Review Article

An Epidemiological Review of Mobile Telephones and Cancer

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Abstract

Mobile telecommunication technology became commercially available about 20-25 years ago in different countries around the world. The industry has grown exponentially over the years and, currently, the number of mobile phone users is estimated to be over 3.8 billion, more than half the world's population. Thus, because of such a large population-at-risk, any health hazard from these devices promises to have a large epidemiological impact. Intense speculation and investigation into the relationship between mobile phone usage and cancer has led to the publication of numerous, often contradictory, reports on this subject. This review aims to provide a large body of reported evidence to help medical professionals disseminate evidence-based information to their patients. ©

Introduction

The mobile telecommunications industry has seen astounding growth since it was first introduced about 20-25 years ago in various countries. At present, there are about 3.8 billion mobile phone users in the world, accounting for more than half the total population.[1] With such high usage in the general population and with use beginning in childhood, we can expect a large number of people to be exposed to electromagnetic radiation for a long period of time. The relation of mobile phone usage and cancer has been a long-standing suspicion and, often, contradictory reports have been presented on this subject. This review attempts to present a comprehensive body of recent data on this issue to help medical professionals disseminate evidence-based information to their patients.

Basic Physics Pertaining To Mobile Phones

Mobile phone handsets are devices that transmit and receive radio frequency (RF) waves. These RF waves are transmitted bidirectionally between the handset and the base station towers located terrestrially.[2] The most commonly used wireless protocol is the Global System for Mobile communication (GSM) standard (accounting for 80% of all users), followed by the Code Division Multiple Access (CDMA) standard.

Mobile phones can further be of two types – analog and digital. Analog phones transmit and receive at a much higher power than do digital phones. This is because loss of data in analog transmission tends to be higher and, to counteract this effect, power has to be higher to maintain smooth transmission. In contrast, digital technologies involve binary encoding of data (voice) that resist data loss better. Hence, digital phone transmissions occur at a lower power. All mobile phones generate an electromagnetic field (EMF). An EMF is the space around a transmitter that contains waves of electromagnetic radiation. Human tissues absorb radiation to different extents, depending on their frequency (or wavelength, which is proportional to 1/frequency). For example, visible light (375-750 THz; 1THz=10^6 MHz) will be absorbed to a different extent compared to radio waves (1800 MHz), even if both are being transmitted at the same power (power=energy emitted per unit time; Watts). Thus, the specific absorption rate (SAR) is a measure of radiation absorption per unit weight of tissue and is expressed as watts per kilogram (W/kg). The radiation absorption of the human body is maximally efficient at frequencies between 30-300 MHz. The International Commission on Non-Ionising Radiation Protection (ICNIRP) has laid regulations to control to whole-body SAR below 2 W/kg (averaged over 10 grams of tissue) for users of mobile phone handsets.

Human Studies Of Mobile Phones And Cancer

A large proportion of research on incidence of cancer in mobile phone users has taken place in Europe, followed by North America, while Asian studies, mainly from Japan, are relatively few. Studies have attempted to study a wide variety of neoplastic phenomena affecting different body systems. The most frequently studied malignancies include the intracranial tumors, such as astrocytomas, gliomas and acoustic neuromas. The studies have mostly been case-control studies using patient-reported usage information to correlate duration and intensity of usage to the association with cancer.

