K, Pieper S and Adey W R (1988). Increased ornithine decarboxylase activity in cultured cells exposed to low energy modulated microwave fields and phorbol ester tumor promoters. *Cancer Res*, **48**, 4222.

Cain C D, Thomas D L and Adey W R (1997). Focus formation of C3H/10$T_{1/2}$ cells and exposure to a 836.55 MHz modulated radiofrequency field. *Bioelectromagnetics*, **18**, 237.

Cantor K P, Dosemeci M, Brinton L A and Stewart P A (1995). Re: Breast cancer mortality among female electrical workers in the United States. *J Natl Cancer Inst,* **87**, 227.

Carpenter R L (1979). Ocular effects of microwave radiation. *Bull NY Acad Sci*, **55**, 1048.

Chadwick P J (1998). Occupational exposures to electromagnetic fields: the practical application of NRPB guidelines. Chilton, NRPB-R301.

Chapman S and Schofield W N (1998a). Lifesavers and Samaritans: emergency use of cellular (mobile) phones in Australia. *Accid Anal Prev*, **30**, 815.

Chapman S and Schofield W N (1998b). Emergency use of cellular (mobile) telephones. *Lancet*, **351,** 650.

Chagnaud J-L, Moreau J-M and Veyret B (1999). No effect of short-term exposure to GSM-modulated low-power microwaves on benzo(α)pyrene-induced tumours in rat. *Int J Radiat Biol,* **75(10)**, 1251.

Chen K M, Samuel A and Hoopingarner R (1974). Chromosomal aberration of living cells induced by microwave radiation. *Environ Lett*, **6**, 37.

Chernovetz M E, Justesen D R and Oke A F (1977). A teratological study of the rat: microwave and infrared radiations compared. *Radio Sci*, **12S**, 191.

Chizhenkova R A and Safroshkina A A (1996). Electrical reactions of brain to microwave irradiation. *Electro-Magnetobiology*, **15**, 253.

Chou C-K and Guy A W (1978). Effects of electromagnetic fields on isolated nerve and muscle preparation. *IEEE Trans Microwave Theory Tech*, **26,** 141.

Chou C-K and Guy A W (1979). Microwave-induced auditory responses in guinea pigs: relationship of threshold and microwave-pulse duration. *Radio Sci*, **14,** 193.

Chou C-K, Guy A W and Galambos R (1982). Auditory perception of radiofrequency electromagnetic fields. *J Acoust Soc Am*, **71,** 1321.

Chou C-K, Guy A W, Borneman L E, Kunz L L and Kramar P (1983). Chronic exposure of rabbits to 0.5 and 5 mW/cm^2 2450-MHz CW microwave radiation. *Bioelectromagnetics*, **4(1)**, 63.

Chou C K, Yee K C and Guy A W (1985). Auditory response in rats exposed to 2,450 MHz electromagnetic fields in a circularly polarized waveguide. *Bioelectromagnetics*, **6**, 323.

Chou C-K, Guy A W, Kunz L L, Johnson R B, Crowley J J and Krupp J H (1992). Long-term, low-level microwave irradiation of rats. *Bioelectromagnetics*, **13**, 469.

Ciaravino V, Meltz M L and Erwin D N (1987). Effects of radiofrequency radiation and simultaneous exposure with mitomycin C on the frequency of sister chromatid exchanges in Chinese hamster ovary cells. *Environ Mutagen*, **9**, 393.

Ciaravino V, Meltz M L and Erwin D N (1991). Absence of a synergistic effect between moderate-power radio-frequency electromagnetic radiation and adriamycin on cell-cycle progression and sister-chromatid exchange. *Bioelectromagnetics*, **12**, 289.

Cleary S F (1990a). Cellular effects of radiofrequency electromagnetic fields. IN *Biological Effects and Medical Applications of Electromagnetic Energy* (O P Gandhi, Ed). Englewood Cliffs, New Jersey, Prentice-Hall, p 339.

Cleary S F (1990b). Biological effects of radiofrequency electromagnetic fields. IN *Biological Effects and Medical Applications of Electromagnetic Energy* (O P Gandhi, Ed). Englewood Cliffs, New Jersey, Prentice-Hall, p 236.

Cleary S F (1995). Effects of radiofrequency radiation on mammalian cells and biomolecules *in vitro*. IN *Electromagnetic Fields: Biological Interactions and Mechanisms* (M Blank, Ed). Washington, American Chemical Society, p 467.

Cleary S F, Liu L-M and Merchant R E (1990a). Glioma proliferation modulated *in vitro* by isothermal radiofrequency radiation exposure. *Radiat Res*, **121**, 38.

Cleary S F, Liu L-M and Merchant R E (1990b). *In vitro* lymphocyte proliferation induced by radiofrequency electromagnetic radiation under isothermal conditions. *Bioelectromagnetics*, **11**, 47.

Cleary S F, Cao G and Liu L M (1996). Effects of isothermal 2450 MHz microwave radiation on the mammalian cell cycle: comparison with effects of isothermal 27 MHz radiofrequency radiation exposure. *Bioelectrochem Bioenerget*, **39**, 167.

Cohen S M and Ellwein, L B (1991). Genetic errors, cell proliferation and carcinogenesis. *Cancer Res*, **51**, 6493.

Cullinan W E, Herman J P, Battaglia D F, Akil H and Watson S J (1995). Pattern and time course of immediate early gene expression in rat brain following acute stress. *Neuroscience*, **64**, 477.

D'Andrea J A (1991). Microwave radiation absorption: behavioural effects. *Health Phys*, **61**, 29.

D'Andrea J A (1999). Behavioral evaluation of microwave irradiation. *Bioelectromagnetics*, **20**, 64.

D'Andrea J A, Gandhi O M, Kesner R P (1976). Behavioral effects of resonant electromagnetic power absorption in rats. IN *Biological Effects of Electromagnetic Waves*. Selected papers of the UNSC/URSI Annual Meeting, Boulder, Colorado, October 1975 (C C Johnson and M L Shore, Eds). Rockville, Maryland, US Department of Health, Education and Welfare, Volume 1, p 257.

D'Andrea J A, Gandhi O P and Lords J L (1977). Behavioral and thermal effects of microwave radiation at resonant and non-resonant wavelengths. *Radio Sci*, **12**, 251.

D'Andrea J A, Gandhi O P, Lords J L, Durney C H, Johnson C C and Astle L (1979). Physiological and behavioral effects of chronic exposure to 2450 MHz microwaves. *J Microwave Power*, **14,** 351.

References

D'Andrea J A, Gandhi O P, Lords J L, Durney C H, Astle L, Stensaas S and Schoenberg A A (1980). Physiological and behavioral effects of prolonged exposure to 915 MHz microwaves. *J Microwave Power*, **15**, 123.

D'Andrea J A, Dewitt J R, Gandhi O P, Stensaas S, Lords J L and Neilson H C (1986a). Behavioral and physiological effects of chronic 2450 MHz microwave irradiation of the rat at 0.5 mW/cm^2. *Bioelectromagnetics*, **7**, 45.

D'Andrea J A, Dewitt J R, Emmerson R Y, Bailey C, Stensaas S and Gandhi O P (1986b). Intermittent exposure of rats to 2450 MHz microwaves at 2.5 mW/cm^2. Behavioural and physiological effects. *Bioelectromagnetics*, **7**, 315.

D'Andrea J A, Thomas A, Hatcher D J and DeVietti T L (1992). Rhesus monkey contrast sensitivity during exposure to high peak power 5.6-GHz microwave pulses. IN *Abstracts, The First World Congress for Electricity and Magnetism in Biology and Medicine*, June 1992, Orlando, Florida, p 62.

Danniells C, Duce I, Thomas D, Sewell P, Tattersall J and de Pomerai D (1998). Transgenic nematodes as biomonitors of microwave-induced stress. *Mutat Res,* **399**, 55.

Dardalhon M, Averbeck D and Berteaud A J (1981). Studies on possible genetic effects of microwaves in procaryotic and eucaryotic cells. *Radiat Environ Biophys*, **20**, 37.

Davis R L and Mostofi F K (1993). Cluster of testicular cancer in police officers exposed to hand-held radar. *Am J Ind Med*, **24,** 231.

De Lorge J O (1984). Operant behavior and colonic temperature of Macaca mulatta exposed to radio frequency fields at and above resonant frequencies. *Bioelectromagnetics*, **5**, 233.

De Lorge J O and Ezell C S (1980). Observing responses of rats exposed to 1.28- and 5.62-GHz microwaves. *Bioelectromagnetics*, **1**, 183.

de Pomerai D I, Mutwakil M H A Z, Henshaw J and Emerson L (1999). Mobile 'phone effects on nematode worms: animal *in vivo* studies using *Caenorhabditis elegens*. Presented at Mobile Phones and Health Conference, September 1999, Gothenburg, Sweden.

DETR (Department of the Environment, Transport and the Regions) (1998). Department of the Environment, Transport and the Regions and the National Assembly for Wales Code of Best Practice. Telecommunications prior approval procedures as applied to mast/tower development.

DETR (Department of the Environment, Transport and the Regions) (2000). Government targets mobile phone drivers in new campaign. Press notice 0041, 21 January 2000.

Dimbylow P J and Mann S M (1994). SAR calculations in an anatomically realistic model of the head for mobile communication transceivers at 900 MHz and 1.8 GHz. *Phys Med Biol*, **39**, 1537.

D'Inzeo G, Bernardi P, Eusebi F, Grassi F, Tamburello C and Zani B M (1988). Microwave effects on acetylcholine-induced channels in cultured chick myotubes. *Bioelectromagnetics*, **9**, 363.

Dreyer N A, Loughlin J E and Rothman K J (1999). Cause-specific mortality in cellular telephone users. *JAMA*, **282**, 1814.

Dolk H, Shaddick G, Walls P, Grundy C, Thakrar B, Kleinschmidt L and Elliott P (1997a). Cancer incidence near radio and television transmitters in Great Britain. 1: Sutton Coldfield transmitter. *Am J Epidemiol*, **145,** 1.

Dolk H, Elliott P, Shaddick G, Walls P and Thakrar B (1997b). Cancer incidence near radio and television transmitters in Great Britain. 2: All high power transmitters. *Am J Epidemiol*, **145,** 10.

Drogichina E A, Konchalovskaya N M, Glotova K V, Sadcikova N M and Snegova G V (1966). On the problems of autonomic and cardiovascular disturbances under the effects of ultrahigh frequency electromagnetic fields. ATD Report 66-124, Library of Congress, Aerospace Technology Division. *Gig Tr Prof Zabol,* **10**, 13.

DTI (1999). Electromagnetic compatibility aspects of radio-based mobile telecommunications. LINK Research Communications Programme. London, Department of Trade and Industry.

Dutta S K, Nelson W H, Blackman C F and Brusick D J (1979). Lack of microbial genetic response to 2.45-GHz CW and 8.5- to 9.6-GHz pulsed microwaves. *J Microwave Power Electromag Energy*, **14**, 275.

Dutta S K, Subramoniam A, Ghosh B and Parshad R (1984). Microwave radiation induced calcium ion efflux from human neuroblastoma cells in culture. *Bioelectromagnetics*, **5**, 71.

Dutta S K, Ghosh B and Blackman C F (1989). Radiofrequency radiation-induced calcium ion efflux enhancement from human and other neuroblastoma cells in culture. *Bioelectromagnetics*, **10(2)**, 197.

Dutta S K, Das K, Ghosh B and Blackman C F (1992). Dose dependence of acetylcholinesterase activity in neuroblastoma cells expopsed to modulated radio-frequency electromagnetic radiation. *Bioelectromagnetics*, **13**, 317.

EC (European Commission) (1996). Possible Health Effects Related to the Use of Radiotelephones. Proposals for a Research Programme by a European Commission Expert Group. Brussels, EC.

EC (European Commission) (1999). Council Recommendation of 12 July 1999 on the Limitation of Exposure of the General Public to Electromagnetic Fields (0 Hz to 300 GHz). *Off J Eur Commun*, L199, 59 (1999/519/EC).

EC (European Commission) (2000). Communication from the Commission on the Precautionary Principle. Brussels, EC, COM(2000)1, February 2000.

Edwards G S, Davis C C, Saffer J D and Swicord, M L (1984). Resonant microwave absorption of selected DNA molecules. *Phys Rev Lett*, **53(13)**, 1284.

Elwood J M (1999). A critical review of epidemiologic studies of radiofrequency exposure and human cancers. *Environ Health Perspect*, **107,** 155.

Erwin D E and Hurt W (1993). Biological effects of ultrawideband emission. IN *EMP Human Health Effects Science Review Panel Proceedings*, 16–18 March 1993. Washington DC, Theater Nuclear Warfare Program and Operational Medicine and Fleet Support, Bureau of Medicine and Surgery, p 91.

Eulitz C, Ullsperger P, Freude G and Elbert T (1998). Mobile phones modulate response patterns of human brain activity. *NeuroReport,* **9**, 3229.

FEI (Federation of the Electronics Industry) (2000). *Personal communication.*

Fesenko E E, Makar V R, Novoselova E G and Sadovnikov V B (1999). Microwaves and cellular immunity. I. Effect of whole body microwave irradiation on tumour necrosis factor production in mouse cells. *Bioelectrochem Bioenerg,* **49(1)**, 29.

Foster K R (2000a). *Personal communication*.

Foster K R (2000b). *Personal communication*.

Foster K R, Epstein B R and Gealt M A (1987). *Resonances* in the dielectric absorption of DNA? *Biophy J*, **52**, 421.

Frei M R, Berger R E, Dusch S J, Guel V, Jauchem J R, Merritt J H and Stedham M A (1998a). Chronic exposure of cancer-prone mice to low-level 2450 MHz radiofrequency radiation. *Bioelectromagnetics*, **19**, 20.

