Mobile phone

Mobile phone providers use electromagnetic radiation (EMR) with frequencies ranging from 900 to 1800 MHz. The increasing use of mobile phones has been accompanied by several potentially pathological consequences, such as neurological diseases related to hippocampal (HIPPON) and dorsal root ganglion neuron (DRGN). The TRPV1 channel is activated different stimuli, including CapN, high temperature and oxidative stress.

Ertilav et al. investigated the contribution TRPV1 to mitochondrial oxidative stress and apoptosis in HIPPON and DRGN following long term exposure to 900 and 1800 MHz in a rat model. Twenty-four adult rats were equally divided into the following groups: (1) control, (2) 900 MHz, and (3) 1800 MHz exposure. Each experimental group was exposed to EMR for 60 min/ 5 days of the week during the one year. The 900 and 1800 MHz EMR exposure induced increases in TRPV1 currents, intracellular free calcium influx (Ca2+), reactive oxygen species (ROS) production, mitochondrial membrane depolarization (JC-1), apoptosis, and caspase 3 and 9 activities in the HIPPON and DRGN. These deleterious processes were further increased in the 1800 MHz experimental group compared to the 900 MHz exposure group. In conclusion, mitochondrial oxidative stress, programmed cell death and Ca2+ entry pathway through TRPV1 activation in the HIPPON and DRGN of rats were increased in the rat model following exposure to 900 and 1800 MHz cell frequencies. Our results suggest that exposure to 900 and 1800 MHz EMR may induce a dose-associated, TRPV1-mediated stress response ¹.

Mobile phone use has been increasing rapidly in the past decades and, in parallel, so has the annual incidence of certain types of brain tumors. However, it remains unclear whether this correlation is coincidental or whether use of mobile phones may cause the development, promotion or progression of specific cancers.

The 1985-2014 incidence of selected brain cancer subtypes in England were analyzed and compared to counterfactual 'synthetic control' timeseries.

Annual 1985-2014 incidence of high grade glioma, glioblastoma multiforme, and malignant neoplasms of the temporal and parietal lobes in England were modelled based on population-level covariates using Bayesian structural time series models assuming 5,10 and 15year minimal latency periods. Post-latency counterfactual 'synthetic England' timeseries were nowcast based on covariate trends. The impact of mobile phone use was inferred from differences between measured and modelled time series.

There is no evidence of an increase in malignant glioma, glioblastoma multiforme, or malignant neoplasms of the parietal lobe not predicted in the 'synthetic England' time series. Malignant neoplasms of the temporal lobe however, have increased faster than expected. A latency period of 10 years reflected the earliest latency period when this was measurable and related to mobile phone penetration rates, and indicated an additional increase of 35% (95% Credible Interval 9%:59%) during 2005-2014; corresponding to an additional 188 (95%CI 48-324) cases annually.

A causal factor, of which mobile phone use (and possibly other wireless equipment) is in agreement with the hypothesized temporal association, is related to an increased risk of developing malignant neoplasms in the temporal lobe 2 .

Mobile phone

Previous studies have shown a consistent association between long-term use of mobile and cordless phones and glioma and vestibular schwannoma, but not for meningioma. When used these phones emit radiofrequency electromagnetic fields (RF-EMFs) and the brain is the main target organ for the handheld phone. The International Agency for Research on Cancer (IARC) classified in May, 2011 RF-EMF as a group 2B, i.e. a 'possible' human carcinogen. The aim of a study was to further explore the relationship between especially long-term (>10 years) use of wireless phones and the development of malignant brain tumors.

Hardell et al., conducted a new case-control study of brain tumour cases of both genders aged 18-75 years and diagnosed during 2007-2009. One population-based control matched on gender and age (within 5 years) was used to each case. Here, they report on malignant cases including all available controls. Exposures on e.g. use of mobile phones and cordless phones were assessed by a selfadministered questionnaire. Unconditional logistic regression analysis was performed, adjusting for age, gender, year of diagnosis and socio-economic index using the whole control sample. Of the cases with a malignant brain tumour, 87% (n=593) participated, and 85% (n=1,368) of controls in the whole study answered the questionnaire. The odds ratio (OR) for mobile phone use of the analogue type was 1.8, 95% confidence interval (CI)=1.04-3.3, increasing with >25 years of latency (time since first exposure) to an OR=3.3, 95% CI=1.6-6.9. Digital 2G mobile phone use rendered an OR=1.6, 95% CI=0.996-2.7, increasing with latency >15-20 years to an OR=2.1, 95% CI=1.2-3.6. The results for cordless phone use were OR=1.7, 95% CI=1.1-2.9, and, for latency of 15-20 years, the OR=2.1, 95% Cl=1.2-3.8. Few participants had used a cordless phone for >20-25 years. Digital type of wireless phones (2G and 3G mobile phones, cordless phones) gave increased risk with latency >1-5 years, then a lower risk in the following latency groups, but again increasing risk with latency >15-20 years. Ipsilateral use resulted in a higher risk than contralateral mobile and cordless phone use. Higher ORs were calculated for tumours in the temporal and overlapping lobes. Using the meningioma cases in the same study as reference entity gave somewhat higher ORs indicating that the results were unlikely to be explained by recall or observational bias. This study confirmed previous results of an association between mobile and cordless phone use and malignant brain tumours. These findings provide support for the hypothesis that RF-EMFs play a role both in the initiation and promotion stages of carcinogenesis³⁾.

Prospective studies

During 7 years' follow-up, 51,680 incident invasive cancers and 1,261 incident intracranial CNS tumours occurred. Risk among ever vs never users of mobile phones was not increased for all intracranial CNS tumours (RR = 1.01, 95% CI = 0.90-1.14, P = 0.82), for specified CNS tumour types nor for cancer at 18 other specified sites. For long-term users compared with never users, there was no appreciable association for glioma (10+ years: RR = 0.78, 95% CI = 0.55-1.10, P = 0.16) or meningioma (10+ years: RR = 1.10, 95% CI = 0.66-1.84, P = 0.71). For acoustic neuroma, there was an increase in risk with long term use vs never use (10+ years: RR = 2.46, 95% CI = 1.07-5.64, P = 0.03), the risk increasing with duration of use (trend among users, P = 0.03).

In this large prospective study, mobile phone use was not associated with increased incidence of glioma, meningioma or non-CNS cancers ⁴⁾.

Metaanalysis

Results of epidemiological studies on the association between use of mobile phone and brain cancer are ambiguous, as well as the results of 5 meta-analysis studies published to date. Since the last meta-analysis (2009), new case-control studies have been published, which theoretically could affect the conclusions on this relationship. Therefore, Bortkiewicz et al. decided to perform a new meta-analysis.

They conducted a systematic review of multiple electronic data bases for relevant publications. The inclusion criteria were: original papers, case-control studies, published till the end of March 2014, measures of association (point estimates as odds ratio and confidence interval of the effect measured), data on individual exposure. Twenty four studies (26 846 cases, 50 013 controls) were included into the meta-analysis. A significantly higher risk of an intracranial tumor (all types) was noted for the period of mobile phone use over 10 years (odds ratio (OR) = 1.324, 95% confidence interval (CI): 1.028-1.704), and for the ipsilateral location (OR = 1.249, 95% CI: 1.022-1.526). The results support the hypothesis that long-term use of mobile phone increases risk of intracranial tumors, especially in the case of ipsilateral exposure. Further studies are needed to confirm this relationship. ⁵.

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