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Int J Radiat Biol. 2009 May;85(5):454-64.

Absence of genotoxic potential of 902 MHz (GSM) and 1747 MHz (DCS) wireless communication signals: In vivo two-year bioassay in B6C3F1 mice.

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Abstract

PURPOSE: The aim of the present investigation was to determine the incidence of micronuclei in peripheral blood erythrocytes of B6C3F1 mice that had been chronically exposed to radiofrequencies (RF) used for mobile communication.

MATERIALS AND METHODS: 'Ferris wheels' were used to expose tube-restrained male and female mice to simulated environmental RF signals of the Global System for Mobile Communications (GSM, 902 MHz) or Digital Cellular System (DCS, 1747 MHz). RF signals were applied to the mice for 2 hours/day on 5 days/week for two years, at maximal whole-body-averaged specific absorption rates of 0.4, 1.3, and 4.0 W/kg body weight. Concurrent sham-exposed mice, cage controls, and positive controls injected with mitomycin C were included in this investigation. At necropsy, peripheral blood smears were prepared, and coded slides were stained using May-Grunwald-Giemsa or acridine orange. The incidence of micronuclei was recorded for each mouse in 2000 polychromatic and 2000 normochromatic erythrocytes.

RESULTS: There were no significant differences in the frequency of micronuclei between RF-exposed, sham-exposed, and cage control mice, irrespective of the staining/counting method used. Micronuclei were, however, significantly increased in polychromatic erythrocytes of the positive control mice.

CONCLUSIONS: In conclusion, the data did not indicate RF-induced genotoxicity in mice after two years of exposure.

Publication Types, MeSH Terms

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Radiat Res. 2008 May;169(5):561-74.

Genetic damage in mammalian somatic cells exposed to radiofrequency radiation: a meta-analysis of data from 63 publications (1990-2005).

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Abstract

During the last several decades, numerous researchers have examined the potential of in vitro and /or in vivo exposure of radiofrequency(RF) radiation to damage the genetic material in mammalian somatic cells. A meta-analysis of reported data was conducted to obtain a quantitative estimate (with 95% confidence intervals) of genotoxicity in RF-radiation-exposed cells compared with sham-exposed/unexposed control cells. The extent of genotoxicity was assessed for various end points, including single- and double-strand breaks in the DNA, incidence of chromosomal aberrations, micronuclei and sister chromatid exchanges. Among the several variables in the experimental protocols used in individual investigations, the influence of three specific variables related to RF-radiation exposure characteristics was examined in the meta-analysis: frequency, specific absorption rate, and exposure as continuous-wave, pulsed-wave and occupationally exposed/cell phone users. The overall data indicated that (1) the difference between RF-radiation exposure was small with few exceptions; (2) at certain RF radiation exposure conditions, there were statistically significant increases in genotoxicity for some end points; and (3) the mean indices for chromosomal aberrations and micronuclei in RF-radiation -exposed and sham-/unexposed controls were within the spontaneous levels reported in the historical database. Considerable evidence for publication bias was found in the meta-analysis.

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Radiat Res. 2003 Apr;159(4):558-64.

Genotoxic potential of 1.6 GHz wireless communication signal: in vivo two-year bioassay.

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Abstract

Timed-pregnant Fischer 344 rats (from nineteenth day of gestation) and their nursing offspring (until weaning) were exposed to a far-field 1.6 GHz Iridium wireless communication signal for 2 h/day, 7 days/week. Far-field whole-body exposures were conducted with a field intensity of 0.43 mW/cm(2) and whole-body average specific absorption rate (SAR) of 0.036 to 0.077 W/kg (0.10 to 0.22 W/kg in the brain). This was followed by chronic, head-only exposures of male and female offspring to a near-field 1.6 GHz signal for 2 h/day, 5 days/week, over 2 years. Near-field exposures were conducted at an SAR of 0.16 or 1.6 W/kg in the brain. Concurrent sham-exposed and cage control rats were also included in the study. At the end of 2 years, all rats were necropsied. Bone marrow smears were examined for the extent of genotoxicity, assessed from the presence of micronuclei in polychromatic erythrocytes. The results indicated that the incidence of micronuclei/2000 polychromatic erythrocytes were not significantly different between 1.6 GHz-exposed, sham-exposed and cage control rats. The group mean frequencies were 5.6 +/- 1.8 (130 rats exposed to 1.6 GHz at 0.16 W/kg SAR), 5.4 +/- 1.5 (135 rats exposed to 1.6 GHz at 1.6 W/kg SAR), 5.6 +/- 1.7 (119 sham-exposed rats), and 5.8 +/- 1.8 (100 cage control rats). In contrast, positive control rats treated with mitomycin C exhibited significantly elevated incidence of micronuclei/2000 polychromatic erythrocytes in bone marrow cells; the mean frequency was 38.2 +/- 7.0 (five rats). Thus there was no evidence for excess genotoxicity in rats that were chronically exposed to 1.6 GHz compared to sham-exposed and cage controls.

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