Several studies have found a greater association of long-

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term mobile phone usage with intracranial malignancies. Mild et al[3] showed a significant association between analog phone use and brain tumors, with the greatest risk being for grade III-IV astrocytoma after a latency period of >10 years after first use. The association with tumors after a >10 year latency was significant for analog, digital as well as cordless phones. Lakhola et al[4] showed that there was no increased glioma risk with regular mobile phone use, even when analog or digital phones were analyzed separately. However, ipsilateral tumor risk was borderline for usage ≥ 10 years, while risk for contralateral usage was not significant. A study by Hardell et al[5] that sought to observe the prevalence of various cancers amongst regular mobile phone users found a significant association between acoustic neuromas and astrocytomas in users of analog, digital and cordless phones. The same study did not find significant associations for other tumors, such as salivary gland tumors, non-Hodgkin lymphoma (NHL) or testicular cancer. Another study by Hardell et al[6] quantified exposure in terms of >2000 hours of cumulative usage of mobile phones and studied associations with various cancers. At this level of exposure, the risk for malignant brain tumors was significantly higher for all three types of mobile phones. Also, ipsilateral exposures with these phones were associated with risk for cancer. The same study also found that the risk for developing high-grade astrocytoma was higher with usage >10 years with analog phones and digital phones, but not with cordless phones. Another study using the 10-year exposure criteria, by Schuz et al,[7] found an association with gliomas, though not with meningiomas or any tumors with lower durations of exposure. Hepworth et al[8] found that glioma occurrence was associated with ipsilateral mobile phone usage, while Schoemaker et al[9] made a similar observation for acoustic neuromas. Another study by Hardell et al[10] found that use of analog phones increased the risk for brain tumors, which increased on ipsilateral usage. High-risk groups identified in this study were persons aged 20-29 years having an exposure for >5 years from either analog or cordless phones. Two recent meta-analyses showed that long-term (>10 years) usage of mobile phones was associated with a greater risk of intracranial tumors,[11] with the greatest risk being for ipsilateral gliomas and ipsilateral acoustic neuromas.[12]

In-vitro studies of human glioma cells (MO54), measuring phosphorylation of various heat-shock proteins, showed no increased tumorigenic effects of mobile phone radiation.[13] Several studies,[14-18] however, have not shown any association between intracranial malignancies and mobile telephone usage. Some of these studies have included exposures of >10 years,[18] exposure from cordless phone base-units,[17] or even predominant unilateral use.[16] Two time-trend analyses have been published highlighting the change in incidence of various tumors since the introduction of mobile phone technology. Both showed no significant rise in the incidence of intracranial malignancies despite the exponential growth of the mobile telephone industry.[19,20] A Norweigian cohort study[21] found that all standardized incidence ratios (tumor incidence in exposed group divided by incidence in general Norwegian population) included 1 in the 95% confidence interval for brain tumors, acoustic neuromas, salivary gland tumors, eye tumors, leukemias, including after long-term (≥ 10 yrs exposure) to mobile phone radiation.

Sadetzki et al[22] showed that there was a significant association between the occurrence of benign or malignant parotid neoplasms and the use of mobile phones. The study found that individuals with high exposure and non-hands-free users were an at-risk group for developing these tumors on the side of predominant usage. Hardell et al[23] studied the association between NHL and mobile phone usage. The study found that B-cell type malignancies were not associated with mobile phone use. T-cell NHL after >5 years of use had a higher occurrence in users of cordless phones, but not digital or analog mobile phones. Also, risks for certain subtypes like cutaneous T-cell lymphoma and T-cell lymphoma/leukemia were raised for users of digital and cordless phones, but not analog phones. Studies on other cancers by Hardell et al[24] found no association between mobile phone usage and testicular cancers (seminoma and non-seminoma tumors). Linet et al[25] studied lifetime exposures to mobile phone radiation as <10 hours, 10-100 hours and >100 hours and found no associations, for any of the groups, with incidence of NHL. A study of malignant parotid tumors by Lonn et al[26] showed no association with mobile phone exposure, even when exposures exceeded 10 years.

Though the evidence is largely conflicting and there is no clear, unanimous association with any particular cancer, certain trends have been prominent. Use of analog technology is probably associated with more malignant tumors than digital. This is probably the effect of the higher power of transmission associated with analog phones. Cordless phones have also been implicated in several studies. Exposure to mobile phones for greater than 10 years has been found in many studies to raise the risk of cancer. Usage of the mobile handset predominantly on a particular side has been shown to raise the risk of cancers arising on that side, probably after many years of exposure. One study[27] highlighted important urban-rural differences in incidence rates of cancers associated with mobile phone usage. Given the fact that most of the Indian population lives in rural areas, this may be an important epidemiological factor mitigating the overall impact of cellular technology on cancer in India. Given the current evidence, these factors can, at best, be considered as possible risk factors and be avoided as much as possible.

