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

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INTRODUCTION

The use of cellular telephones is increasing rapidly; by the end of the year 2000, there were more than 200 million users worldwide. Therefore, radiofrequency (RF) electromagnetic fields used for mobile telecommunication represent rapidly increasing environmental influences. Concern is spreading that these fields may exert negative effects on health, including the development of cancer. Doubts concerning the safety of mobile telecommunication were supported by a publication of Repacholi et al. (1), who reported an enhanced development of experimental lymphomas in transgenic mice exposed to RF fields (900 MHz pulsed at 217 Hz). However, previous experimental and epidemiological studies, which were reviewed critically by Moulder et al. (2) and are discussed in detail later, have yielded no or at most weak evidence that the low-intensity RF fields used for mobile telecommunication may possess carcinogenic or tumor-promoting effects. However, the worldwide spread of mobile telecommunication requires special caution, since even a marginal cancer-elevating effect could harm thousands of individuals. For this reason, further experimental and epidemiological studies are urgently needed, and an International EMF Project has been launched by the WHO. In the agenda of this program, investigations on chemically induced animal tumors are ex-

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explicitly included. The current study was designed to analyze the effect of RF fields on 7,12-dimethylbenz[a]anthracene (DMBA)-induced mammary cancers in female Sprague-Dawley rats, which is an accepted model (3) for human breast cancer, being part of the NCI Chemoprevention Drug Development Program (4). The field applied consisted of a GSM-like signal (900 MHz pulsed at 217 Hz, pulse width 577 μs) of low power flux density, so that thermal effects were not expected. At the beginning of the experiment, when the animals were young and small, the specific absorption rates (SARs) averaged over the whole body were close to the limit for general public exposure in humans (i.e. 80 mW/kg) (5–8). The SARs declined up to 50% in the course of the study as the rats grew in size. The current experiments represent a model study of the interaction of the electromagnetic fields (EMFs) used for mobile telecommunication with biological systems. The analogy refers to the case of the whole-body exposure of humans in the far field of the transmitting antenna of a base station. Because the penetration depth of EMFs in biological tissue at 900 MHz, is about 3 cm (9), the EMF penetrates the body of the rats investigated almost completely, whereas the human body is exposed primarily at its surface.

Materials and Methods

Three experiments (I, II, III) were carried out under strictly standardized conditions and were started on the same day of three subsequent years.

Animals and Chemical Induction of Mammary Tumors

The studies were approved by the Animal Care Committee of the regional government of Tübingen and were performed according to the approved protocols. A total of 120 female Sprague-Dawley rats aged 38 days (I), 43 days (II), and 34 days (III) were purchased from Charles River Wiga (Sulzfeld, Germany) and were allowed to acclimate over a period of 13 days (I), 8 days (II), and 17 days (III) to the environmental conditions in our animal facility. Twelve animals were housed per cage. They received tap water and food ad libitum (pellets, ssniff from R/M- H; Soest, Germany). The lighting regimen comprised 12 h of illumination per 24 h, with lights turned on at 0700 h European Summer time throughout the year. Air temperature (T) and relative air humidity (rAH) were recorded daily; the average values were in close agreement in the experimental and control rooms, respectively. On June 18, 1997 (I), 1998 (II), and 1999 (III), when the animals were 51 days old, the experiment was initiated by administering a single intragastric dose of 8.75 mg DMBA (5 mg DMBA/100 g body weight) in 1 ml of peanut oil (10). In the evening of the same day, the RF-field exposure was started.

Radiofrequency Electromagnetic Field Exposure

A sketch of an exposure chamber used for the experiments is shown in Fig. 1. The chambers were designed for continuous low-level RF irradiation of restrained animals. They were manufactured from sheet steel, each containing in its bottom part a transparent polymethacrylate (Plexiglas) cage with a ground area of about 0.4 m² and a height of 20 cm. Each animal cage was equipped with three stainless steel nipples for drinking. The nipples were connected by silicone tubing to a water bottle fixed outside the exposure chamber. Each cage was covered by a Plexiglas lid perforated with holes, each 1 cm in diameter and spaced 1 cm from each other.