Frei M R, Jauchem J R, Dusch S J, Merritt J H, Berger R E and Stedham M A (1998b). Chronic, low-level (1.0 W/kg) exposure of mice prone to mammary cancer to 2450 MHz microwaves*. Radiat Res,* **150**, 568.

Frey A H, Feld S R and Frey B (1975). Neural function and behaviour: defining the relationship. *Ann NY Acad Sci*, **247**, 433.

Freude G, Ullsperger P, Eggert S and Ruppe I (1998). Effects of microwaves emitted by cellular phones on human slow brain potentials. *Bioelectromagnetics*, **19**, 384.

Fritze K, Sommer C, Schmitz B, Mies G, Hossman K-A, Kiessling M and Wiessner C (1997a). Effect of global system for mobile communication (GSM) microwave exposure on blood–brain permeability in rat. *Acta Neuropathol*, **94**, 465.

Fritze K, Wiessner C, Kuster N, Sommer C, Gass P, Hermann D M, Kiessling M and Hossmann K-A (1997b). Effect of Global system for mobile communication microwave exposure on the genomic response of the rat brain. *Neuroscience,* **81(3)**, 627.

Fröhlich H (1968). Long-range coherence and energy storage in biological systems. *Int J Quantum Chem*, **II**, 641.

Fröhlich H (1980). The biological effects of microwaves and related questions. *Adv Electronics Electron Phys*, **53**, 85.

Fröhlich H (1986). Coherent excitation in active biological systems. In *Modern Bioelectrochemistry* (F Gutmann and H Keyzer, Eds). New York, Plenum Press, p 241.

Furia L, Hill D W and Gandhi O P (1986). Effect of millimeter-wave irradiation on growth of *Saccharomyces cerevisiae*. *IEEE Trans Biomed Eng*, **BME-33**, 993.

Gabriel C (2000). *Personal communication.*

Gabriel C, Grant E H, Tata R, Brown P R, Gestblom B and Noreland E (1987). Microwave absorption in aqueous solutions of DNA. *Nature*, **328**, 145.

References

Galvin M J, Parks D L and McRee D L (1981). Influence of 2.45 GHz microwave radiation on enzyme activity. *Radiat Environ Biophys*, **19**, 149.

Gandhi C R and Ross D H (1989). Microwave induced stimulation of ^{32}P incorporation into phosphoinositides of rat brain synaptosomes. *Radiat Environ Biophys*, **28**, 223.

Garaj-Vrhovac V (1999). Micronucleus assay and lymphocyte mititic activity in risk assessment of occupational exposure to microwave radiation. *Chemosphere*, **39(13)**, 2301.

Garaj-Vrhovac V, Horvat D and Koren Z (1990a). The effect of microwave radiation on the cell genome. *Mutat Res*, **243**, 8.

Garaj-Vrhovac V, Horvat D and Koren Z (1990b). Comparison of chromosome aberration and micronucleus induction in human lymphocytes after occupational exposure to vinyl chloride monomer and microwave radiation. *Periodicum Biologorium*, **92**, 411.

Garaj-Vrhovac V, Horvat D and Koren Z (1991). The relationship between colony-forming ability, chromosome aberrations and incidence of micronuclei in V79 Chinese hamster cells exposed to microwave radiation. *Mutat Res*, **263**, 143.

Garaj-Vrhovac V, Fucic A and Horvat D (1992). The correlation between the frequency of micronuclei and specific chromosome aberrations in human lymphocytes exposed to microwave radiation *in vitro*. *Mutat Res*, **281**, 181.

Garland F C, Gorham E, Garland C and Ferns J A (1988). Non-Hodgkin's lymphomas in US Navy personnel. *Arch Environ Health*, **43,** 425.

Garland F C, Shaw E, Gorham E D, Garland C F, White M R and Sinsheimer P J (1990). Incidence of leukaemia in occupations with potential electromagnetic field exposure in United States Navy personnel. *Am J Epidemiol*, **132**, 293.

Garson O M, McRobert T L, Campbell L J, Hocking B A and Gordon I (1991). A chromosomal study of worked with long-term exposure to radiofrequency radiation. *Med J Aust*, **155**, 289.

Goldsmith, J R (1995). Epidemiological evidence of radiofrequency radiation (microwave) effects on health in military, broadcasting and occupational studies. *Int J Occup Environ Health*, **1**, 47.

Gordon Z V (1966). Problems of industrial hygiene and the biological effects of electromagnetic super-high frequency fields. *Medicina* (in Russian).

Gos P, Eicher B, Kohli J and Heyer W-D (1997). Extremely high frequency electromagnetic fields at low power density do not affect the division of exponential phase *Saccharomyces cerevisiae* cells. *Bioelectromagnetics*, **18**, 142.

Goswami P C, Albee L D, Parsian A J, Baty J D, Moros E G, Pickard W F, Roti Roti J L and Hunt C R (1999). Proto-oncogene mRNA levels and activities of multiple transcription factors in C3H10T½ murine embryonic fibroblasts exposed to 835.62 and 847.74 MHz cellular phone communication frequency radiation. *Radiat Res*, **151**, 300.

Gottlob L R and Madden D J (1999). Age differences in the strategic allocation of visual attention. *J Gerontol B Psychol Sci Soc Sci*, **54**, 165.

Goud G N, Usha Rani M U, Reddy P P, Reddi O S, Rao M S and Saxena V K (1982). Genetic effects of microwave radiation in mice. *Mutat Res*, **103**, 39.

Grayson J K (1996). Radiation exposure, socioeconomic status and brain tumor risk in US Air Force: a nested case–control study. *Am J Epidemiol*, **143**, 480.

Grin A N (1974). Effects of microwave on catecholamine metabolis in brain. US Joint Pub Research Device Rep, JPRS 72066.

Grundler W, Kaiser F, Keilmann F and Walleczek J (1992). Mechanisms of electromagnetic interaction with cellular systems. *Naturwissenschaften*, **79**, 551.

Guy A W, Chou C-K, Kunz L L, Crowley J and Krupp J (1985). *Effects of Long-term Low-level Radiofrequency Radiation Exposure on Rats.* Volume 9. Summary. Brooks Air Force Base, Texas, USAF School of Aerospace Medicine (USAFSAM TR 85-11).

Guy A W, Kramar P O, Harris C A and Chou C K (1980). Long-term 2450-MHz CW microwave irradiation of rabbits: methodology and evaluation of ocular and physiologic effects. *J Microwave Power*, **15**, 37.

Haidler T, Knasmueller S, Kundi M and Haidler M (1994). Clastogenic effects of radiofrequency radiations on chromosomes of Tradescantia. *Mutat Res*, **324**, 65.

Haigney D (2000). Mobile phone use whilst driving: the safest setup? *Personal communication*.

Hamnerius Y, Rasmuson A and Rasmuson B (1985). Biological effects of high-frequency electromagnetic fields on *Salmonella typhimurium* and *Drosophila melanogaster*. *Bioelectromagnetics*, **6**, 405.

Hansson Mild K, Oftedal G, Sandström M, Wilén J, Tynes T, Haugsdal B and Hauger E (1998). Comparison of symptoms experienced by users of analogue and digital mobile phones. A Swedish–Norwegian epidemiological study. Arbetslivsrapport 1998:23. Solna, Sweden, Arbetslivsinstitutet.

Hardell L, Nasman A, Pahlson A, Hallquist A and Hansson Mild K (1999). Use of cellular telephones and the risk for brain tumours: a case–control study. *Int J Oncol*, **15**, 113.

Hayes R B, Brown L M, Pottern L M, Gomez M, Kardaun J W, Hoover R N, O'Connell K J, Sutzman R E and Javadpour N (1990). Occupation and risk for testicular cancer: a case–control study. *Int J Epidemiol,* **19**, 825.

Hermann D M and Hossman K-A (1997). Neurologic effects of microwave exposure related to mobile communication. *J Neurol Sci*, **152**, 1.

Hibshoosh H, Johnson M and Weinstein I B (1991). Effects of overexpression of ornithine decarboxylase (ODC) on control of oncogene-induced cell transformation. *Oncogene*, **6**, 739.

Higashikubo R, Culbreath V O, Spitz D R, LaRegina M C, Pickard W F, Straube W L, Moros E G and Roti Roti J L (1999). Radiofrequency electromagnetic fields have no effect on the *in vivo* proliferation of the 9L brain tumour. *Radiat Res*, **152**, 665.

Hocking B, Gordon I R, Grain H L and Hatfield G E (1996). Cancer incidence and mortality and proximity to TV towers. *Med J Aust*, **165**, 601.

Hocking B (1998). Preliminary report: symptoms associated with mobile phone use. *Occup Med*, **48**, 357.

Holly E A, Aston D A, Ahn D K and Smith A H (1996). Intraocular melanoma linked to occupations and chemical exposures. *Epidemiology,* **7,** 55.

Huang A T, Engle M E, Elder J A, Kinn J B and Ward T R (1977). The effect of microwave radiation (2450 MHz) on the morphology and chromosomes of lymphocytes. *Radio Sci*, **12(S)**, 173.

Hunt E L, King N W and Phillips R D (1975). Behavioral effects of pulsed microwave radiation. *Ann NY Acad Sci*, **247**, 440.

Hyland, G J (1998). Non-thermal bioeffects induced by low-intensity microwave irradiation of living systems. *Engineer Sci Educ J*, **7**, 261.

ICNIRP (1996). Health issues related to the use of hand-held radiotelephones and base transmitters. *Health Phys*, **70,** 587.

ICNIRP (1998a). Guidelines for limiting exposure to time-varying electric, magnetic and electromagnetic fields (up to 300 GHz). *Health Phys*, **74(4)**, 494.

ICNIRP (1998b). Response to questions and comments on ICNIRP guidelines. *Health Phys*, **75(4)**, 438.

IEEE/ANSI (1991). Standard for safety levels with respect to human exposure to radiofrequency electromagnetic fields, 3 kHz to 300 GHz. New York, IEEE, C95.1-1991.

INIRC (1988). Guidelines on limits of exposure to radiofrequency electromagnetic fields in the frequency range from 100 kHz to 300 GHz. *Health Phys*, **54(1)**, 115.

INIRC (1990). Interim guidelines on limits of exposure to 50/60 Hz electric and magnetic fields. *Health Phys*, **58(10)**, 113.

Imaida K, Taki M, Yamaguchi T, Ito T, Watanabe S, Wake K, Aimoto A, Kamimura Y, Ito N and Shirai T (1998a). Lack of promoting effects of the electromagnetic near-field used for cellular phones (929.2 MHz) on rat liver carcinogenesis in a medium-term liver bioassay. *Carcinogenesis*, **19**, 311.

Imaida K, Taki M, Watanabe S-i, Kamimura Y, Ito T, Yamaguchi T, Ito N and Shirai T (1998b). The 1.5 GHz electromagnetic near-field used for cellular phones does not promote rat liver carcinogenesis in a medium-term liver bioassay. *Jpn J Cancer Res*, **89**, 995.

Inaba R, Shishido K, Okada A and Moroji T (1992). Effects of whole body microwave exposure on the rat brain contents of biogenic amines. *Eur J Appl Physiol*, **65**,124.

References

Inskip P D, Hatch E E, Stewart P A, Heineman E F, Ziegler R G, Dosemeci M, Parry D, Rothman N, Boice J D Jr, Wilcosky T C, Watson D J, Shapiro W R, Selker R G, Fine H A, Black P McL, Loeffler J S and Linet M S (1999). Study design for a case–control investigation of cellular telephones and other risk factors for brain tumours in adults. *Radiat Prot Dosim*, **86,** 45.

Ivaschuck O I, Jones R A, Ishida-Jones T, Haggren W, Adey W R and Phillips J I (1997). Exposure of nerve growth factor-treated PC-12 rat pheochromocytoma cells to a modulated radiofrequency field at 836.55 MHz: effects on c-jun and c-fos expression. *Bioelectromagnetics*, **18**, 223.

Jauchem J R (1997). Exposure to extremely-low-frequency electromagnetic fields and radiofrequency radiation: cardiovascular effects on humans. *Int Arch Occup Environ Health*, **70(1)**, 9.

Jauchem J R (1998). Health effects of microwave exposures: a review of the recent (1995–1998) literature. *J Microwave Power Electromag Energy*, **33(4)**, 263.

Jauchem J R and Frei M R (1992). Heart rate and blood pressure changes during radiofrequency irradiation and environmental heating. *Comp Biochem Physiol A*, **101(1)**, 1.

Jauchem J R and Frei M R (1995). High-peak-power microwave pulses: effects on heart rate and blood pressure in unanaesthetised rats. *Aviat Space Environ Med*, **66(10)**, 992.

Jensh R P (1984a). Studies of the teratogenic potential of exposure of rats to 6000-MHz microwave radiation. I. Morphologic analysis at term. *Radiat Res*, **97**, 272.

Jensh R P (1984b). Studies of the teratogenic potential of exposure of rats to 6000-MHz microwave radiation. II. Postnatal psychophysiologic evaluations. *Radiat Res*, **97**, 282.

Jensch R P (1997). Behavioural teratologic studies using microwave radiation: is there an increased risk from exposure to cellular phones and microwave ovens? *Reproduct Toxicol*, **11(4)**, 601.

Jensh R P, Vogel W H and Brent R L (1983a). An evaluation of the teratogenic potential of protracted exposure of pregnant rats to 2450-MHz microwave radiation. I. Morphologic analysis at term. *J Toxicol Environ Health*, **11**, 23.

Jensh R P, Vogel W H and Brent R L (1983b). An evaluation of the teratogenic potential of protracted exposure of pregnant rats to 2450-MHz microwave radiation. II. Postnatal physiologic analysis. *J Toxicol Environ Health*, **11**, 37.

Johansen C and Olsen J H (1999). Cellular telephones, magnetic field exposure, risk of brain tumours and cancer at other sites: a cohort study. *Radiat Prot Dosim*, **83,** 155.