Though significant associations between mobile phone usage and cancer have been shown, it must be kept in mind that the most reliable studies – prospective cohort studies – are lacking.[28] In the absence of these, a causal relationship cannot be established for mobile phone radiation and cancer. However, these case-control studies and retrospective analyses direct further research and help in formulating study design for large cohort studies.

Animal Studies Of Mobile Phones And Cancer

Various animal models have been used to test effects of RF radiation on cancer. Salient findings from relevant animal studies have been summarized in Table 1.[29-48] Radiation exposures simulating mobile phone use are usually standardized using the SAR and the specific frequencies associated with mobile telephones or cordless phones. Studies have been performed to assess risk of RF radiation in causing cancers in animals, as well as the risk of enhancing tumor activity caused by known carcinogens. Several studies have used chemical- or radiation-induced tumors as controls and compared effects of RF radiation on the test group. Other studies have used transgenic animal models, while some have used virus-infected animal models to initiate tumorigenesis and then observe effects of RF on tumor behaviour. In vitro studies have also been performed using animal neural cell lines. On the whole, the available data suggest that RF radiation exposure simulating mobile telephone usage neither initiates, nor promotes carcinogenesis in animal models.

Conclusion

There is considerable public concern about effects of mobile phone radiation on carcinogenesis. Since the head

Table 1 : Animal studies evaluating relationship between radio frequency radiation and cancer.