Within the exposure chamber, the animal cage was located in the far field of a flat spiral antenna (AIL, New York) emitting a clockwise circularly polarized, GSM-like RF signal (900 MHz pulsed with 217 Hz, pulse width 577 μs) which was at a distance of 130 cm from the bottom of the cage. The mean power flux density of the field at the bottom of the cage was 100 μW/cm², with a variation of ±3 dB between the center and the corners of the cage (see Fig. 2). It was measured with a small isotropic electric probe, and data were transmitted through glass fiber wire to a laptop computer placed outside the chamber. It should be noted that it was adequate to measure only the electric component of the applied electromagnetic field, since the electric and the magnetic components are equivalent under the existing far-field conditions. Reflections of the electromagnetic waves within the exposure chamber were sufficiently atten-
SAR Values

The RF fields in the exposure chambers were circularly polarized to achieve independence from the orientation of the animals in case of possible biological interactions. The fields had an average power flux density of 100 $\mu$W/cm$^2$ at the bottom of the animal cages, with a variation of $\pm 3$ dB between the corners ($50 \mu$W/cm$^2$) and the center ($200 \mu$W/cm$^2$). The SAR characterizing the amount of electromagnetic energy absorbed by a rat in the experiment was determined by means of computer simulations with MAFIA software (CST GmbH, Darmstadt, Germany) using an anatomically correct model of the body of a rat (11). The accuracy of the calculation of SAR values depends strongly on the varying dielectric properties of the animal tissues, leading to an uncertainty of less than 20%. For an adult female Sprague-Dawley rat weighing 300 g, the SAR for the whole body was 17.5–70 mW/kg. Since the amount of electromagnetic energy absorbed by young animals with small body dimensions is higher than that for older animals, the SAR for the whole body was bound to decline continuously during the course of the experiment:

Young animals, at the beginning of the experiment (aged 51 days, weighing 150 g), exhibited SARs for the whole body in the range between 32.5 mW/kg and 130 mW/kg, which lay close to the limits set for non-occupational exposure in humans, whereas older rats, at the end of the experiment (aged 11 to 12 months, weighing 400 g), exhibited lower SARs for the whole body, between 15 mW/kg and 60 mW/kg. The reason for this phenomenon is that young animals have a body length (excluding the tail) that is approximately half the wavelength of the RF signal. At half the wavelength, because of a resonance effect, the absorbed power is maximal, and it then decreases as the animals get older and grow in size. Since the animals could move freely in their cages, it is probable that comparable doses of electromagnetic energy were absorbed by animals of similar size and age, leading to similar SARs for the whole body within the limits given above. When animals rested and “piled up”, the SARs of the animals lying underneath were about half of the value for a single animal moving around or lying separately. It is noted that the above-mentioned SAR values are based on calculations for a standard rat model, and it is conceivable that they may have shown larger variations if they had been based on actual phantom measurements of individual rats of different sizes and ages.

Detection and Histopathological Characterization of the Mammary Tumors

Based on previous experience with this breast cancer model, the carcinogen dose applied should lead to development of the mammary tumors starting 2 months after DMBA administration. Therefore, animals were palpated for the presence of mammary tumors once weekly beginning the seventh week after treatment (by the same investigator in all three experiments). This examination was carried out blindly in experiment III. The time and site of detection were recorded for each tumor in an animal. The respective animal was killed humanely when the largest tumor exceeded 1–2 cm in diameter to allow optimal histopathological tumor classification before necrosis occurred. Animals were dissected completely, and all possible positions along the milk line were checked for the presence of additional mammary tumors that were too small to be detected by palpation. All tissues removed were fixed in 10% phosphate-buffered formaldehyde solution, and paraffin-embedded sections were stained with hematoxylin and eosin and prepared for subsequent histopathological investigations. The mammary tumors were characterized according to Russo et al. (12) as well as Warburton and Gusterson (13). The pathologist was not told whether the tissue was from an RF-field- or sham-exposed rat.