Johnson R B, Spackman D, Crowley J, Thompson D, Chou C-K, Kunz L L and Guy A W (1983). *Effects of Long-term Low-level Radiofrequency Radiation Exposure on Rats*. Volume 4. Open field behavior and corticosterone. Brooks Air Force Base, Texas, USAF School of Aerospace Medicine (USAFSAM TR 83-42).

Jokela K, Leszczynski D, Paile W, Salomaa S, Puranen L and Hyysalo P (1999). Radiation safety of handheld mobile phones and base stations. Stockholm, STUK - S161.

Jorritsma J B and Konings A W (1984). The occurrence of DNA strand breaks after hyperthermic treatments of mammalian cells with and without radiation. *Radiat Res*, **98**, 198.

Juutilainen J *et al* (1998). Effects of radiofrequency radiation on the development of cancer in mice. Presented at Bioelectromagnetics Society 20th Annual Meeting, St Petersburg, Florida, June 1998.

Juutilainen J, Heikinnen P, Kosma V-M, Hongisto T, Huuskonen H, Hyysalo P, Komulainen H, Kumlin T, Lahtinen T, Land S, Penttila I, Puranen L and Väänänen A (*in press*). Do pulse-modulated or continuous 900 MHz RF fields enhance the carcinogenic effect of ionising radiation in mice? IN *Proceedings 1st International Medical Scientific Congress "Non-Ionizing High-frequency EM Radiations: Researching the Epidemiology and Clinical Evidence"*, Rome, November 1999.

Kaiser F (1983). IN *Biological Effects and Dosimetry of Nonionizing Radiation, Radiofrequency and Microwave Energies* (M Grandolfo, S M Michaelson and A Rindi, Eds). New York, Plenum Press, p 251.

Kallén B, Malmquist G and Moritz U. Delivery outcome among physiotherapists in Sweden: is non-ionizing radiation a fetal hazard? *Arch Environ Health*, **37,** 81.

Kandel E R, Schwartz J H and Jessel T M (2000). *Principles of Neural Science* (4th edition). New York, McGraw-Hill Professional Publishing.

Kamimura Y, Saito K-i, Saiga T and Amenmiya Y (1994). Effect of 2.45 GHz microwave irradiation on monkey eyes. *IEICE Trans Commun*, **E77-B**, 762.

Kaplan I T, Metlay M, Zaret M M and Birenbaum L (1971). Absence of heart rate effects in rabbits during low-level microwave irradiation. *IEEE Trans Microwave Theory Tech*, **19**, 168.

Kerbacher J J, Meltz M L and Erwin D N (1990). Influence of radiofrequency radiation on chromosome aberrations in CHO cells and its interaction with DNA-damaging agents. *Radiat Res*, **123**, 311.

Khalil A M, Qassem W F and Suleiman M M (1993). A preliminary study on the radiofrequency field-induced cytogenetic effects in cultured human lymphocytes. *Dirasat*, **20**, 121.

Khillare B and Behari J (1998). Effect of amplitude-modulated radiofrequency radiation on reproduction pattern in rats. *Electro-Magnetobiology*, **17(1)**, 43.

Kiselev R I and Zalyubovskaya N P (1976). Study of inhibiting effect of superhigh frequency millimetre wave on adenoviruses. US Joint Pub Research Service Rep, JPRS L/5615, p 71.

Kittel A, Siklow L, Thuroczy G and Somosy Z (1996). Qualitative enzyme historchemistry and microanalysis revela changes in ultra-structural distribution of calcium and calcium activated ATPases after microwave irradiation of the medial habenula. *Acta Neuropathol*, **92**, 362.

Kohli M, Mei W N, Prohofsky E W and Van Zandt L L (1981). Calculated microwave absorption of double-helical B-conformation poly(dG).poly(dC). *Biopolymers*, **20**, 853.

Koivisto M, Revonsuo A, Krause C M, Haarala C, Sillanmäki L, Laine M and Hämäläinen H (2000). Effects of 902 MHz electromagnetic field emitted by cellular phones on response times in humans. *NeuroReport,* **11**, 413.

Koivisto M, Krause C M, Revonsuo A, Laine M and Hämäläinen H (*in press*). The effects of electromagnetic field emitted by GSM phones on working memory. *NeuroReport*.

Krause C M, Sillanmäki L, Koivisto M, Häggqvist A, Saarela C, Revonsuo A, Laine M and Hämäläinen H (2000). Effects of electromagnetic field emitted by cellular phones on the EEG during a memory task. *NeuroReport*, **11**, 761.

Kristensen T S (1989). Cardiovascular disease and the work environment. A critical review of the epidemiologic literature on nonchemical factors. *Scan J Work Environ Health*, **15**, 165.

Kubota S, Kiyosawa H, Nomura Y, Yamada T and Seyama Y (1997). Ornithine decarboxylase overexpression in mouse $10T_{1/2}$ fibroblasts. Cellular transformation and invasion. *J Natl Cancer Inst*, **89**, 567.

Kues H A and D'Anna S A (1987). Changes in the monkey eye following pulsed 2.45 GHz microwave exposure. IN *Proceedings of the Ninth Annual Conference of the IEEE Engineering in Medicine and Biology Society*, November 1987, Boston, Maryland, p 689.

Kues H A and Monahan J C (1992a). Microwave-induced changes in the primate eye. *Johns Hopkins APL Tech Dig*, **13**, 244.

Kues H A and Monahan J C (1992b). Pulsed microwave-induced ocular changes in the restrained non-human primate. IN Abstracts, *The First World Congress for Electricity and Magnetism in Biology and Medicine*, June 1992, Orlando, Florida, p 62.

Kues H A, Hirst L W, Lutty G A, D'Anna S A and Dunkelberger G R (1985). Effects of 2.45-GHz microwaves on primate corneal endothelium. *Bioelectromagnetics*, **6**, 177.

Kues H A, McLeod D S, D'Anna S A, Lutty G A and Monahan J C (1988). Histological evaluation of microwave-induced vascular leakage in the iris. IN *Abstracts, 10th Annual Meeting of the Bioelectromagnetics Society*, June 1988, Stamford, Connecticut, p 49.

Kues H A, McLeod D S, D'Anna S A, Johnson M A, Pery C R and Monahan J C (1991). Microwave-induced electroretinographic changes in the primate. IN *Abstracts, 13th Annual Meeting of the Bioelectromagnetics Society*, June 1991, Salt Lake City, Utah, p 53.

Kues H A, Monahan J C, D'Anna S A, McLeod D S, Lutty G A and Koslov S (1992). Increased sensitivity of the non-human primate eye to microwave radiation following ophthalmic drug pretreatment. *Bioelectromagnetics*, **13**, 379.

Kues H A, D'Anna S A, Osiander R, Green, W R and Monahan J C (1999). Absence of ocular effects after either single or repeated exposure to 10 mW/cm^2 from a 60 GHz CW source. *Bioelectromagnetics*, **20**, 463.

Kwee S and Raskmark P (1998). Changes in cell proliferation due to environmental non-ionizing radiation 2. Microwave radiation. *Bioelectrochem Bioenerg*, **44**, 251.

References

Lagorio S, Rossi P, Vecchia P, DeSantis M, Bastianini L, Fusilli M, Ferrucci A, Desideri E and Comba P (1997). Mortality of plastic-ware workers exposed to radiofrequencies. *Bioelectromagnetics*, **18,** 418.

Lai H (1992). Research on the neurological effects of non-ionizing radiation at the University of Washington. *Bioelectromagnetics*, **13**, 513.

Lai H and Singh N P (1995). Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells. *Bioelectromagnetics*, **16**, 207.

Lai H and Singh N P (1996). Single- and double-strand DNA breaks in rat brain cells after acute exposure to radiofrequency electromagnetic radiation. *Int J Radiat Biol,* **69**, 513.

Lai H and Singh N P (1997). Melatonin and a spin-trap compound block radiofrequency electromagnetic radiation-induced DNA strand breaks in rat brain cells. *Bioelectromagnetics*, **18**, 446.

Lai H, Horita A, Chou C-K and Guy A W (1987). Low-level microwave irradiation affects central cholinergic activity in the rat. *J Neurochem*, **48**, 40.

Lai H, Carino M A, Horita A and Guy A W (1989a). Low-level microwave irradiation and central nervous cholinergic systems. *Pharmacol Biochem Behav,* **33**, 131.

Lai H, Carino M A, Horita A and Guy A W (1989b). Low-level microwave irradiation and central cholinergic activity: a dose response study. *Bioelectromagnetics*, **10,** 203.

Lai H, Carino M, Horita A and Guy A W (1990). Corticotropin-releasing factor antagonist blocks microwave induced decreases in high-affinity choline uptake in the rat brain. *Brain Res Bull*, **25**, 609.

Lai H, Carino M A, Wen Y F, Horita A and Guy A W (1991). Naltrexonew pretreatment blocks microwave-induced changes in central cholinergic receptors. *Bioelectromagnetics*, **12**, 27.

Lai H, Horita A and Guy A W (1994). Microwave irradiation affects radial-arm maze performance in the rat. *Bioelectromagnetics*, **15**, 95.

Lamble D, Kauranen T, Laakso M and Summala H (1999). Cognitive load and detection in thresholds in car following situations: safety implications for using mobile (cellular) telephones while driving. *Accid Anal Prev*, **31**, 617.

Lary J M and Conover D L (1987). Teratogenic effects of radiofrequency radiation. *IEEE Eng Med Biol Mag*, March 1987, 42.

Lary J M, Conover D L, Foley E D and Hanser P L (1982). Teratogenic effects of 27.12 MHz radiofrequency radiation in rats. *Teratology*, **26**, 299.

Lary J M, Conover D L, Foley E D and Hanser P L (1983). Teratogenicity of 27.12 MHz radiation in rats is related to duration of hyperthermia exposure. *Bioelectromagnetics*, **4**, 249.

Liburdy R P and Vanek P F Jr (1987). Microwaves and the cell membrane. III. Protein shedding is oxygen and temperature dependent: evidence for cation bridge involvement. *Radiat Res*, **109**, 382.

Liddle C G, Putnam J P and Huey O P (1994). Alteration in life span of mice chronically exposed to 2450 MHz CW microwaves. *Bioelectromagnetics*, **15**, 177.

Lin-Liu S and Adey W R (1982). Low frequency amplitude modulated microwave fields change calcium efflux rates from synaptosomes. *Bioelectromagnetics*, **3**, 309.

Linz K W, von Westphalen C, Streckert J, Hansen V and Meyer R (1999). Membrane potential and currents of isolated heart muscle cells exposed to pulsed radio frequency fields. *Bioelectromagnetics*, **20**, 497.

Litovitz T A, Krause D, Penafiel M, Elson E C and Mullins J M (1993). The role of coherence time in the effect of microwaves on ornithine decarboxylase activity. *Bioelectromagnetics*, **14**, 395.

Litovitz T A, Penafiel M, Krause D, Zhang D and Mullins J M (1997a). The role of temporal sensing in bioelectromagnetic effects. *Bioelectromagnetics*, **18**, 388.

Litovitz T A, Penafiel L M, Farrel J M, Krause D, Meister R and Mullins J M (1997b). Bioeffects induced by exposure to microwaves are mitigated by superposition of ELF noise. *Bioelectromagnetics*, **18,** 422.

Liu D-S, Astumian R D and Tsong T Y (1990). Activation of Na^+ and K^+ pumping modes of (Na, K)-ATPase by an oscillating electric field. *J Biol Chem*, **265,** 7260.

Lloyd D C, Saunders R D, Finnon P and Kowalczuk C I (1984). No clastogenic effect from *in vitr*o microwave irradiation of G_0 human lymphocytes. *Int J Radiat Biol*, **46**, 135.

Lloyd D C, Saunders R D, Moquet J E and Kowalczuk C I (1986). Absence of chromosomal damage in human lymphocytes exposed to microwave radiation with hyperthermia. *Bioelectromagnetics*, **7**, 235.

Lu S-H, Mathur S P, Stuck B, Zwick H, D'Andrea J M, Merritt J H, Lutty G, Mcleod D S and Johnson M (2000). Effects of high peak power microwaves on the retina of the rhesus monkey. *Bioelectromagnetics*, **21**, 1.

McKelvey-Martin V J, Green M H L, Schmezer P, Pool-Zobel B L, De Méo M P and Collins A (1993). The single cell gel electrophoresis assay (comet assay): a European review. *Mutat Res*, **288**, 47.

McKenzie D R, Yin Y and Morrell S (1998). Childhood incidence of acute lymphoblastic leukemia and exposure to broadcast radiation in Sydney – a second look. *Aust NZ J Public Health*, **22,** 360.

McKnight A J and McKnight A S (1993). The effect of cellular phones use upon driver attention. *Accid Anal Prev*, **25**, 259.

McRee D I (1980). Soviet and Eastern European research on biological effects of microwave radiation. *Proc IEEE*, **68**, 84.

McRee D I and Wachtel H (1980). The effects of microwave radiation on the vitality of isolated frog sciatic nerves. *Radiat Res*, **82**, 536.

McRee D I, Elder J A, Gage M I, Reiter L W, Rosenstein L S, Shore M L, Galloway W D, Adey W R and Guy A W (1979). Effects of nonionizing radiation on the central nervous system, behavior and blood: a progress report. *Environ Health Perspect*, **30,** 123.

McRee D I, MacNichols G and Livingston G K (1981). Incidence of sister chromatid exchange in bone marrow cells of the mouse following microwave exposure. *Radiat Res*, **85**, 340.

McRee D I, Galvin M J and Mitchell C L (1988). Microwave effects on the cardiovascular system: a model for studying the responsivity of the autonomic nervous system to microwaves. IN *Electromagnetic Fields and Neurobehavioral Function* (M E O'Connor and R H Lovely, Eds). New York, Alan R Liss, p 153.

Maes A, Verschaeve L, Arroyo A, De Wagter C and Vercruyssen L (1993). *In vitro* cytogenetic effects of 2450 MHz waves on human peripheral blood lymphocytes. *Bioelectromagnetics*, **14**, 495.