Study	Results
Yilmaz et al[29]	Speech-condition exposure vs. no exposure in Sprague-Dawley rats. 20 min/day exposure for 1 month with 900 MHz mobile phone radiation did not alter anti-apoptotic bcl-2 protein in brain and testes.
Moquet et al[30]	Murine neuroblastoma (N2a) cells: Exposed 935 MHz GSM radiation vs. no exposure. Continuous exposure for 24h showed no significant differences in apoptosis compared to the unexposed group.
Smith et al[31]	Exposure of 2h/day, 5d/week up to 104 weeks to GSM or DCS wireless signals at SARs of 0.44, 1.33 and 4.0 W/kg was studied in Wistar rats. No significant differences between exposed and unexposed rats in terms of: incidence of primary neoplasms, number of rats with >1 primary neoplasm, multiplicity and latency of neoplasms, number of rats with metastases and number of benign and malignant neoplasms.
Sommer et al[32]	UMTS exposure (24h/day, 7d/week, 0.4 W/kg SAR) vs no exposure in AKR/J mice (animal lymphoma model)No differences between groups in terms of: number of ill animals, mean survival time and severity code of disease.
Oberto et al[33]	Exposure to 900 MHz radiation pulsed at 217 Hz at whole-body SAR 0.5, 1.4 or 4 W/kg in Pim1 transgenic mice exposed for 1h/day, 7d/week. No effect on incidence of tumors at any site.
Shirai et al[34]	Incidence of brain tumors in F344 rats exposed to 1.95 GHz W-CDMA radiation at brain SAR of 0.67 and 2 W/kg. There was no significant increase in incidence or number of brain tumors, either in the males or females, in the exposed rats.
Tillmann et al[35]	Carcinogenic effects of 902 MHz and 1747 MHz GSM and DCS radiation on B6C3F1 mice exposed for 2h/day, 5d/week over 2 years to SARs of 0.4, 1.3 4.0 W/kg. There was no significant increase in the incidence of any particular tumor type in the exposed groups.
Joubert et al[36]	Cultured cortical neurons from Wistar rats: Non-exposure vs. exposure to 900 MHz GSM radiation at SAR 0.25 W/kg for 24h. No significant increase in apoptosis rate between exposed and unexposed cells 0 and 24h post-exposure.
Yu et al[37]	900MHz GSM radiation did not enhance DMBA-induced mammary tumor development in Sprague-Dawley rats.
Huang et al[38]	DMBA-induced skin tumorigenesis was not seen to be promoted in mice exposed to 849 MHz or 1763 MHz radio frequency radiation. A control group treated with a tumor promoter (TPA) showed significant rise in skin tumors.
Sommer et al[39]	Effects of 900 MHz GSM radiation (24h/day, 7d/week at SAR 0.4 W/kg) on AKR/J miceSurvival rate and lymphoma incidence was unchanged in exposed mice compared to unexposed controls.
Anane et al[40]	900MHz GSM radiation did not enhance DMBA-induced mammary tumor development in Sprague-Dawley rats.
La Regina et al[41]	There was no significant effect on the incidence of spontaneous tumors in F344 rats chronically exposed to 835.62 MHz FDMA or 847.74 MHz CDMA radiation.
Heikkinen et al[42]	Effects of radio frequency radiation on ultraviolet-induced skin tumorigenesis in ornithine transcarbamoylase transgenic and non-transgenic F344 rats. RF exposures did not give a statistically significant effect on the development of skin tumors in either transgenic or non-transgenic rats, or in combined analysis, but tumor development appeared slightly accelerated especially in non-transgenic rats.
Bartsch et al[43]	GSM signal at 900 MHz pulsed at 217 Hz did not increase tumor development in DMBA-induced mammary tumor in female Sprague-Dawley rats.
Heikkinen et al[44]	RF radiation of 902.5 MHz at SAR 1.5 W/kg or 902.4 MHz at 0.35 W/kg SAR did not increase X-ray-induced tumorigenesis in mice compared to mice not exposed to RF radiation.
Zook et al[45]	Promotive effects of RF radiation on ethylnitrosourea (ENU)-induced brain tumors were studied Sprague-Dawley rats irradiated with continuous-wave or pulsed-wave 860 MHz RF (SAR=1 W/kg) for 6 h/day, 5 d/week from 2 up to 24 months of age. The exposed animals showed no significant increase in incidence of brain tumors.
Adey et al[46]	Effects of frequency-modulated 836.55 MHz RF (at SAR=1 W/kg in females and 1.2 W/kg in males, simulating hand-held mobile phone use) on ethylnitrosourea (ENU)-induced brain tumors in F344 rats was studied. No RF field-mediated changes were observed in number, incidence, or histological type of either spontaneous or ENU-induced brain tumors, nor were gender differences detected in tumor numbers.
Inoue et al[47]	Neurite outgrowth increased 10-fold in a PC12 cell line (rat pheochromocytoma cell line) exposed to 2.45 GHz (200W) microwave irradiation compared to non-exposed controls.
Qutob et al[48]	Effect on global gene expression in U87MG mouse glioblastoma cells exposed to 1.9GHz pulse-modulated EMF field for 4h at SARs of 0.1, 1 and 10 W/kg. No effects on gene expression compared to non-exposed control cells.

SAR, specific absorption rate; DCS is also known as GSM-1800 MHz; GSM, global system for mobile communications (originally from Groupe Spécial Mobile); UMTS, universal mobile telecommunications system or 3GSM; W-CDMA, wide-band code division multiple access; DMBA, 7,12-dimethylbenz(a)anthracene; FDMA, frequency-division multiple access; RF, radio frequency.

receives the highest RF radiation exposure from mobile phones, research has been most intensive in this region. Some tumors, like astrocytomas, gliomas and acoustic neuromas, have been shown by several studies to be associated with mobile phone usage. However, research into this field is fraught with certain drawbacks, such as recall bias, inaccurate radiation dose quantification, selection bias and so on.[49,50] Animal studies have provided no consistent relation between cancer and non-thermal range RF exposure. Based on the human studies reviewed, certain factors come forth as having a possible risk towards development of cancer, such as use of analog or cordless phones, predominant unilateral usage and exposure >10 years. While it may well be prudent to avoid these practises, it must be emphasized that clear, long-term cohort studies have not been conducted to prove any causative role of mobile phone radiation in cancer pathogenesis.