Quantitative Evaluation of the Development of Mammary Tumors and Statistics

Based upon the histopathological classification described above, the probability of detecting the first malignant tumor as well as the first benign tumor in each animal was calculated for the groups of RF-field-exposed and sham-exposed animals according to Kaplan and Meier (14). This statistical method includes censoring in the case of incomplete information if, e.g., no tumor of the relevant type was present at the time of death. The results of the statistical analysis are presented in the form of Kaplan-Meier plots in which the horizontal axis represents the time since DMBA administration and the vertical axis the estimated probability of tumor detection, i.e. the cumulative tumor incidence. For each group of animals, the median tumor latency was calculated (i.e. the time when the cumulative tumor incidence equals 50%), and the groups were compared using the Peto and Peto Wilcoxon test (14). The development of malignant and benign tumors in both the control and experimental groups of all three experiments together was compared by means of the likelihood ratio test subsequent to fitting a proportional hazards model (14).

RESULTS

Since our aim was to observe the development of mammary tumors in the carcinogen-treated animals and to facilitate a reliable histopathological classification at the same time, it was necessary to kill the respective animals as soon as their initially detected mammary tumors reached 1–2 cm in diameter. Therefore, neither the measured tumor volume nor the number of tumors per animal could be used for comparative purposes as is commonly done in so-called
“stop experiments” when all the animals are killed simultaneously.

Histopathology of DMBA-Induced Mammary Tumors

The tumors developed in the mammary glands along the milk line, predominantly in the thoracic regions, and were classified histopathologically as malignant and as benign types. The latency was shorter for malignant tumors than for benign tumors, as reported previously (15). The malignant tumors were exclusively adenocarcinomas with different degrees of differentiation, whereas the benign tumors were adenomas, adenofibromas or fibroadenomas. A few adenomas showed a transition toward malignancy; in this case, the tumors were categorized as malignant during the subsequent steps of evaluation. In experiment I, two RF-field-exposed animals had to be excluded from the evaluation because tumor specimens were lost during histological processing.

Tumor Development

Figure 3 depicts the development of malignant (left panels) and benign (right panels) tumors in all three experiments. Table 1 summarizes the results of the statistical analyses of each experiment, namely, median tumor latency and cumulative tumor incidence at the end of the experiment.

**Experiment I.** The left-hand panel of Fig. 3 (top) shows the time course of the cumulative incidence of malignant tumors for the groups of RF-field-exposed animals and their sham-exposed controls. The median tumor latency was statistically significantly longer for RF-field-exposed animals than for controls (278 days compared to 145 days, \( P = 0.009 \)), but the cumulative tumor incidences at day 334 (the latest day on which both curves are comparable) were more or less identical, 82% for exposed animals and 79% for control animals (see Table 1). The right-hand panel of Fig. 3 (top) illustrates the corresponding time course of the cumulative incidence of benign tumors in the experimental and control groups. It is apparent that benign mammary tumors were observed later than malignant tumors, at a time when the development of malignant tumors was more or less completed. The median latencies for benign tumors were practically identical for RF-field-exposed animals and for controls (310 days compared to 316 days, \( P = 0.878 \)), and the cumulative tumor incidences at day 334 were also almost the same, 91% for the exposed and 90% for the control animals.

**Experiment II.** The middle panels of Fig. 3 show that there were no differences between RF-field-exposed and sham-exposed animals in the development of either malignant or benign tumors. The statistical analyses (see Table 1) revealed identical median tumor latencies of 95 days for malignant tumors in both groups; tumor latencies for benign tumors were \( >265 \) days in controls and 221 days in the RF-field-exposed group, which is statistically insignificant (\( P = 0.758 \) and \( P = 0.557 \) for differences between the groups for the development of malignant and benign tumors, respectively). Cumulative tumor incidences for malignant tumors were 84% in the controls and 94% in experimental animals. Cumulative tumor incidences for benign tumors were 38% in sham-exposed animals and 60% in RF-field-exposed animals.