Maes A, Collier M, Slaets D and Verschaeve L (1995). Cytogenetic effects of microwaves from mobile communication frequencies (954 MHz). *Electro-Magnetobiology*, **14**, 91.

Maes A, Collier M, Slaets D and Verschaeve L (1996). 954 MHz microwaves enhance the mutagenic properties of mitomycin C. *Environ Mol Mutagen*, **28,** 26.

Maes A, Collier M, Van Gorp U, Vandoninck S and Verschaeve L (1997). Cytogenetic effects of 935.2-MHz (GSM) microwaves alone and in combination with mitomycin C. *Mutat Res*, **393**, 151.

Maes A, Collier M and Verschaeve L (*in press*). Cytogenetic effects of 900 MHz (GSM) microwaves in human lymphocytes. *Bioelectromagnetics*.

Magras I N and Xenos T D (1997). RF radiation-induced changes in the prenatal development of mice. *Bioelectromagnetics*, **18**, 455.

Malyapa R S, Ahern E W, Straube W L, Moros E G, Pickard W F and Roti Roti J L (1997a). Measurement of DNA damage following exposure to 2450 MHz electromagnetic radiation. *Radiat Res*, **148**, 608.

Malyapa R S, Ahern E W, Straube W L, Moros E G, Pickard W F and Roti Roti J L (1997b). Measurement of DNA damage following exposure to electromagnetic radiation in the cellular communications frequency band (835.62 and 847.74 MHz). *Radiat Res*, **148**, 618.

Malyapa R S, Ahern E W, Bi C, Straube W L, LaRegina M, Pickard W F and Roti Roti J L (1998). DNA damage in rat brain cells after *in vivo* exposure to 2450 MHz electromagnetic radiation and various methods of euthanasia. *Radiat Res*, **149**, 637.

Manikowska-Czerska E, Czerski P and Leach W M (1985). Effects of 2.45 GHz microwaves on meiotic chromosomes of male CBA/CAY mice. *J Hered,* **76**, 71.

Mann K and Roschke J (1996). Effects of pulsed high-frequency electromagnetic fields on human sleep. *Neuropsychobiology,* **33**, 41.

Mann S M, Cooper T G, Allen S G, Blackwell R P and Lowe A J (*in press*). Exposure to radio waves near mobile telephone base stations. Chilton, NRPB.

Marcickiewitcz J, Chazan B, Niemiek T, Sokolska G, Troszynski M, Luczak M and Szmiegielski S (1986). Microwave radiation enhances teratogenic effect of cytosine arabinoside in mice. *Biol Neonate*, **50**, 75.

References

Marton L J and Pegg A E (1995). Polyamines as targets for therapeutic intervention. *Ann Rev Pharm Toxicol*, **35**, 55.

Maskarinec G, Cooper J and Swygert L (1994). Investigation of increased incidence in childhood leukemia near radio towers in Hawaii: preliminary observations. *J Environ Pathol Toxicol Oncol*, **13,** 33.

MDA (Medical Devices Agency) (1997). Electromagnetic Compatibility of Medical Devices with Mobile Communications. London, MDA DB 9702.

Meltz M L, Walker K A and Erwin D N (1987). Radiofrequency (microwave) radiation exposure of mammalian cells during UV-induced DNA repair synthesis. *Radiat Res*, **110**, 255.

Meltz M L, Eagan P and Erwin D N (1989). Absence of mutagenic interaction between microwaves and mitomycin C in mammalian cells. *Environ Mol Mutagen*, **13**, 294.

Meltz M L, Eagan P and Erwin D N (1990). Proflavin and microwave radiation: absence of a mutagenic interaction. *Bioelectromagnetics*, **11**, 149.

Merritt J H, Chamness A F, Hartzell R H and Allen S J (1977). Orientation effects on microwave-induced hyperthermia and neurochemical correlates. *J Microwave Power*, **12,** 167.

Merritt J H, Shelton W W and Chamness A F (1982). Attempts to alter Ca-45^{2+} binding to brain tissue with pulse-modulated microwave energy. *Bioelectromagnetics*, **3**, 457.

Mickley G A, Cobb B L, Mason P A and Farrell S (1994). Disruption of a putative working memory task and selective expression of brain *c-fos* following microwave-induced hyperthermia*. Physiol Behav,* **55**, 1029.

Milham S (1985). Mortality in workers exposed to electromagnetic fields. *Environ Health Perspect*, **62,** 297.

Milham S (1988). Increased mortality in amateur radio operators due to lymphatic and haematopoietic malignancies. *Am J Epidemiol*, **127,** 50.

Millar D B, Christopher J P, Hunter J and Yeandle S S (1984). The effect of exposure of acetylcholinesterase to 2450 MHz microwave radiation. *Bioelectromagnetics*, **5**, 165.

Mitchell C L, McRee D I, Peterson J and Tilson H A (1988). Some behavioral effects of short-term exposure of rats to 2.45 GHz microwave radiation. *Bioelectromagnetics*, **9**, 259.

Mitchell C L, McRee D I, Peterson N J, Tilson H A, Shandala M G, Rudnev M I, Varetskii V V and Navakatikyan M I (1989). Results of a United States and Soviet Union joint project on nervous system effects of microwave radiation. *Environ Health Perspect*, **81**, 201.

Mitchell D S, Switzer W G and Bronaugh E L (1977). Hyperactivity and disruption of operant behavior in rats after multiple exposures to microwave exposure. *Radio Sci*, **12**, 263.

Miura K, Morimoto K and Koizumi A (1986). Effects of temperature on chemically induced sister-chromatid exchange in human lymphocytes. *Mutat Res*, **174**, 15.

Modak A T, Stavinoha W B and Dean U P (1981). Effect of short electromagnetic pulses on brain acetylcholine content and spontaneous motor activity in mice. *Bioelectromagnetics,* **2**, 89.

Moe K E, Lovely R H, Myers D E, Guy A W (1976). Physiological and behavioral effects of chronic low level microwave radiation in rats. IN *Biological Effects of Electromagnetic Waves.* Selected papers of the UNSC/URSI Annual Meeting, Boulder, Colorado, October 1975 (C C Johnson and M L Shore, Eds). Rockville, Maryland, US Department of Health, Education and Welfare, Volume 1, p 248.

Montaigne K and Pickard W F (1984). Offset of the vacuolar potential of Characean cells in response to electromagnetic radiation over the range 250 Hz – 250 kHz *Bioelectromagnetics*, **5**, 31.

Morgan R W, Kelsh M A, Zhao K, Exuzides A, Heringer S and Negrete W. Radiofrequency exposure and mortality from cancer of the brain and lymphatic/hematopoietic systems. *Epidemiology*, **11,** 118.

Morrissey J J, Raney S, Heasley E, Rathinavelu P, Dauphnee M and Fallon J H (1999). Iridium exposure increases c-*fos* expression in the mouse brain only at levels which likely result in tissue heating. *Neuroscience*, **92**, 1539.

Moshier J A, Dosescu J, Skunca M and Luck G D (1993). Transformation of NIH/3T3 cells by ornithine decarboxylase overexpression. *Cancer Res*, **53**, 2618.

Moshier J A, Malecka-Panas E, Geng H, Dosescu J, Tureaud J, Skunca M and Majumda A P (1994). Ornithine decarboxylase transformation of NIH/3T3 cells is mediated by altered epidermal growth factor receptor activity. *Cancer Res*, **55**, 5358.

Moulder J E, Erdreich L S, Malyapa R S, Merritt J, Pickard W F and Vijayalaxmi D Z (1999). Cell phones and cancer: what is the evidence for a connection? *Radiat Res*, **151**, 513.

Muhm J M (1992). Mortality investigation of workers in an electromagnetic pulse test program. *J Occup Med*, **34,** 287.

Nagawa H, Tsurita G and Ueno S (1999). Effects of 1.439 MHz microwave exposure on the brain in Sprague-Dawley rats. IN *Health Effects of Mobile Telephones*, Proceedings URSI General Assembly, Toronto, August 1999.

Nelson B K, Conover D L, Brightwell W S, Shaw P B, Werren D, Edwards R M and Lary J M (1991). Marked increase in the teratogenicity of the combined administration of the industrial solvent 2-methoxyethanol and radiofrequency radiation in rats. *Teratology*, **43**, 621.

Neubauer C, Phelan A M, Kues H and Lange D G (1990). Microwave irradiation of rats at 2.45 GHz activates pinocytotic-like uptake of tacers by capillary endothelial cells of cerebral cortex. *Bioelectromagnetics*, **11**, 261.

NIEHS (National Institute of Environmental Health Sciences) Working Group Report (1998). *Assessment of the Health Effects from Exposure to Power-line Frequency Electric and Magnetic Fields* (C J Porter and M S Wolfe, Eds). Research Triangle Park, NC, US National Institutes of Health, NIH Publication No. 98-3981, p 311.

Novoselova E G, Fesenko E E, Makar V R and Sadovnikov V B (1999). Microwaves and cellular immunity. II. Immunostimulating effects of microwaves and naturally occurring antioxidant nutrients. *Bioelectrochem Bioenerg,* **49(1)**, 37.

NRC (National Research Council) (1997). *Possible Health Effects of Exposure to Residential Electric and Magnetic Fields*. Washington DC, National Academy Press.

NRPB (1992). Electromagnetic fields and the risk of cancer. Report of an Advisory Group on Non-ionising Radiation. *Doc NRPB*, **3(1)**, 1.

NRPB (1993a). Statement by NRPB: restrictions on human exposure to static and time varying electromagnetic fields and radiation. *Doc NRPB*, **4(5)**, 1.

NRPB (1993b). Restrictions on human exposure to static and time varying electromagnetic fields and radiation: scientific basis and recommendations for the implementation of the Board's Statement. *Doc NRPB*, **4(5)**, 7.

NRPB (1993c). Electromagnetic fields and the risk of cancer. Summary of the views of the Advisory Group on Non-ionising Radiation on epidemiological studies published since its 1992 report. *Doc NRPB*, **4(5)**, 65.

NRPB (1994). Electromagnetic fields and the risk of cancer. Supplementary report by the Advisory Group on Non-ionising Radiation (12 April 1994). *Doc NRPB*, **5(2)**, 77.

NRPB (1999a). Statement by NRPB: advice on the 1998 ICNIRP guidelines for limiting exposure to time-varying electric, magnetic and electromagnetic fields (up to 300 GHz). *Doc NRPB,* **10(2)**, 1.

NRPB (1999b). 1998 ICNIRP guidelines for limiting exposure to time-varying electric, magnetic and electromagnetic fields (up to 300 GHz): advice on aspects of implementation in the UK. *Doc NRPB,* **10(2)**, 5.

O'Connor M E (1980). Mammalain teratogenesis and radio-frequency fields. *Proc IEEE*, **68**, 56.

O'Connor M E (1988). Prenatal microwave exposure and behaviour. IN *Electromagnetic Fields and Neurobehavioral Function* (M E O'Connor and R H Lovely, Eds). New York, Alan Liss, p 265.

O'Connor M E (1999). Intrauterine effects in animals exposed to radiofrequency and microwave fields. *Teratology*, **59**, 287.

Oscar K J and Hawkins T D (1977). Microwave alteration of the blood–brain barrier system of rats. *Brain Res*, **126**, 281.

Ouellet-Hellstrom R and Stewart W F (1993). Miscarriages among female physical therapists who report using radio- and microwave-frequency electromagnetic radiation. *Am J Epidemiol*, **138,** 775.

Owen R D (2000). Possible health risks of radiofrequency exposure from mobile telephones. *Epidemiology*, **11,** 99.

Pakhomova O N, Pakhomov A G and Akyel Y (1997). Effect of millimeter waves on UV-induced recombination and mutagenesis in yeast. *Bioelectrochem Bioenerg*, **43**, 227.

Pearce N, Reif J and Fraser J (1989). Case–control studies of cancer in New Zealand electrical workers. *Int J Epidemiol*, **18**, 51.

References

Pederson G F and Anderson J B (1999). RF and ELF exposure from cellular phone handsets: TDMA and CDMA systems. *Radiat Prot Dosim*, **83**, 131.

Penafiel L M, Litovitz T, Krause D, Desta A and Mullins J M (1997). Role of modulation on the effect of microwaves on ornithine decarboxylase activity in L929 cells. *Biolectromagnetics*, **18**, 132.

Pennes H H (1948). Analysis of tissue and arterial blood temperature in the resting human forearm. *J Appl Physiol*, **1,** 92.

Penrose R (1994). *Shadows of the Mind*. Oxford, Oxford University Press.

Phelan A M, Lange D G, Kues H A and Lutty G A (1992). Modification of membrane fluidity in melanin-containing cells by low-level microwave radiation. *Bioelectromagnetics*, **13**,131.

Philippova T M, Novoselov V I and Alekseev S I (1994). Influence of microwaves on different types of receptors and the role of peroxidation of lipids on receptor-protein shedding. *Bioelectromagnetics,* **15**,183.

Phillips J L, Ivaschuk O, Ishida-Jones T, Jones R A, Campbell-Beachler M and Haggren W (1998). DNA damage in Molt-4 T-lymphoblastoid cells exposed to cellular telephone radiofrequency fields *in vitro*. *Bioelectrochem Bioenerget*, **45**, 103.

Phillips L P, Blackwell D B, Clancy C J, Donner M D, Tice R T, Hook G H and McRee D M (1999). Genotoxicity of radio frequency radiation fields generated from analog, TDMA, CDMA and PCS technology evaluated using a three test *in vitro* battery. *Environ Mol Mutagen*, **33 (Suppl 30)**, 49.

Pokorny J and Wu T-M (1998). *Biophysical Aspects of Coherence and Biological Order*. Prague, Springer, p 240.