References

1. GSM Association Market Data Summary. Available at: http://www.gsmworld.com/newsroom/market-data/market_data_summary.htm [Last accessed November 25, 2008]
2. Kapdi M, Hoskote SS, Joshi SR. Health hazards of mobile phones: an Indian perspective. J Assoc Physicians India 2008;56:893-7.
3. Mild KH, Hardell L, Carlberg M. Pooled analysis of two Swedish case-control studies on the use of mobile and cordless telephones and the risk of brain tumours diagnosed during 1997-2003. Int J Occup Saf Ergon 2007;13(1):63-71.
4. Lahkola A, Auvinen A, Raitanen J, Schoemaker MJ, Christensen HC, Feychting M, Johansen C, Klaeboe L, Lönn S, Swerdlow AJ, Tynes T, Salminen T. Mobile phone use and risk of glioma in 5 North European countries. Int J Cancer 2007;120(8):1769-75.
5. Hardell L, Mild KH, Carlberg M, Söderqvist F. Tumour risk associated with use of cellular telephones or cordless desktop telephones. World J Surg Oncol 2006;4:74.
6. Hardell L, Carlberg M, Hansson Mild K. Pooled analysis of two case-control studies on use of cellular and cordless telephones and the risk for malignant brain tumours diagnosed in 1997-2003. Int Arch Occup Environ Health 2006;79:630-9.
7. Schüz J, Böhler E, Berg G, Schlehofer B, Hettinger I, Schlaefer K, Wahrendorf J, Kunna-Grass K, Blettner M. Cellular phones, cordless phones, and the risks of glioma and meningioma (Interphone Study Group, Germany). Am J Epidemiol 2006;163:512-20.
8. Hepworth SJ, Schoemaker MJ, Muir KR, Swerdlow AJ, van Tongeren MJ, McKinney PA. Mobile phone use and risk of glioma in adults: case-control study. BMJ 2006;332:883-7.
9. Schoemaker MJ, Swerdlow AJ, Ahlbom A, Auvinen A, Blaasaas KG, Cardis E, et al. Mobile phone use and risk of acoustic neuroma: results of the Interphone case-control study in five North European countries. Br J Cancer 2005;93:842-8.
10. Hardell L, Mild KH, Carlberg M, Hallquist A. Cellular and cordless telephone use and the association with brain tumors in different age groups. Arch Environ Health 2004;59:132-7.
11. Kan P, Simonsen SE, Lyon JL, Kestle JR. Cellular phone use and brain tumor: a meta-analysis. J Neurooncol 2008;86:71-8.
12. Hardell L, Carlberg M, Söderqvist F, Hansson Mild K. Meta-analysis of long-term mobile phone use and the association with brain tumours. Int J Oncol 2008;32:1097-103.
13. Miyakoshi J, Takemasa K, Takashima Y, Ding GR, Hirose H, Koyama S. Effects of exposure to a 1950 MHz radio frequency field on expression of Hsp70 and Hsp27 in human glioma cells. Bioelectromagnetics 2005;26:251-7.
14. Takebayashi T, Varsier N, Kikuchi Y, Wake K, Taki M, Watanabe S, et al. Mobile phone use, exposure to radiofrequency electromagnetic field, and brain tumour: a case-control study. Br J Cancer 2008;98:652-9.
15. Klaeboe L, Blaasaas KG, Tynes T. Use of mobile phones in Norway and risk of intracranial tumours. Eur J Cancer Prev 2007;16:158-64.
16. Takebayashi T, Akiba S, Kikuchi Y, Taki M, Wake K, Watanabe S, Yamaguchi N. Mobile phone use and acoustic neuroma risk in Japan. Occup Environ Med 2006;63:802-7.
17. Schüz J, Böhler E, Schlehofer B, Berg G, Schlaefer K, Hettinger I, et al. Radiofrequency electromagnetic fields emitted from base stations of DECT cordless phones and the risk of glioma and meningioma (Interphone Study Group, Germany). Radiat Res 2006;166:116-9.