**Experiment III.** The bottom panels of Fig. 3 demonstrate a lack of any difference between RF-field-exposed and sham-exposed animals for the development of malignant as well as benign tumors. Table 1 lists the results of the statistical analysis: There was no difference between RF-field-exposed and sham-exposed animals in the development of malignant (\( P = 0.939 \)) and benign (\( P = 0.568 \)) tumors, with median tumor latencies of 216 days in controls and 195 days in RF-field-exposed animals for malignant tumors and of 293 and 321 days for benign tumors. The cumulative tumor incidences were similar in both groups for malignant tumors.
Comparison between experiments I–III. The likelihood ratio test revealed that tumor development as a whole differed significantly in the three different experiments \((P < 0.0001\) for malignant tumors and \(P < 0.0005\) for benign tumors). However, there was no significant difference between the development of tumors in the sham- and RF-field-exposed groups \((P = 0.47\) for malignant tumors and \(P = 0.38\) for benign tumors). Moreover, there were no significant interactions between the three experiments and the two treatments \((P = 0.12\) for malignant tumors and \(P = 0.41\) for benign tumors). The risk ratios were 1.08 (95% CI: 0.91–1.29) and 0.96 (95% CI: 0.85–1.07) for benign and malignant tumors, respectively.

**DISCUSSION**

The overall result of the current experiments was that the RF field applied did not affect the development of DMBA-induced mammary tumors, but it must be stressed that a statistically significantly delayed median latency was found for malignant tumors in experiment I, whereas no effect occurred in experiments II and III. These results must be viewed in the framework of related studies; therefore, a review of the current literature is given below.

Several reports deal with experimental studies on cancer in laboratory animals. Experiments relating to mammary cancer have used spontaneous breast carcinomas in C3H mice, which are induced by the mouse mammary tumor virus. In an initial study, such tumors appeared more rapidly under 2450 MHz continuous microwave fields at either 5 mW/cm² or 15 mW/cm² (2 h/day, 6 days/week over 6 months) \((16)\). However, if irradiated animals were compared to controls kept under the same confined conditions, no significant differences were detectable, indicating that the reported effects were due to confinement stress. Mammary tumor development in C3H/HeJ mice also was not stimulated by a weak 435 MHz RF field at 1 mW/cm² (horizontally polarized and pulsed with 10,000 pulses/s) applied for 22 h/day, 7 days/week over 21 months \((17)\) or by a 2450 MHz field, circularly polarized continuous wave (CW), given for 20 h/day, 7 days/week over 18 months \((18, 19)\). In the same animal model, exposure to ultra-wideband pulses (including frequencies in the RF range) had no significant effects on mammary tumor development \((20)\).

With respect to other experimental solid tumors, RF fields failed to show stimulatory effects. Continuous RF irradiation at 2450 MHz of 10 mW/cm² \((3\) h/day, 6 days/week for 5 months) had no tumor-promoting effect on dimethylhydrazine-induced colon cancer in mice \((21)\), and microwave irradiation \((2450\ MHz,\ CW\ or\ pulsed, 1\ mW/cm²)\) failed to stimulate the growth of transplantable B16 melanomas in C57/6f black mice \((22)\). Long-term exposure of tumor-bearing rats to a 929.2 MHz field (pulsed according to the Japanes cellular telephone standard) applied for 90 min/day, 5 days/week over 6 weeks did not promote diethylnitrosamine-induced liver cancers \((23)\), and short-term exposure to GSM-modulated low-power microwaves \((900\ MHz,\ 55\ or\ 200\ \mu W/cm²)\) given 2 h/day, 5 days/week over 2 weeks elicited no effect on benzo(a)pyrene-induced sarcomas \((24)\). Hormone-independent brain tumors in rats induced chemically \((25)\) were not affected by frequency-modulated, circularly polarized 836 MHz fields; those results were supported by a similar recent study applying CW or pulsed-wave 860 MHz fields to rats with ethylnitrosourea (ENU)-induced brain tumors \((26)\). 9L gliosarcoma cells