Power R S, David H E, Mutwakil M H A Z, Fletcher K, Daniells C, Nowell M A, Dennis J L, Martinelli A, Wiseman R, Wharf E and de Pomerai D I (1998). Stress-inducible transgenic nematodes as biomonitors of soil and water pollution. *J Biosci*, **23**, 513.

Prausnitz S and Susskind C (1962). Effects of chronic microwave irradiation on mice*. IRE Trans Biomed Electron*, **9**, 104.

Preece A W, Iwi G, Davies-Smith A, Wesnes K, Butler S, Lim E and Varey A (1999). Effect of a 915-MHz simulated mobile phone signal on cognitive function in man. *Int J Radiat Biol*, **75**, 447.

Preskorn S H, Edwards W D and Justesen D R (1978). Retarded tumor growth and greater longevity in mice after fetal irradiation by 2450 MHz microwaves. *J Surg Oncol*, **10,** 483.

Prohovsky E W and Eyster J M (1974). Prediction of giant breathing and rocking modes in double helical RNA. *Phys Lett,* **A50**, 329.

Pu J S, Chen J, Yang Y H and Bai Y Q (1997). The effects of 3000-MHz microwave irradiation on electroencephalic energy and energy meatbolism in mouse brain. *Electro-Magnetobiology,* **16**, 243.

Redelmeir D A and Tibshirani, R J (1997). Association between cellular-telephone calls and motor vehicle collisions. *J Med*, **336**, 453.

Reid S W J and Gettinby G (1998). Radio-frequency electromagnetic field from mobile phones. *Lancet*, **352,** 576.

Reiser H, Dimpfel W and Schober F (1995). The influence of electromagnetic fields on human brain activity. *Eur J Med Res*, **1**, 27.

Reiter R J (1991). Pineal melatonin: cell biology of its synthesis and of its physiological interactions. *Endocr Rev*, **12(2)**, 151.

Reiter R J (1993). The melatonin rhythm – both a clock and a calendar. *Experientia*, **49**, 654.

Repacholi M H (1998). Low level exposure to radiofrequency electromagnetic fields: health effects and research needs. *Bioelectromagnetics*, **19**, 1.

Repacholi M H and Cardis E (1997). Criteria for EMF health risk assessment. *Radiat Prot Dosim*, **72**, 305.

Repacholi M H, Basten A, Gebski V, Noonan D, Finnie J and Harris A W (1997). Lymphomas in Eμ-*Pim1* transgenic mice exposed to pulsed 900 MHz electromagnetic fields. *Radiat Res,* **147**, 631.

Resnekov L (1981). Noise, radio frequency radiation and the cardiovascular system. *Circulation*, **63**, 264A.

Roberti B, Heebels G H, Hendrix J C M, De Greef A H A M and Wolhuis O L (1975). Preliminary investigations of the effects of low-level microwave radiation on spontaneous motor activity in rats. *Ann NY Acad Sci*, **247**, 417.

Roberts N J Jr (1983). Radiofrequency and microwave effects on immunological and hematopoietic systems. IN *Biological Effects and Dosimetry of Nonionizing Radiation, Radiofrequency and Microwave Energies* (M Grandolfo, S M Michaelson and A Rindi, Eds). New York, Plenum Press, p 429.

Roberts N J Jr, Michaelson S M and Lu S-T (1986). The biological effects of radiofrequency radiation: a critical review and recommendations. *Int J Radiat Biol,* **50**, 379.

Robinette C D, Silverman C and Jablon S (1980). Effects upon health of occupational exposure to microwave radiation (radar). *Am J Epidemiol*, **112**, 39.

Roschke J and Mann K (1997). No short-term effects of digital mobile radio telephone on the awake human electroencephalogram. *Bioelectromagnetics*, **18**, 172.

Rothman K J, Loughlin J E, Funch D P and Dreyer N A (1996). Overall mortality of cellular telephone customers. *Epidemiology*,**7,** 303.

Rotkovska D, Moc J, Kautska J, Bartonicknov A, Keprtova J and Hofer M (1993). Evaluation of the biological effects of police radar RAMER 7E. *Environ Health Perspect*, **101**, 134.

Royal Society of Canada Expert Panel Report (1999). A review of the potential health risks of radiofrequency fields from wireless telecommunication devices. An Expert Panel Report prepared at the request of the Royal Society of Canada for Health Canada. Ottawa, Royal Society of Canada, RSC.EPR 99-1.

Sadčikova M N (1974). Clinical manifestations of reactions to microwave irradiation in various occupational groups. IN *Biological Effects and Health Hazards of Microwave Radiation* (P Czerski, K Ostrowski, M L Shore, C Silverman, M J Suess and B Waldeskog, Eds). Warsaw, Polish Medical Publishers, p 261.

Salford L G, Brun A, Persson B R R and Eberhardt J (1993). Experimental studies of brain tumour development during exposure to continuous and pulsed 915 MHz radiofrequency radiation. *Bioelectrochem Bioenerget*, **30**, 313.

Salford L G, Brun A, Sturesson K, Eberhardt J and Persson B R R (1994). Permeability of the blood brain barrier induced by 915 MHz electromagnetic radiation, continuous wave and modulated at 8, 16, 50, 200 Hz. *Microscopy Res Tech*, **27**, 535.

Salzinger K (1994). Behavioral effects of electromagnetic fields in animals. IN *Biological Effects of Electric and Magnetic Fields*, Volume 1, Sources and Mechanisms (D O Carpenter and S Ayrapetyan, Eds). New York, Academic Press, p 315.

Santini R, Honsi M, Deschaux P and Pacheco H (1988). B16 melanoma development in black mice exposed to low-level microwave radiation. *Bioelectromagnetics*, **9**, 105.

Santo, L (1983). *Escherichia coli* induction by millimetre waves (meeting abstract). Techniques in studies of biological effects of low level millimetre waves.

Sanza J N and de Lorge J (1977). Fixed interval behavior of rats exposed to mcirowaves at low power densities. *Radio Sci*, **12**, 273.

Sarkar S, Ali S and Behari J (1994). Effect of low power microwave on the mouse genome: a direct DNA analysis. *Mutat Res,* **320**, 141.

Saunders R D and Kowalczuk C I (1981). Effect of 2450 MHz microwave radiation and heat on mouse spermatogenic epithelium. *Int J Radiat Biol,* **40**, 623.

Saunders R D, Darby S C and Kowalcazuk C I (1983). Dominant lethal studies in male mice after exposure to 2450 MHz microwave radiation. *Mutat Res,* **117**, 345.

Saunders R D, Kowalczuk C I, Beechey C V and Dunford R (1988). Studies of the induction of dominant lethals and translocations in male mice after chronic exposure to microwave radiation. *Int J Radiat Biol,* **53**, 983.

Scarfi M R, Lioi M B, d'Ambrosio G, Massa R, Zeni O, De Pietro R and De Berardino D (1996). Genotoxic effects of mitomycin-C and microwave radiation on bovine lymphocytes. *Electro-Magnetobiology*, **15**, 99.

Scholl D M and Allen S J (1979). Skilled visual-motor performance by monkeys in a 1.2-GHz microwave field. *Radio Sci*, **14**, 247.

Schrot J, Thomas J R and Banvard R A (1980). Modification of the repeated acquisition of response sequences in rats by low-level microwave exposure. *Bioelectromagnetics*, **1**, 89.

Schwan H P (1985). EM-field induced force effects. IN *Interactions between Electromagnetic Fields and Cells (*A Chiabrera, C Nicolini and H P Schwan, Eds). New York, Plenum Press.

References

Science and Technology Committee (1999). Third Report. Scientific advisory system: mobile phones and health. Volume 1, Report and Proceedings of the Committee.

Scott A C (1985). Soliton oscillations in DNA. *Phys Rev A*, **31**, 3518.

Scottish Parliament Transport and the Environment Committee (2000). Third Report. Report on inquiry into the proposals to introduce new planning procedures for telecommunications developments.

Seaman R L and Wachtel H (1978). Slow and rapid responses to CW and pulsed microwave radiation by individual Aplysia pacemakers. *J Microwave Power*, **13,** 77.

Seaman R L and Lebovitz R M (1989). Thresholds of cat cochlear nucleus neurons to microwave pulses. *Bioelectromagnetics*, **10,** 147.

Selvin S, Schulman J and Merrill D W (1992). Distance and risk measures for the analysis of spatial data: a study of childhood cancers. *Soc Sci Med*, **34,** 769.

Shandala M G, Dumanskii U D, Rudnev M I, Ershova L K and Los I P (1979). Study of nonionizing microwave radiation effects upon the central nervous system and behavior reactions. *Environ Health Perspect*, **30**,115.

Sheppard A R, Bawin S M and Adey W R (1979). Models of long-range order in cerebral macromolecules: effects of sub-ELF and of modulated VHF and UHF fields. *Radio Sci*, **14(S),** 141.

Shelton W W Jr and Merritt J H (1981). *In vitro* study of microwave effects on calcium efflux in rat brain tissue. *Bioelectromagnetics*, **2**, 161.

Sienkiewicz Z J, Cridland N A Kowalczuk C I and Saunders R D (1993). Biological effects of electromagnetic fields and radiation. IN *The Review of Radio Science 1990–1992* (W R Stone, Ed). New York, Oxford University Press, p 737.

Sienkiewicz Z J, Blackwell R P, Haylock R G E, Saunders R D and Cobb B L (2000). Low-level exposure to pulsed 900 MHz microwave radiation does not cause deficits in the performance of a spatial memory task in mice. *Bioelectromagnetics*, **21**, 151.

Skidmore W D and Baum S J (1974). Biological effects in rodents exposed to 10^8 pulses of electromagnetic radiation. *Health Phys*, **26**, 391.

Smialowicz R J, Rogers R R, Garner R J, Riddle M M, Luebke R W and Towe D G (1983). Microwaves (2,450 MHz) suppress murine natural killer cell activity. *Bioelectromagnetics*, **4**, 371.

Snyder S H (1971). The effect of microwave irradiation on the turnover rate of serotonin and norepinephrine and the effect of microwave metabolozing enzymes. Washington DC, US Army Medical Research and Development Command Final Report, Contract No DADA 17-69-C-9144.

Spalding J F, Freyman R W and Holland L M (1971). Effects of 800 MHz electromagnetic radiation on body weight, activity, haematopoiesis and life span in mice. *Health Phys*, **20**, 421.

Stagg R B, Thomas W J, Jones R A and Adey W R (1997). DNA synthesis and cell proliferation in C6 glioma and primary glial cvells exposed to a 836.55 MHz modulated radiofrequency field. *Bioelectromagnetics*, **18**, 230.

Stark K D C, Krebs T, Altpeter E, Manz B, Griot C and Abelin T (1997). Absence of chronic effect of exposure to short-wave radio broadcast signal on salivary melatonin concentrations in dairy cattle. *J Pineal Res*, **22**, 171.

Steele R and Hanzo L (1999). *Mobile Communications* (2nd edition). New York, Wiley.

Stevens R G (1987). Electric power use and breast cancer: a hypothesis. *Am J Epidemiol*, **125**, 556.

Strayer D L, Johnston W A and Grison S (1999). Driven to distraction: studies of driving and cellular phone use. *Abs Psychonomic Soc*, **4**, 16 (conference abstract).

Swerdlow A J (1997). Epidemiology of chronic diseases in relation to radiofrequency radiation exposure: issues in interpretation of the current literature and future directions for research. IN *Proceedings of Seminar of Non-thermal Effects of RF Electromagnetic Fields*, Munich, Germany, November 1996, ICNIRP 3/97, p191.

Szmigielski S, Szudzinski A, Pietraszek A, Bielec M and Wrembel J K (1982). Accelerated development of spontaneous and benzopyrene-induced skin cancer in mice exposed to 2450-MHz microwave radiation. *Bioelectromagnetics*, **3**, 179.

Szmigielski S, Bielec M, Lipski S and Sokolska G (1988). Immunologic and cancer-related aspects of exposure to low-level microwave and radiofrequency fields. IN *Modern Bioelectricity* (A A Marino, Ed). New York, Marcel Dekker, p 861.

Szudzinski A, Pietraszek A, Janiak M, Wrembel J, Kalczek M and Szmigielski S (1982). Acceleration of the development of benzopyrene-induced skin cancer in mice by microwave radiation. *Arch Dermatol Res,* **274**, 303.

Szmigielski S (1996). Cancer mortality in subjects occupationally exposed to high-frequency (radiofrequency and microwaves) electromagnetic radiation. *Sci Total Environ*, **180,** 9.

Takashima S, Onaral B and Schwan H P (1979). Effects of modulated RF energy on the EEG of mammalian brains. *Radiat Environ Biophys*, **16**, 15.

Taskinen H, Kyyrönen P and Hemminki K. Effects of ultrasound, shortwaves and physical exertion on pregnancy outcome in physiotherapists. *J Epidemiol Commun Health*, **44,** 196.

Taylor E M and Ashleman B T (1974). Analysis of central nervous system involvement in the microwave auditory effect. *Brain Res*, **74,** 201.

Tenforde T S and Liburdy R P (1988). Magnetic deformation of phospholipid bilayers: effects on liposome shape and solute permeability at prophase transition temperatures. *J Theoret Biol*, **133,** 385.

Thomas T L, Stolley P D, Stemhagen A, Fontham E T H, Bleecker M L, Stewart P A and Hoover R N (1987). Brain tumor mortality risk among men with electrical and electronics jobs: a case–control study. *J Natl Cancer Inst*, **79,** 233.

Thuroczy G, Kubinyi G, Bodo M, Bakos J and Szabo L D (1994). Simultaneous response of brain electrical activity (EEG) and cerebral circulation (REG) to microwave exposure in rats. *Rev Environ Health*, **10**, 135.

Tice R T *et al* (1999). Tests of mobile phone signals for activity in genotoxicity and other laboratory assays. Presented at Annual Meeting of the Environmental Mutagen Society, 29 March 1999, Washington DC.