18. Lönn S, Ahlbom A, Hall P, Feychting M; Swedish Interphone Study Group. Long-term mobile phone use and brain tumor risk. Am J Epidemiol 2005;161:526-35.
19. Röösli M, Michel G, Kuehni CE, Spoerri A. Cellular telephone use and time trends in brain tumour mortality in Switzerland from 1969 to 2002. Eur J Cancer Prev 2007;16:77-82.
20. Muscat JE, Hinsvark M, Malkin M. Mobile telephones and rates of brain cancer. Neuroepidemiology 2006;27:55-6.
21. Schüz J, Jacobsen R, Olsen JH, Boice JD Jr, McLaughlin JK, Johansen C. Cellular telephone use and cancer risk: update of a nationwide Danish cohort. J Natl Cancer Inst 2006;98:1707-13.
22. Sadetzki S, Chetrit A, Jarus-Hakak A, Cardis E, Deutch Y, Duvdevani S, et al. Cellular phone use and risk of benign and malignant parotid gland tumors—a nationwide case-control study. Am J Epidemiol 2008;167:457-67.
23. Hardell L, Eriksson M, Carlberg M, Sundström C, Mild KH. Use of cellular or cordless telephones and the risk for non-Hodgkin's lymphoma. Int Arch Occup Environ Health 2005;78:625-32.
24. Hardell L, Carlberg M, Ohlson CG, Westberg H, Eriksson M, Hansson Mild K. Use of cellular and cordless telephones and risk of testicular cancer. Int J Androl 2007;30:115-22.
25. Linet MS, Taggart T, Severson RK, Cerhan JR, Cozen W, Hartge P, Colt J. Cellular telephones and non-Hodgkin lymphoma. Int J Cancer 2006;119:2382-8.
26. Lönn S, Ahlbom A, Christensen HC, Johansen C, Schüz J, Edström S, et al. Mobile phone use and risk of parotid gland tumor. Am J Epidemiol 2006;164:637-43.
27. Hardell L, Carlberg M, Hansson Mild K. Use of cellular telephones and brain tumour risk in urban and rural areas. Occup Environ Med 2005;62:390-4.
28. Auvinen A, Toivo T, Tokola K. Epidemiological risk assessment of mobile phones and cancer: where can we improve? Eur J Cancer Prev 2006;15:516-23.
29. Yilmaz F, Dasdag S, Akdag MZ, Kilinc N. Whole-body exposure of radiation emitted from 900 MHz mobile phones does not seem to affect the levels of anti-apoptotic bcl-2 protein. Electromagn Biol Med 2008;27:65-72.
30. Moquet J, Ainsbury E, Bouffler S, Lloyd D. Exposure to low level GSM 935 MHz radiofrequency fields does not induce apoptosis in proliferating or differentiated murine neuroblastoma cells. Radiat Prot Dosimetry 2008 Jun 10. [Epub ahead of print]
31. Smith P, Kuster N, Ebert S, Chevalier HJ. GSM and DCS wireless communication signals: combined chronic toxicity/carcinogenicity study in the Wistar rat. Radiat Res 2007;168:480-92.
32. Sommer AM, Bitz AK, Streckert J, Hansen VW, Lerchl A. Lymphoma development in mice chronically exposed to UMTS-modulated radiofrequency electromagnetic fields. Radiat Res 2007;168:72-80.
33. Oberto G, Rolfo K, Yu P, Carbonatto M, Peano S, Kuster N, Ebert S, Tofani S. Carcinogenicity study of 217 Hz pulsed 900 MHz electromagnetic fields in Pim1 transgenic mice. Radiat Res 2007;168:316-26.
34. Shirai T, Ichihara T, Wake K, Watanabe S, Yamanaka Y, Kawabe M, et al. Lack of promoting effects of chronic exposure to 1.95-GHz W-CDMA signals for IMT-2000 cellular system on development of N-ethylnitrosourea-induced central nervous system tumors in F344