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**TABLE 1**

<table>
<thead>
<tr>
<th>Statistical parameters</th>
<th>Experiment I</th>
<th>Experiment II</th>
<th>Experiment III</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Median tumor latency and 95% confidence limits (days)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant tumors:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sham-exposed</td>
<td>145 (104–229)</td>
<td>95 (82–140)</td>
<td>216 (132–251)</td>
</tr>
<tr>
<td>RF-field-exposed</td>
<td>276 (173–329)</td>
<td>95 (82–130)</td>
<td>195 (111–265)</td>
</tr>
<tr>
<td>Benign tumors:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sham-exposed</td>
<td>316 (264–331)</td>
<td>&gt;265</td>
<td>293 (251–343)</td>
</tr>
<tr>
<td>RF-field-exposed</td>
<td>310 (306–330)</td>
<td>221</td>
<td>321 (251–343)</td>
</tr>
<tr>
<td><strong>Statistical significance of difference between groups</strong>&lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Malignant tumors:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sham-exposed</td>
<td>0.009</td>
<td>0.758; NS</td>
<td>0.939; NS</td>
</tr>
<tr>
<td>RF-field-exposed</td>
<td>0.878; NS</td>
<td>0.557; NS</td>
<td>0.568; NS</td>
</tr>
<tr>
<td>Benign tumors:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sham-exposed</td>
<td>79%</td>
<td>84%</td>
<td>91%</td>
</tr>
<tr>
<td>RF-field-exposed</td>
<td>82%</td>
<td>94%</td>
<td>81%</td>
</tr>
<tr>
<td><strong>Cumulative tumor incidence on last day of observation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malignant tumors:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sham-exposed</td>
<td>90%</td>
<td>38%</td>
<td>89%</td>
</tr>
<tr>
<td>RF-field-exposed</td>
<td>91%</td>
<td>60%</td>
<td>92%</td>
</tr>
</tbody>
</table>

<sup>a</sup> Tumor development in the different groups was compared using the Peto and Peto Wilcoxon test. NS = not significant.
in rats also were not stimulated by a 835.62-MHz frequency-modulated RF carrier or 847.74-MHz code-division multiple-access (CDMA) modulated RF field applied 4 h/day, 5 days/week for 150 days (27). Stereotactically implanted glioma cells in rats were not affected by 915 MHz CW (1 W) or fields pulse-modulated at different frequencies between 4 and 217 Hz (28). It is interesting to note that Fischer F344 rats chronically exposed to a 836.55 MHz field of the North American TDMA standard (2 h/day, 4 days/week for 2 years) tended to show a reduced incidence of spontaneous as well as ENU-induced central nervous system tumors (29). There has been only one report indicating that pulsed RF fields used for mobile telecommunication may be cancer-stimulating. Transgenic mice developed significantly more nonlymphoblastic lymphomas under a weak 900 MHz field pulsed with 217 Hz (0.26–1.3 mW/cm²) which was applied 30 min/day for 18 months (31). This does not necessarily imply that these fields may stimulate or even induce lymphomas in normal mice, but such malignancies should be part of future experimental studies.

These experimental findings should be considered in the framework of the relevant epidemiological studies on RF fields and cancer, which are summarized briefly. Hocking et al. (30) initially reported an association between the incidence of mortality from childhood leukemia and the proximity to TV towers. A reanalysis of the data, however, showed that one highly exposed local government area contributed all of the excess of leukemia and that there was no longer any positive correlation if this area was excluded from the analysis (31). Dolk et al. (32) reported an elevated risk for the development of adult leukemia as well as skin and bladder cancer for the Sutton Coldfield transmitter, but when they analyzed a total of 20 transmitters, the same authors could not confirm the magnitude and pattern of risk for adult leukemia (33). Therefore, Jauchem (34), reviewing the literature between 1995–1998, concluded that the evidence for any proven health effects of low-level microwave exposure was minimal to nonexistent. When epidemiological studies dealing with the effect of occupational radiofrequency exposure on cancer development were reviewed critically (35–44), no convincing evidence was found for such a correlation (2, 45), although some of these studies reported the detection of tumor-stimulatory effects, including effects on the development of leukemia. Such claims appear to be unjustified, since these studies had methodological flaws, e.g. inadequate collection of data on the quantity and quality of radiofrequency exposure as well as possible distortions due to confounding factors that may have stimulated malignancy.