Toler J C, Shelton W W, Frei M R, Merritt J H and Stedham M A (1997). Long-term, low-level exposure of mice prone to mammary tumours to 435 MHz radiofrequency radiation. *Radiat Res*, **148**, 227.

Tynes T, Andersen A and Langmark F (1992). Incidence of cancer in Norwegian workers potentially exposed to electromagnetic fields. *Am J Epidemiol*, **136,** 81.

Tynes T, Hannevik M, Anderson A, Vistnes A I and Haldorsen T (1996). Incidence of breast cancer in Norwegian female radio and telegraph operators. *Cancer Causes Control*, **7,** 197.

UNEP/WHO/IRPA (1993). Electromagnetic Fields (300 Hz – 300 GHz). Geneva, World Health Organization, Environmental Health Criteria 137.

Urban P, Lukas E and Roth Z (1998). Does acute exposure to the electromagnetic field emitted by a mobile phone influence visual evoked potentials? A pilot study. *Centr Eur J Public Health*, **6**, 288.

Van Leeuwen G M J, Lagendijk J J W, Van Leersum B J A M, Zwamborn A P M, Hornsleth S N and Kotte A N T J (1999). Calculation of brain temperatures due to exposure to a mobile phone. *Phys Med Biol*, **44**, 2367.

Van Zandt L L (1986). Resonant microwave absorption by dissolved DNA. *Phys Rev Letts*, **57(16)**, 2085.

Varma M M and Traboulay E A Jr (1976). Evaluation of dominant lethal test and DNA studies in measuring mutagenicity caused by non-ionizing radiation. IN *Biological Effects of Electromagnetic Waves*. Selected papers of the UNSC/URSI Annual Meeting, Boulder, Colorado, October 1975 (C C Johnson and M L Shore, Eds). Rockville, Maryland, US Department of Health, Education and Welfare, Volume 1, p 386.

Varma L M and Traboulay E A (1977). Comparison of native and microwave irradiated DNA. *Experiention*, **33/12**, 1649.

Varma M M, Dage E L and Joshi S R (1976). Mutagenicity induced by non-ionizing radiation in swiss male mice IN *Biological Effects of Electromagnetic Waves*. Selected papers of the UNSC/URSI Annual Meeting, Boulder, Colorado, October 1975 (C C Johnson and M L Shore, Eds). Rockville, Maryland, US Department of Health, Education and Welfare, Volume 1, p 386.

Vasquez M V, Clancy C J, Blackwell D B, Donner M D, Tice R T, Hook G H and McRee D M (1999). Genotoxicity of radio frequency radiation fields generated from analog, TDMA, CDMA and PCNA in human blood cells evaluated using single gel (SCG) electrophoresis and the cytochalasin B micronucleus assay. *Environ Mol Mutagen*, **33(Suppl 30)**, 66.

Verschaeve L (1995). Can non-ionizing radiation induce cancer? *Cancer J*, **8,** 237.

Verschaeve L and Maes A (1998). Genetic, carcinogenic and teratogenic effects of radiofrequency fields. *Mutat Res*, **410**, 141.

References

Vijayalaxmi D Z, Frei M R, Dusch S J, Guel V, Meltz M L and Jauchem J R (1997a). Frequency of micronuclei in the peripheral blood and bone marrow of cancer-prone mice chronically exposured to 2450-MHz radiofrequency radiation. *Radiat.Res*, **147**, 495.

Vijayalaxmi D Z, Mohan N, Meltz M L and Wittler M A (1997b). Proliferation and cytogenetic studies in human blood lymphocytes exposed in vitro to 2450-MHz radiofrequency radiation. *Int J Radiat Biol*, **72**, 751.

Vijayalaxmi D Z, Frei M R, Dusch S J, Guel V, Meltz M and Jauchem, J R (1998). Correction of an error in calculation in the article "Frequency of micronuclei in the peripheral blood and bone marrow of cancer prone mice chronically exposed to 2450 MHz radiofrequency radiation. (*Radiat Res*, **147**, 495, 1997). *Radiat Res*, **149**, 308.

Vijayalaxmi, D Z, Seaman R L, Belt M L, Doyle J M, Mathur S P and Prihodas T J (1999). Frequency of micronuclei in the blood and bone marrow cells of mice exposed to ultra-wideband electromagnetic radiation. *Int J Radiat Biol*, **75(1)**, 115.

Violanti J M (1997). Cellular phones and traffic accidents. *Public Health*, **111**, 423.

Violanti, J M (1998). Cellular phones and fatal traffic collisions. *Accid Anal Prev*, **30**, 519.

Violanti J M and Marshall J R (1996). Cellular phones and traffic accidents: an epidemiological approach. *Accid Anal Prev*, **29**, 265.

Vollrath L, Spessert R, Kratzsch T, Keiner M and Hollmann H (1997). No short-term effects of high frequency electromagnetic fields on the mammalian pineal gland. *Bioelectromagnetics*, **18**, 376.

Vorobyov V V, Galchenko A A, Kukushkin N L and Akoev I G (1997). Effects of weak microwave fields amplitude modulated at ELF on EEG of symmetric brain areas in rats. *Bioelectromagnetics*, **18**, 293.

Wachtel H, Seaman R and Joines W (1975). Effects of low-intensity microwaves on isolated neurons. *Ann N Y Acad Sci*, **247**, 46.

Wagner P, Roschke J, Mann K, Hiller W and Frank C (1998). Human sleep under the influence of pulsed radiofrequency electromagnetic fields: a polysomnographic study using standardized conditions. *Bioelectromagnetics*, **19**, 199.

Wainwright P R (*in press*). Thermal effects of radiation from cellular telephones. *Phys Med Biol.*

Walters T J, Mason P A, Sherry C J, Stevens C and Merritt J H (1995). No detectable bioeffects following acute exposure to high peak power ultra-wide band electromagnetic radiation in rats. *Aviat Space Environ Med*, **66(6)**, 562.

Wang B and Lai H (1999). Acute exposure to pulsed 2450-MHz microwaves affects water-maze performance of rats. *Bioelectromagnetics*, **21**, 52.

Wang Z, Van Dorp R, Weidema A F and Ypey D L (1991). No evidence for effects of mild microwave irradiation on electrophysiological and morphological properties of cultured embryonic rat dorsal root ganglion cells. *Eur J Morphol*, **29**, 198.

Which? (2000). The ring of truth. London, Consumers Association, April, p 13.

WHO (2000). Electromagnetic fields and public health: cautionary policies. Geneva, World Health Organization.

Williams W M, Del Cerro M and Michaelson S M (1984). Effect of 2450 MHz microwave energy on the blood–brain barrier to hydrophilic molecules. B. Effect on the permeability to HRP. *Brain Res Rev*, **7**, 171.

Williams G M and Whysner J (1996). Epigenetic carcinogens: evaluation and risk assessment. *Experi Toxicol Pathol*, **48**, 189.

Wolff S, James T L, Young G B, Margulis A R, Bodycote J and Afzal V (1985). Magnetic resonance imaging: absence of *in vitro* cytogenetic damage. *Radiology*, **155**, 163.

Wood S J, Scott I R, Netell J J and Tattersall J E H (2000). Effects of low intensity radiofrequency electromagnetic fields on electrical activity in rat hippocampal slices. *Personal communication* (*manuscript in preparation).*

Wu R Y, Chiang H, Shao B J, Li N G and Fu Y D (1994). Effects of 2.45 GHz microwave radiation and phorbol ester 12-O-tetradecanoylphorbol-13-acetate on dimethylhydrazine-induced colon cancer in mice. *Bioelectromagnetics*, **15**, 531.

Yang H K, Cain C A, Lockwood J and Tompkins W A F (1983). Effects of microwave exposure on the hamster immune system. I. Natural killer cell activity. *Bioelectromagnetics*, **4**, 123.

Yao K T S (1976). Cytogenic consequences of microwave incubation of mammalian cells in culture. *Genetics*, **83**, S84.

Yao K T S (1982). Cytogenic consequences of microwave incubation of mammalian cells intubated *in vitro. J Hered*, **73**, 133.

Yao K T S and Jiles M M (1970). Effects of 2450 MHz microwave radiation on cultivated rat kangeroo cells. IN *Biological Effects and Health Implications of Microwave Radiation* (S F Cleary, Ed), Proceedings of Medical College of Virginia Symposium, 1969, p 123. Richmond VA, Department of Health, Education and Welfare, Public Health Service, Environmental Health Service, Bureau of Radiology.

Zalyubovskaya N P and Kiselev R I (1978). Biological oxidation in cells under the influence of radiowaves in the millimeter range. US Joint Pub Research Service Rep, JPRS L/7957, p 5.

Zook B C (1998). Radiofrequency irradiation of the brain of rats. Presented at BEMS 20th Annual Meeting, St Petersburg, Florida, June 1998.

Zook B C (1999). The carcinogenicity of RF radiation to the brain of rats. IN *Proceedings 11th International Congress of Radiation Research*. Dublin, Radiation Research Society, 1999, p 280.

Glossary

The descriptions below are intended to help the reader understand the text; they are not necessarily definitive scientific terms, for which the reader is advised to consult specialist sources.

Words in bold are defined separately.

Analogue	The original cellular technology used in the transmission of speech by Vodafone and Cellnet since 1985, operating as an analogue system at 900 MHz. Typically accessed by high powered phones installed in cars.
AM	Amplitude modulation.
Action potential	Voltage produced across a nerve cell membrane by a stimulus. It arises from the entry of sodium ions across the cell membrane, which results in membrane depolarisation.
Antenna	Device designed to radiate or receive electromagnetic energy.
APC	Adaptive Power Control. System used to control mobile phones and base stations in order to ensure that the radiated power does not exceed the minimum consistent with high quality communication. The system effectively operates to reduce average radiated powers.
Base station	Facility providing transmission and reception for radio systems. For macrocells, the infrastructure comprises either roof- or mast-mounted antennas and an equipment cabinet or container. For smaller microcells and picocells, the antennas and other equipment may be housed in a single unit.
Case–control study	An investigation into the extent to which a group of persons with a specific disease (the cases) and comparable people who do not have the disease (the controls) differ with respect to exposure to putative risk factors.
CDMA	Code Division Multiple Access. System that encodes signals to a number of users, so that all of these users can simultaneously use a single, wide frequency band. Each user's handset decodes the information for that user, but cannot access information for any other user.
Cell and Cellular	A cell in the context of mobile phone technology is the area of geographical coverage from a radio base station. "Cellular" describes such systems, but is often used to distinguish the original analogue systems from the later digital **PCN** systems, although the latter themselves have cells.
Chromosomes	Rod-shaped bodies found in the **nucleus** of cells in the body. They contain the genes or hereditary material. Human beings possess 23 pairs.
Cohort study	An investigation into the extent to which a group of individuals (the cohort) about whom certain exposure information is collected, and the ascertainment of the occurrence of diseases at later times. For each individual, information on prior exposures can be related to subsequent disease experience.

CJD	Creutzfeldt-Jakob disease.
Confidence interval (CI)	An interval calculated from data when making inferences about an unknown parameter. In hypothetical repetitions of the study, the interval will include the parameter in question on a specified percentage of occasions (for example, 95% for a 95% confidence interval).
CW	Continuous wave.
Decibel (dB)	A measure of the increase or decrease in power at two points expressed in logarithmic form. Gain = $10 \log_{10}(P_2/P_1)$.
DECT	Digital Enhanced Cordless Telecommunications.
Digital	Technology introduced in the 1990s as a method of transmitting speech and data. Offers increased security, and technical advantages with low powered phones.
DNA	Deoxyribonucleic acid. The compound that controls the structure and function of cells and is the material of inheritance.
DTX	Discontinuous transmission. System regulating mobile phones to ensure that transmission occurs only during speech. The system has the effect of reducing the time of exposure to approximately half (assuming an equal conversation).
EEG	Electroencephalogram. Measurement of changing voltages associated with brain activity.
EIRP	Equivalent isotropically radiated power. This is the power that would have to be emitted in *all directions* to produce a particular intensity and so takes account of the transmitter power plus the characteristics of the antenna.
Electric field	Produces a force on a charged object. Measured in units of volts per metre.
Electromagnetic fields	The electric and magnetic fields associated with electromagnetic radiation.
Electromagnetic radiation	A wave of electric and magnetic energy that travels or *radiates* from a source.
EMF	Electromagnetic field.
ERP	"Evoked" or "Event-related" potential.
FDD	Frequency division duplex.
Frequency	The number of complete cycles of an electromagnetic wave in a second. Measured in units of **hertz (Hz)**.
Genes	Biological units of heredity. They are arranged along the length of **chromosomes**.
Gene expression	The realisation of genetic information encoded in **genes** to produce functional protein or **RNA**.
GSM	Global System for Mobile Communications or *Groupe Spéciale Mobile*. The international, pan-European operating standard for the new generation of digital cellular mobile communications. Enables mobile phones to be used across national boundaries. PCN operators work to the same standard but at different frequency allocations.
Hertz (Hz)	Unit of frequency. One cycle per second.