rats. Bioelectromagnetics 2007;28:562-72.

35. Tillmann T, Ernst H, Ebert S, Kuster N, Behnke W, Rittinghausen S, Dasenbrock C. Carcinogenicity study of GSM and DCS wireless communication signals in B6C3F1 mice. Bioelectromagnetics 2007;28:173-87.
36. Joubert V, Leveque P, Cueille M, Bourthoumieu S, Yardin C. No apoptosis is induced in rat cortical neurons exposed to GSM phone fields. Bioelectromagnetics 2007;28:115-21.
37. Yu D, Shen Y, Kuster N, Fu Y, Chiang H. Effects of 900 MHz GSM wireless communication signals on DMBA-induced mammary tumors in rats. Radiat Res 2006;165:174-80.
38. Huang TQ, Lee JS, Kim TH, Pack JK, Jang JJ, Seo JS. Effect of radiofrequency radiation exposure on mouse skin tumorigenesis initiated by 7,12-dimethybenz [alpha] anthracene. Int J Radiat Biol 2005;81:861-7.
39. Sommer AM, Streckert J, Bitz AK, Hansen VW, Lerchl A. No effects of GSM-modulated 900 MHz electromagnetic fields on survival rate and spontaneous development of lymphoma in female AKR/J mice. BMC Cancer 2004;4:77.
40. Anane R, Dulou PE, Taxile M, Geffard M, Crespeau FL, Veyret B. Effects of GSM-900 microwaves on DMBA-induced mammary gland tumors in female Sprague-Dawley rats. Radiat Res 2003;160:492-7.
41. La Regina M, Moros EG, Pickard WF, Straube WL, Baty J, Roti Roti JL. The effect of chronic exposure to 835.62 MHz FDMA or 847.74 MHz CDMA radiofrequency radiation on the incidence of spontaneous tumors in rats. Radiat Res 2003;160:143-51.
42. Heikkinen P, Kosma VM, Alhonen L, Huuskonen H, Komulainen H, Kumlin T, et al. Effects of mobile phone radiation on UV-induced skin tumourigenesis in ornithine decarboxylase transgenic and non-transgenic mice. Int J Radiat Biol 2003;79:221-33.
43. Bartsch H, Bartsch C, Seebald E, Deerberg F, Dietz K, Vollrath L, et al. Chronic exposure to a GSM-like signal (mobile phone) does not stimulate the development of DMBA-induced mammary tumors in rats: results of three consecutive studies. Radiat Res 2002;157:183-90.
44. Heikkinen P, Kosma VM, Hongisto T, Huuskonen H, Hyysalo P, Komulainen H, et al. Effects of mobile phone radiation on X-ray-induced tumorigenesis in mice. Radiat Res 2001;156:775-85.
45. Zook BC, Simmens SJ. The effects of 860 MHz radiofrequency radiation on the induction or promotion of brain tumors and other neoplasms in rats. Radiat Res 2001;155:572-83.
46. Adey WR, Byus CV, Cain CD, Higgins RJ, Jones RA, Kean CJ, et al. Spontaneous and nitrosourea-induced primary tumors of the central nervous system in Fischer 344 rats exposed to frequency-modulated microwave fields. Cancer Res 2000;60:1857-63.
47. Inoue S, Motoda H, Koike Y, Kawamura K, Hiragami F, Kano Y. Microwave irradiation induces neurite outgrowth in PC12m3 cells via the p38 mitogen-activated protein kinase pathway. Neurosci Lett 2008;432:35-9.
48. Qutob SS, Chauhan V, Bellier PV, Yauk CL, Douglas GR, Berndt L, et al. Microarray gene expression profiling of a human glioblastoma cell line exposed in vitro to a 1.9 GHz pulse-modulated radiofrequency field. Radiat Res 2006;165:636-44.
49. Kundi M, Mild K, Hardell L, Mattsson MO. Mobile telephones and cancer—a review of epidemiological evidence. J Toxicol Environ Health B Crit Rev 2004;7:351-84.
50. Lahkola A, Salminen T, Auvinen A. Selection bias due to differential participation in a case-control study of mobile phone use and brain tumors. Ann Epidemiol 2005;15:321-5.