From the review of the literature, we concluded that there is no evidence that RF fields stimulate the development of solid tumors. This is in agreement with the overall results of our current studies. One of the reports cited even indicates that such fields may be tumor-inhibitory (29), supporting the results of the first experiment in this report.

It remains to be discussed why the current three experiments, despite highly standardized conditions, yielded different results. Since the time course of mammary tumor development in the three control groups differed, it may be assumed that this was the reason for the varying RF-field effects observed in the animals. However, this does not appear to be the case, since the inhibitory effect on the development of malignant tumors was found in the first experiment, which had a median tumor latency of 145 days in the controls, which is well within the range of the controls in the other experiments (i.e. 95 and 216 days). Temporal differences in tumor development in the control groups of the three experiments were probably due to the different pre-experimental accommodation periods after transfer from the animal breeder into our laboratory. A direct relationship exists between accommodation periods and the median latency of malignant tumors between controls (experiment II: accommodation period/latency = 8 days/95 days; I: 13/145; III: 17/216). This illustrates that a short accommodation period may accelerate tumor development, possibly due to elevated stress hormone secretion (glucocorticoids, prolactin) in response to transportation and the new environment. Increased glucocorticoid secretion enhances the metabolic activation of DMBA (46, 47), and prolactin stimulates the growth of such mammary tumors (48). It therefore must be ensured in future studies that animals adjust to the new environment for a sufficiently long and strictly controlled period of e.g. 3 weeks before administration of DMBA. In an attempt to explain the varying effects of RF-field exposure in the current experiments, it may be argued further that the tumor-inhibitory effect seen in the first experiment could be due to non-blind palpation. The objectivity of this procedure, however, is illustrated by the fact that in experiment II the same investigator also palpated non-blindly without detecting an effect of RF-field exposure on tumor development. The same finding was also obtained in experiment III, when a blinded protocol was used.

What could be the actual reason(s) for the discordant results among the three experiments? Here, the known mechanisms involved in the initiation as well as the promotion of DMBA-induced mammary tumors should be considered, namely metabolic activation of DMBA by hepatic microsomal enzymes and conversion to diolopoxide derivatives (46), which form DNA adducts leading to carcinogenesis (49), and subsequent neuroendocrine growth stimulation of the transformed cells by hormones of the pituitary-ovarian axis (50) and by prolactin (48). It appears unlikely that initiation was affected, since the final cumulative tumor incidence at the end of the experiment remained unaffected. It is more likely that tumor promotion was perhaps blocked by radiation through the neuroendocrine system, since pulse-modulated RF fields appear to act on neural as well as brain functions (51, 52) and thus may have reduced pituitary prolactin secretion, which regulates the growth of DMBA-induced mammary tumors in rats (48). Since tumor inhibition was observed only in experi-
ment I, it must be assumed that, despite all attempts to attain the utmost controlled conditions, some unknown endogenous or exogenous biological response modifier (53) of either a chemical or a physical nature may have been present and may have facilitated or down-regulated the sensitivity of the neuroendocrine system toward RF-field exposure.

To put the experimental exposure conditions used in the current studies into perspective for humans living in the vicinity of base station antennas for mobile telecommunication, it can be said that the power flux density of 100 μW/cm² attained with the 13–15 W antennas in the current experiment can be found at a distance of 6.3 m from a typical base station antenna of 200 W with 4 dB gain (assuming far-field conditions). At a distance of about 100 m from such a base station, the resulting power flux density would be about 0.4 μW/cm². Therefore, most residents living near conventional base stations will experience considerably lower power flux densities compared to the conditions prevailing in this experiment.

In conclusion, the results of the current three studies as well as the epidemiological and experimental reports summarized above show that low-intensity RF fields do not appear to enhance solid tumor growth and may, under yet unidentified conditions, even be “cancer protective”. Because of certain experimental and epidemiological indications that lymphoblastic and hematopoietic neoplasias might, in contrast to solid tumors, be stimulated by such fields, it appears to be important and urgent to address this question in future investigations using the same exposure conditions.

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