IMT - 2000	International Mobile Telecommunications - 2000. International name for **UMTS**.
Infrared radiation	**Electromagnetic radiation** capable of producing the sensation of heat and found between visible radiation and **radiofrequency radiation** in the electromagnetic spectrum.
Intensity	The power crossing unit area normal to the direction of wave propagation. Measured in units of watts per square metre (W/m^2). See also **power density**.
Ion	Electrically charged atom or group of atoms.
Ion channel (gate)	Protein that allows the passage of ions across a membrane, down a concentration gradient.
Ion pump	A protein pump that moves ions across a membrane against a concentration gradient.
Magnetic field B	Produces a force on a charged object moving at an angle to it. Measured in tesla (T). See also **magnetic flux density**.
Magnetic flux density	Produces a force on a charged object moving at an angle to it. Measured in tesla (T). See also **magnetic field B**.
Magnetite	Naturally occurring oxide of iron with magnetic properties
Microwave	Electromagnetic radiation of ultra high frequencies between 1 GHz and 300 GHz.
Molecule	Smallest portion of a substance that can exist by itself and retain the properties of the substance.
Mutation	Chemical change in the DNA in the nucleus of a cell. Mutations in sperm or egg cells, or their precursors, may lead to inherited effects in children. Mutations in body cells may lead to effects in the individual.
Neuron(e)	Nerve cell. Basic unit of the nervous system, specialised for the transmission of electrical impulses.
Nucleus	The controlling centre of higher cells. Contains the important material **DNA**.
Order of magnitude	Quantity given to the nearest power of ten. A factor of ten or so.
OFTEL	Office of Telecommunications.
PCN	Personal Communications Network. A mobile system principally directed towards the hand portable, domestic user market and operating with **digital** technology at 1.8 GHz. The two main UK operators are One 2 One and Orange.
Power density	The power crossing unit area normal to the direction of wave propagation. Measured in units of watts per square metre (W/m^2). See also **intensity**.
Radiofrequency radiation	Electromagnetic radiation used for telecommunications and found in the electromagnetic spectrum at longer wavelengths than **infrared radiation**.
Relative risk	The ratio of the disease rate in the group under study to that in a comparison group, with adjustment for confounding factors such as age, if necessary.
RF	**Radiofrequency radiation**.

Risk	The probability or likelihood of injury, harm or damage occurring.
RNA	Ribonucleic acid.
SAR	**Specific energy absorption rate**.
Significance level	The probability of obtaining a result at least as extreme as that observed in the absence of a raised risk. A result that would arise less than 1 in 20 times in the absence of an underlying effect is often referred to a being "statistically significant".
Specific energy absorption rate	The rate at which energy is absorbed by unit mass of tissue in an electromagnetic field. Measured in units of watts per kilogram (W/kg).
Third Generation	The next evolution of mobile phone technology, based on **UMTS** and expected to result in widespread use of video phones and access to multimedia information.
TDD	Time Division Duplex.
TDMA	Time division multiple access. System that divides each frequency band into a number of time slots, each allocated to a single user. Allows several users to operate on the same frequency at the same time.
TETRA	Terrestrial enhanced trunk radio system.
Transcription	The synthesis of **RNA** from **DNA**.
UMTS	Universal Mobile Telecommunications System.
Wavelength	Distance between two successive points of a periodic wave in the direction of propagation, in which the oscillation has the same phase. Measured in units of metres.

Quantities and units used to characterise electromagnetic radiation

Quantity	Unit	Symbol
Frequency	hertz	Hz
Wavelength	metre	m
Electric field strength	volt per metre	V/m
Magnetic field strength*	ampere per metre	A/m
Magnetic field, B/Magnetic flux density*	tesla	T
Intensity/Power density	watt per square metre	W/m^2
Specific energy absorption rate (SAR)	watt per kilogram	W/kg

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

Appendix A

Independent Expert Group on Mobile Phones

Following widespread consultation with interested parties, the Expert Group on Mobile Phones was set up under the Chairmanship of Sir William Stewart FRS, FRSE. Members of the Expert Group were chosen to encompass expert knowledge in epidemiology and experimental biology related to exposures to electromagnetic fields and radiofrequencies, and also knowledge of social science, risk perception and legal issues. There are members with medical and scientific skills in oncology, physics, statistics and neurophysiology. One member is from the World Health Organization (WHO) and there are two members from the Advisory Group on Non-ionising Radiation (AGNIR) of the National Radiological Protection Board (NRPB). There are two members with lay interests.

In addition to full Expert Group members there were observers from the Board of NRPB, from the Department of Health (DH) and from the Department of Trade and Industry (DTI). NRPB staff provided the secretariat and administrative support for the work of the Group.

Brief resumes for Members of the Expert Group are given below.

Chairman

Professor Sir William Stewart FRS, FRSE

Sir William Stewart is President of the Royal Society of Edinburgh and Chairman of the Microbiological Research Authority. He served as Chief Scientific Adviser to the Prime Minister and to the Government (1990–1995). He is a biologist by training. He is Chairman of Tayside University Hospitals NHS Trust. He was the first Head of the UK Government's Office of Science and Technology and has served on various advisory committees including the Royal Commission on Environmental Pollution and the Natural Environment Research Council. He was Chief Executive of the Agricultural and Food Research Council and a former Vice-president of the Royal Society of London.

Vice-chairman

Professor L J Challis OBE

Lawrence Challis is Emeritus Professor of Physics at the University of Nottingham. His university education and the first years of his academic career were at the University of Oxford (1951–59); he then moved to the University of Nottingham. He was appointed to an established chair in 1971, was Pro-Vice-Chancellor before his retirement in 1998 and is now a Leverhulme Emeritus Fellow. His current research interests are on the properties of low dimensional semiconductors. He has published 230 papers and, in 1994, was awarded the Holweck Medal and Prize for his research by the Institute of Physics/French Physical Society. In 1996 he was awarded the OBE for services to scientific research. He has chaired the Royal Society Grant Board for Mathematics and Physics, the Physics

Committee of the then Science and Engineering Research Council and the Solid State Division of the Institute of Physics. He is currently Honorary Editor of *Reports on Progress in Physics* and was until recently Chairman of the European Commission Evaluation Panel for Access to Research Infrastructures.

Members

Professor L W Barclay OBE, FREng

Professor Les Barclay was Deputy Director at the Radiocommunications Agency, responsible for research and radio technology. He is now a consultant in radio regulation, spectrum management and radio propagation, and is a visiting professor at the universities of Bradford, Lancaster and Surrey. He has been chairman of the study group on propagation within the International Telecommunication Union and chairman of the Scientific Committee on Telecommunications within the International Union of Radio Science. He is a Fellow of the Royal Academy of Engineering and is currently a member of the Electronics and Communications Divisional Board of the Institution of Electrical Engineers. He has been awarded the OBE and the Polar Medal.

Mrs M-N Barton MBE

Marie-Noëlle Barton is the National Manager of the Women Into Science and Engineering (WISE) campaign, run by the Engineering Council, the Engineering Marine Training Authority, and the Engineering Employers' Federation. She works with educationalists, employers, Government Departments, politicians and the media to spread the word about WISE. She also oversees the WISE in Northern Ireland, WISE in Scotland and WISE in Wales campaigns to cater for the specific needs of women in these countries. Previously she has worked as a careers adviser with local authorities, and was a language teacher between. She holds an Honorary Doctorate of Technology from Staffordshire University.

Professor C Blakemore FRS

Colin Blakemore studied medical sciences at the University of Cambridge and completed his PhD at the University of California, Berkeley, in 1968. He taught at the University of Cambridge for 11 years and in 1979 took up the Chair of Physiology at the University of Oxford. He is also Director of the Oxford Centre for Cognitive Neuroscience, and was President of the British Association for the Advancement of Science for 1997–98. He has worked as a Visiting Professor at the Massachusetts Institute of Technology, New York University, the University of California and the Salk Institute, and also in Japan, Switzerland, Italy, France, the Czech Republic and China. He holds the degree of DSc from the universities of Cambridge and Oxford, honorary doctorates from Aston and Salford Universities and an honorary fellowship from Cardiff University. He is a Fellow of the Royal Society, the Academy of Medical Sciences and the Institute of Biology, a member of the Academia Europaea and a Foreign Member of the Royal Netherlands Academy of Arts and Sciences. His research has been concerned with many aspects of vision and the early development of the brain. His awards include the 1996 international Alcon Prize for vision research and the 1989 Royal Society Michael Faraday Award for the furtherance of the public understanding of science. He is a member of the NRPB Advisory Group on Non-ionising Radiation.

Professor D N M Coggon

David Coggon studied mathematics and medicine at the universities of Cambridge and Oxford. He is currently Professor of Occupational and Environmental Medicine at Southampton University where he works in the Medical Research Council Environmental Epidemiology Unit. He has been engaged in epidemiological research for more than 20 years, focussing mainly on occupational and

environmental causes of disease. Special interests include the relation of musculoskeletal disorders to physical activities in the workplace, and the health effects of chemical pollutants. He is also a consultant occupational physician and carries out clinical work for Southampton University Hospitals Trust. He is a Fellow of the Academy of Medical Sciences, Chairman of the Advisory Committee on Pesticides, and a member of the Expert Panel on Air Quality Standards and the Industrial Injuries Advisory Council.

Professor Sir David Cox FRS

Sir David Cox was educated at St John's College, Cambridge (MA), and the University of Leeds (PhD). After working in industry, he held academic posts at the University of Cambridge and Birkbeck College, University of London. In 1966 he became Professor of Statistics, and in 1970 Head of the Mathematics Department, at the Imperial College of Science and Technology, University of London. He is a former Warden (1988–94) and an honorary Fellow of Nuffield College, Oxford. He is a Fellow and former Member of Council of the Royal Society and was President of the International Statistical Institute from 1995 to 1997. He has been President of the Royal Statistical Society. He holds a number of awards, including honorary Fellowships at St John's College, Cambridge, and the British Academy, the Guy Medal (in Silver and in Gold) from the Royal Statistical Society, and the degree of DSc from a number of universities. He has also been awarded the Weldon Memorial Prize, University of Oxford, and the Kettering Prize and Gold Medal for Cancer Research. He is a Foreign Honorary Member of the US. National Academy of Sciences. He was editor of *Biometrika* from 1966 to 1991.

Mr J Fellows

John Fellows is a graduate of the University of Edinburgh (MA, politics) and a winner of the Sir William Darling Memorial Prize (1997) for an outstanding contribution to the reputation for the university through project work in local schools. He was President of Edinburgh University Students' Association for 1998–99: his activities included lobbying the Scottish Parliament to set up an inquiry into student finance and campaigning against the Bank of Scotland business partnership with Pat Robertson Financial Services in the USA; other areas of interest included the promotion of widening access to university, and the adoption of ethical investment policies within the University. He is currently a co-ordinator for the Lothian Equal Access Programme for Schools, a project to widen access to higher education, based at the Heriot-Watt and Napier Universities in Edinburgh.

Dr M Repacholi FACPSEM, FARPS

Michael Repacholi is a graduate of the University of Western Australia (BSc, physics), London University (MSc, radiation biology) and Ottawa University (PhD, biology). He is the author or co-author of over 150 scientific publications. He is at present the Coordinator, Occupational and Envirnomental Health, at the World Health Organization (WHO) in Geneva, and has participated in ten WHO non-ionising radiation task groups. He is an Emeritus Chairman of the International Commission on Non-Ionizing Radiation Protection, Fellow and Past President of the Australian Radiation Protection Society and of the Australasian College of Physical Scientists and Engineers in Medicine. He is also a Fellow of the Australian Institute of Physics and a member of the Health Physics Society and of the Bioelectromagnetics Society.

Professor M Rugg FRSE

Michael Rugg obtained his PhD in 1979. Following a postdoctoral year at the University of York, he was appointed to a lectureship in psychology at the University of St Andrews, where he became Professor of Psychology and Head of School in 1992. In 1998 he moved to the Institute of Cognitive Neuroscience, University College London, as Professor of Cognitive Neuroscience and Wellcome Trust Principal Research Fellow. His principal research interests are the cognitive and neurological

bases of human memory, and the non-invasive investigation of human brain function through the use of electroencephalography and functional neuroimaging. During 1998 and 1999 he served on the Department of Health Working Group on Organophospates.

Professor A J Swerdlow

Anthony Swerdlow was educated in medicine at the universities of Cambridge and Oxford. After junior posts in clinical medicine, epidemiology and public health in the Oxford region and London, he worked in epidemiology at the University of Glasgow and at the Office of Population Censuses and Surveys before moving to the London School of Hygiene and Tropical Medicine in 1987. He is currently Professor of Epidemiology at the School. His research is in chronic disease epidemiology, mainly on cancer but also on other diseases including type 1 diabetes and CJD. His research interests have for many years included non-ionising radiation and he is currently a member of the NRPB Advisory Group on Non-ionising Radiation and of the Epidemiology Standing Committee of the International Commission on Non-Ionizing Radiation Protection.

Mr T R K Varma FRCS (Ed)

T R K Varma graduated in medicine (MB;BS) from the University of Bangalore, India, and is a Fellow of the Royal College of Surgeons of Edinburgh. He is currently Consultant Neurosurgeon at the Walton Centre for Neurology and Neurosurgery, Liverpool, and Honorary Cinical Lecturer at the University of Liverpool. He previously held the post of Consultant Neurosurgeon and Honorary Senior Lecturer at Ninewells Hospital and Medical School, Dundee. He is a member of the Council of the Society of British Neurological Surgeons (SBNS) and of the Clinical Standards Committee of SBNS. His special expertise is in the field of stereotactic and functional neurosurgery, including the use of chronic electrical stimulation of the brain in the treatment of movement disorders.

Observers

Professor A D Baddeley FRS *(Member of the National Radiological Protection Board)*

Dr H Walker *(Department of Health)*

Mr G Worsley *(Department of Trade and Industry)*

Secretariat

Dr J W Stather *(National Radiological Protection Board)*

Dr N A Cridland *(National Radiological Protection Board)*

Administrative Support

Karen Roberts *(National Radiological Protection Board)*

Maxine Smith *(National Radiological Protection Board)*

Kate Christie *(Chairman's Office, Dundee)*

Isa MacLean *(Chairman's Office, Dundee)*

Acknowledgements

The work of the Independent Expert Group on Mobile Phones has been dependent on the co-operation of many individuals and organisations. We are particularly grateful to Professor Ken Foster, Professor Robert Adair, and Dr Mario Cortina Borja for helpful advice and useful discussions. Special thanks are also due to Dr Mika Koivisto, who allowed us access to his data prior to publication and to Dr Alan Preece, who permitted us to re-analyse his data on reaction time.

Our work could not have been completed in so short a time without the hard work and excellent administrative support provided by Karen Roberts, Maxine Smith, Kate Christie and Isa MacLean to all of whom we express our gratitude.

Appendix B

Written Evidence

Evidence was submitted to the Expert Group by 174 individuals and organisations:

Mr H Aitkin, Edinburgh
Anonymous, Manningtree, Essex
Anonymous, Surrey
Mrs A C Arnold Silk, Princes Risborough, Bucks
Mr & Mrs R S Ashwood, Tamworth, Staffs
Ms S Atkinson, Atlantic Telecom, Aberdeen

Professor R Baker, Biotechnology & Biological Sciences Research Council (BBSRC), Swindon, Wilts
Dr M Bastide, Laboratoire D'Immunoglogical et Parasitologie, Université Montpellier I, France
Ms J Bates, Stockton-on-Tees, Cleveland
Mrs G Batter, London
Mr G Berry, Newry, Co Down, Northern Ireland
Mr H Best MP, House of Commons, London
Mr R Bolton, Broadcasting, Entertainment, Cinematograph and Theatre Union (BECTU), London
Mr C H Bowden, Department of the Environment, Transport and the Regions, London
Mr S Box, Holmer Green Upper School, Holmer Green, Bucks
Mrs J Bramwell, Russell Lower School, Ampthill, Beds
British Medical Association, London
Professor R Brook, Engineering and Physical Sciences Research Council (EPSRC), Swindon, Wilts
Ms J Brookes, Federation of Communication Services Ltd (FCS), London
Mrs G Burkitt, Holmer Green, Bucks
Mr P D Burrill, Oakwood, Leeds, Yorks

Ms M Cahill, The Wessex Astrologer Ltd
Professor I Campbell, The Maudsley Institute of Psychiatry, London
Dr G L Carlo, Wireless Technology Research, Washington, USA
Master A Carmody, Barnet, Herts
Mr E Carson, Orange PCS Ltd, Dundonald, Northern Ireland
Mr B Claridge, Holmer Green, Bucks
Mrs V Clarke, Oxford, Oxon
Mrs T Clarkson, Leeds, Yorks
Professor R Clausen-Sternwald, Burnley, Hants
Mr I Clement, Glasgow
Professor D Clements-Croome, Department of Construction Management and Engineering, University of Reading, Berks
Mrs F Clifford (e-mail)
Mr R Coghill, Coghill Research Laboratories, Pontypool, Gwent
Ms D C Collins, Belvedere, Kent
Mr B Compton, Solihull, West Midlands
Dr J Connor, Ministry of Defence, London
Dr R A F Cox, Fowlmere, Cambs

Dr D de Pomerai, School of Biological Sciences, University of Nottingham, Notts
Mr E P D'Alton, Irish Campaign Against Microwave Pollution (ICAMP), Dublin, Republic of Ireland
Mrs M Dafforn, Hook, Hampshire
Mr G H David, Aerial Group Limited, Chesham, Bucks
Mrs M Dean, Northern Ireland Families against Telecommunications Transmitter Towers (NIFATT), County Antrim, Northern Ireland
Dr M T Deans, London
Mr J M Dilks, Brant Brougton, Lincs
Mr K Diment, Wantage, Oxon
Mrs E G M Dixon, Christchurch, Dorset
Mrs A R Dobbs, Gilwern, Abergavenny
Mr M Dolan, Mobile Telecoms Advisory Group, Federation of the Electronics Industry, London
Dr D Doyle, Southern General Hospital NHS Trust, Glasgow
Mr M Durrant, Bucksburn, Aberdeen

Mr L Feetham, Winchester City Council, Winchester, Hants
Mr S Fielding, Maidstone, Kent
Mr A Firstenberg, Cellular Phone Taskforce, NewYork, USA
Mr A Foster, Ampthill, Beds
Mr P Foster, Holmer Green, Bucks
Mrs C Frank, London
Mr P Franklin, Orange plc, London
Mr A Freeman, BT Cellnet, Slough
Mr G Fulford, Leigh-on-Sea Town Council, Leigh-on-Sea, Essex

Ms C Gabriel, Microwave Consultants Limited, London
Mr R Gardner, Ministry of Defence, London
Mrs P Garrett, Long Crendon, Bucks
Ms C Gillan MP, House of Commons, London
Mr A W Glass, Aberdeen
Ms J Goddard, Ampthill, Beds
Professor J Golding, Unit of Paediatric and Perinatal Epidemiology, University of Bristol
Mr D Greenaway, Derriford, Plymouth
Mrs J Greenroyd, Milford Haven, Pembrokeshire
Mr J A Greensmith, Chester-Le-Street District Council, Co Durham

Professor L Hardell, Örebro Medical Centre, Örebro, Sweden
Mr Harkin, Communication Workers Union, London
Mr T Harrabin, Vodafone UK Limited, Newbury, Berks
Mr D Harris, Rickmansworth, Herts
Mr P Harrison, Nokia UK Limited, Farnborough, Hants
Mr J N W Hartley, Acklam, Middlesbrough, Cleveland
Mr J M Haynes, Horsham, Sussex
Professor D L Henshaw, H H Wills Physics Laboratory, University of Bristol
Mr P Hibbert (e-mail)
Mr C Hickling, Plymouth, Devon
Mrs S M Hollamby, Herne Bay, Kent
Mr J Hughes, Ingatestone, Essex
Dr G Hyland, Department of Physics, University of Warwick, Coventry, Warks

Dr H Irvine, Greater Glasgow Health Board, Glasgow

Miss S Jameson, Belfast, Northern Ireland

Ms E A Kelley, Council of Wireless Technology Impacts, Novato, California, USA
Professor R Kemp, Galson Sciences Ltd, Oakham, Rutland
Professor Dr M Kundi, Institute of Environmental Health, Vienna, Austria

Dr H Lai, Department of Bioengineering, University of Washington, Seattle, USA
Mr A Laurie, Scientific Generics, Cambridge, Cambs
Professor J Lawton, Natural Environment Research Council (NERC), Swindon, Wilts
Dr R A Lerski, Medical Physics Department, Ninewells Hospital, Dundee
Mr S Liss, Consumers' Association, London
Mr E Lorch, Holmer Green Upper School, Holmer Green, Bucks
Mrs S Ludvigsen, Wrenthorpe, Wakefield
Dr M Lundquist, The Bioelectromagnetic Hygiene Institute, Milwaukee, Wisconsin, USA

Mrs I M Macgillivray, Macduff, Banffshire
Mr D Maddock, Southampton, Hants
Councillor A Maskey, Belfast City Council, Belfast, Northern Ireland
Ms J Matthew, Goffs Oak, Herts
Mr G McAllister, Friends of the Earth Scotland, Edinburgh
Mr M McAllister, Department of Health, Social Security and Public Safety, Belfast, Northern Ireland
Mrs H McCrory, Dublin, Republic of Ireland
Mr J McDonald, Houston, Renfrewshire
Mr V McDonald, Glasgow
Dr T McManus, Deptartment of Public Enterprise, Dublin, Republic of Ireland
Mr G McPherson, Philips Consumer Communications, Basingstoke, Hants
Mr J Meredith, The Career Teachers' Association (NASUWT), Birmingham
Mr A Meyer, Halsey Meyer Higgins Solicitors, London
Mr J Mochnacz, Street, Somerset
Mr J B Moffatt, Celtic League, Farmhill, Mannin, Isle of Man
Dr A Morby, Cardiff University, Cardiff
Mrs S E Morris, Hartburn, Stockton on Tees
Mrs J Mortimer, Monifieth, Angus
Ms Joanne Mueller, USA
Mr R Murtagh, Fowey, Cornwall
Mr P Myott, Liverpool

Mr D Newman, Halton Friends of the Earth, Widnes, Cheshire
NRPB, Chilton, Didcot, Oxon

Mr B O'Brien MP, House of Commons, London
Ms C O'Connell, Dungarnan, County Waterford, Republic of Ireland
Dr I O'Connor, Carrigaline, County Cork, Republic of Ireland
Mr J J O'Neill, Grange Park Primary School, Sunderland, Tyne and Wear

Mr B Page, Luton Borough Council, Luton, Beds
Mr T R W Parker, The Church of Scotland General Trustees, Edinburgh
Mrs V A Parr, Chelmsford, Essex
B Paul, St Margarets Residents Against The Mast, Dover
Mr G P Pells, Didcot, Oxon
Mr A Philips, Powerwatch, Ely, Cambs
Dr A Preece, Biophysics Group, Bristol Oncology Centre, University of Bristol
Mrs M Price, Holmer Green, Bucks
Mr D Prior MP, House of Commons, London

Professor G K Radda, Medical Research Council (MRC), London
Mr M Rands, The Royal Society of Edinburgh, Edinburgh

Mr R Riedlinger, Abbotsford, British Columbia, Canada
Dr A Roberts, The Institution of Electrical Engineers, London
Mr P Roche, Northern Ireland Assembly, Belfast, Northern Ireland
Mr J Royds, Greystones, Co Wicklow, Republic of Ireland

Ms C Sage, Sage Associates, Santa Barbara, California
Dr C J Schilling, Schilling and Schilling, Consultants in Occupational Health, London
Mr A Scott, Salisbury, Wilts
Mrs E Scott, Ampthill, Beds
Alderman J Shannon, Northern Ireland Assembly, Belfast, Northern Ireland
Mr M Simpkins, Holmer Green, Bucks
Mr K Simpson, Bedford, Beds
Mr M Smith, Ampthill, Beds
Mrs M Somers, Cookridge, Leeds, Yorks
Mr D Stevens, Marketex (UK) Ltd, Barham, Kent
Mr P Sutton, Wantage, Oxon

Mr D K Tamerlane, Woking, Surrey
Dr J Tattersall, Defence Evaluation and Research Agency (DERA), Porton Down, Wilts
Mr B Taylor, Essex
Mrs C Terry, Dover, Kent
Mr L F W Thomas, Swanley, Kent
Mrs A Trattles, Stockton, Cleveland
Mr A E Trenary, Fowey, Cornwall
Mrs J Tiffiny, Watford, Herts
Ms R Tilly, London
Ms D Turnbull, Campaign Against Masts in East Oxford (CAMEO), Oxon
Ms M Turner, Monifieth, Dundee

Mr J Van Montfoort, Porlock, Somerset

Dr S J Watkins, Stockport Health Authority
Mr M Watson, Thame, Oxon
Mrs J Watts, Long Ashton, Avon
Councillor Miss M Wellman, Fowey, Cornwall
Mr N Wenham, Ambrosden, Oxon
Mr P & Mrs J Weychan, Fowey, Cornwall
Ms J Whalley MP, House of Commons, London
Ms R C Whetstone, One 2 One, Borehamwood, Herts
Dr A G Whittaker, Department of Chemistry, University of Edinburgh
Mr W G Williams, Southport
Mr P Willis MP, House of Commons, London
Mr A Wilson, Macclesfield, Cheshire
Mr L Wilson, Microshield Industries plc, Enfield, Middlesex

Appendix C

Meetings

Open Meetings

Summaries of the open meetings of the Expert Group have been placed on the Group's web site at www.iegmp.org.uk.

Thursday 11 November 1999, The Playfair Library, University of Edinburgh

Thursday 9 December 1999, The Medical School, University of Liverpool

Thursday 20 January 2000, The Medical School, University of Cardiff

Thursday 27 January 2000, Scientific Societies Lecture Theatre, New Burlington Place, London

Thursday 3 February 2000, Peter Froggatt Centre, The Queen's University of Belfast

Closed Sessions

Summaries of the oral evidence presented to the Expert Group by 28 witnesses in closed sessions have been placed on the Group's web site at www.iegmp.org.uk.

Friday 10 September 1999, Adam House, Adam Street, London

Witnesses	Dr P J Dimbylow, National Radiological Protection Board (NRPB)
	Dr S M Mann, National Radiological Protection Board (NRPB)

Friday 8 October 1999, Adam House, Adam Street, London

Witnesses	Dr A F McKinlay, National Radiological Protection Board (NRPB)
	Professor P Ramsdale, One 2 One
	Ms R Whetstone, One 2 One

Friday 12 November 1999, The Elder Room, University of Edinburgh

Witnesses	Dr H Irvine, Greater Glasgow Health Board
	Dr R A Lerski, Ninewells Hospital, Dundee
	Mr G McAlister, Friends of the Earth Scotland
	Mr J McDonald, Houston, Renfrewshire

Thursday 9 December and Friday 10 December 1999, Crowne Plaza Hotel, Liverpool

Witnesses
Dr A W Preece, University of Bristol
Dr G J Hyland, University of Warwick
Mr M Dolan, Federation of the Electronics Industry
Professor P Ramsdale, One 2 One
Mr P Rumbalow, Orange plc
Mr P Harrison, Nokia UK Limited
Mr J Berle, Federation of the Electronics Industry

Thursday 20 January and Friday 21 January 2000, The Angel Hotel, Cardiff

Witnesses
Mr A Philips, Powerwatch
Mr R Gardner, Ministry of Defence
Mr J Mackay, Ministry of Defence
Mr K Tench, Ministry of Defence
Dr J Tatersall, Defence Evaluation and Research Agency (DERA), Porton Down
Dr D de Pomerai, University of Nottingham

Thursday 3 February and Friday 4 February 2000, The Stormont Hotel, Belfast

Witnesses
Dr H Kennedy, Northern Ireland Families against Telecommunications Transmitter Towers (NIFATT)
Mrs M Dean, Northern Ireland Families against Telecommunications Transmitter Towers (NIFATT)
Mr J Royds, Greystones, Co Wicklow
Mr A Meyer, Halsey, Meyer Higgins, Solicitors

Friday 3 March 2000, Adam House, London

Witnesses
Mr P Myott, Liverpool
Mr L Wilson, Microshield Industries

Tuesday 21 March – Friday 24 March 2000, NRPB, Chilton, Didcot

Witness
Professor R H Clarke, National Radiological Protection Board (NRPB)

Thursday 6 April 2000, Royal Over-Seas League, London